Severe dilated ischemic cardiomyopathy sequelae of infarctions in elderly man

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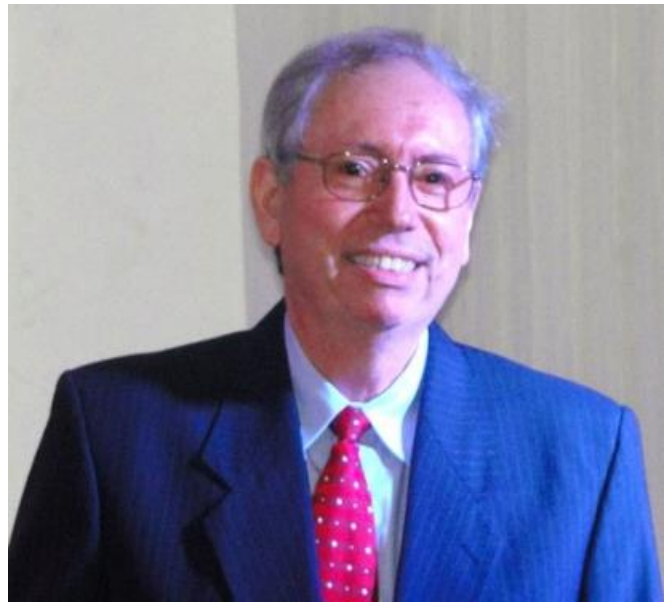

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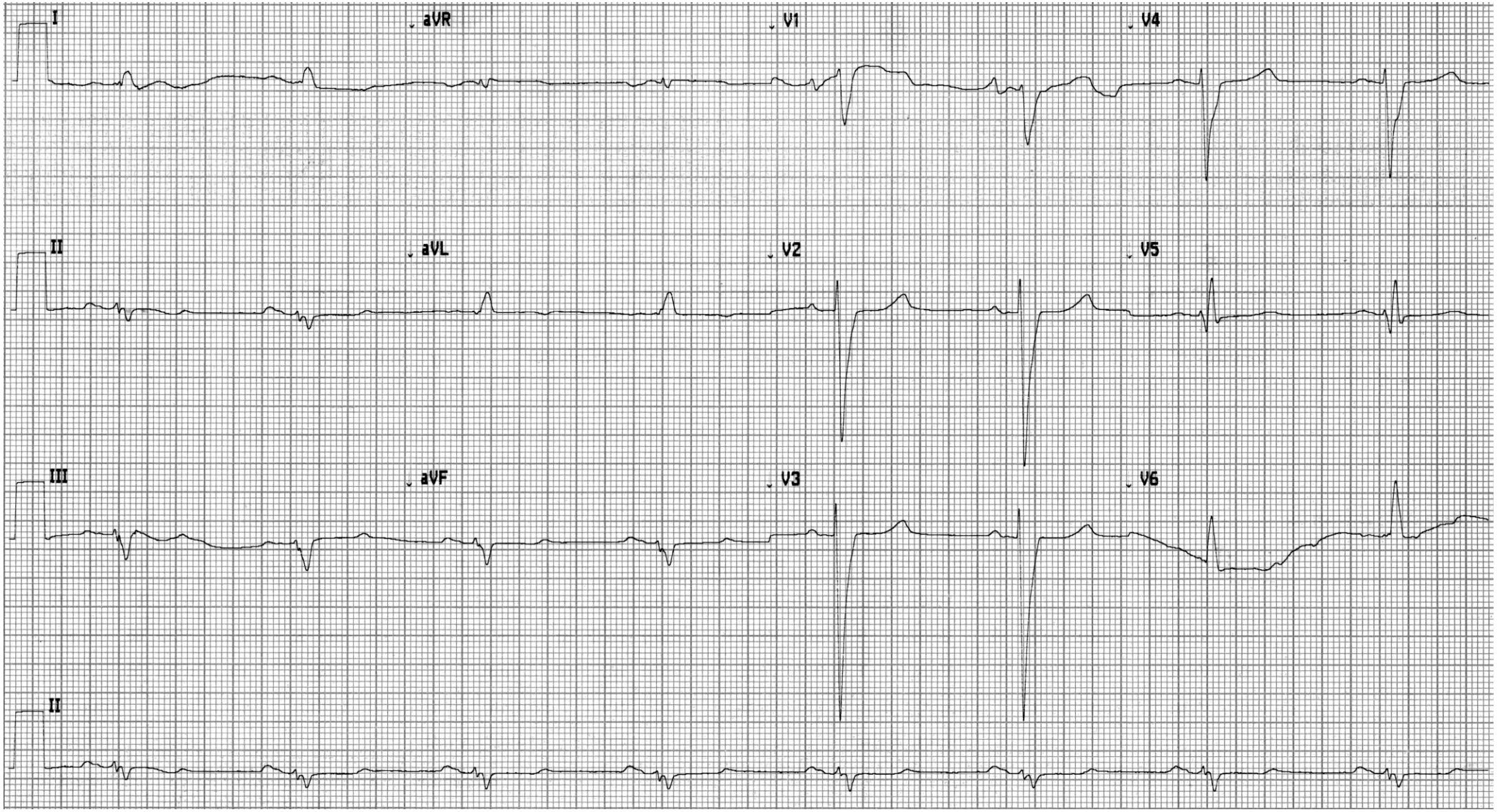
Questions:
Which is the ECG/VCG diagnosis? And why?

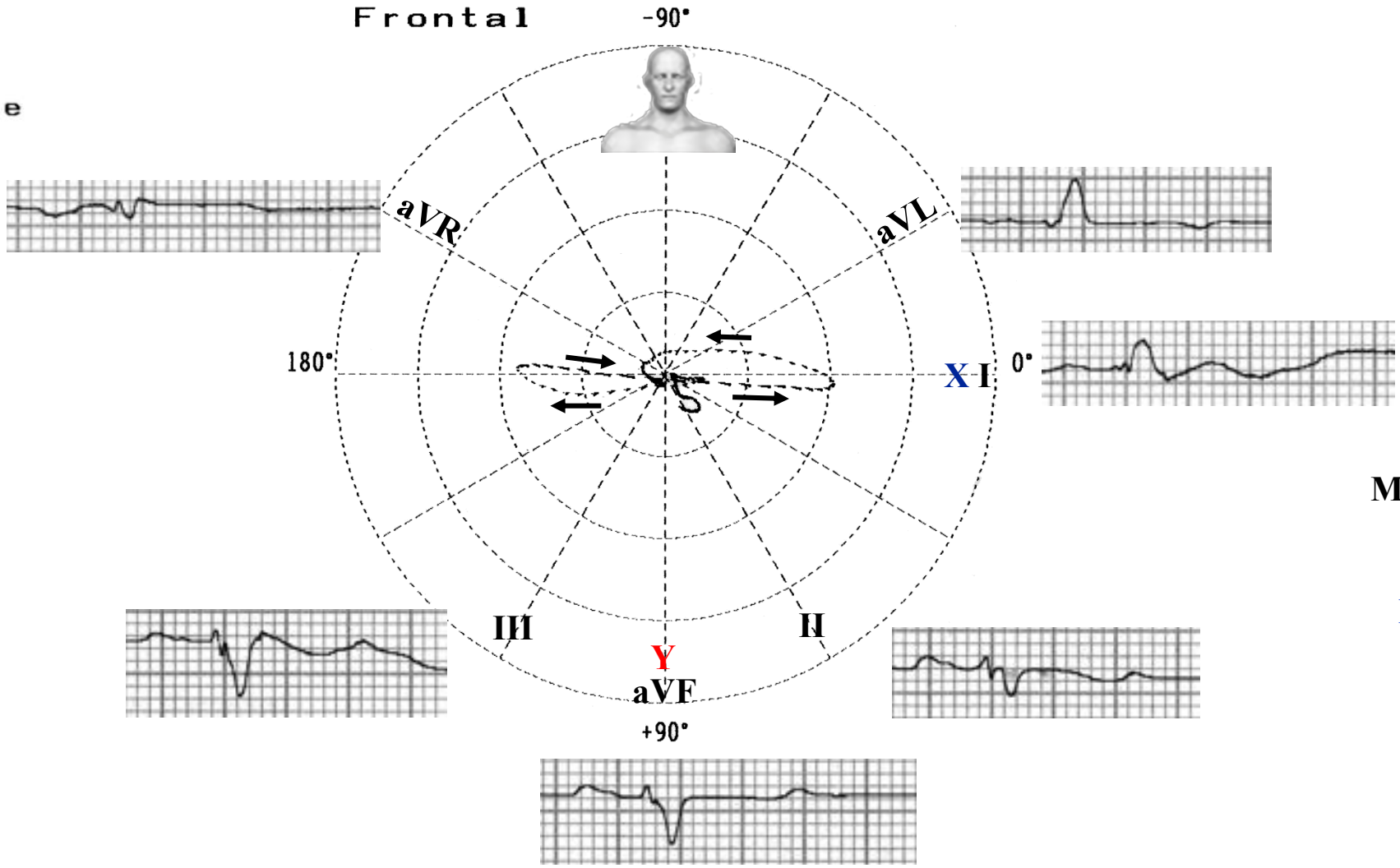
Name: AP; **Age:** 73 y/o; **Weight:** 58 kg; **Height:** 1.68 m; **Date:** Nov 25, 2016; **Medication in use:** carvedilol 25 mg 2x/day, spironolactone 25 mg 1x/day, furosemide 40 mg 1x/day, losartan potassium 25 mg 2x/day, warfarin 5 mg 48h

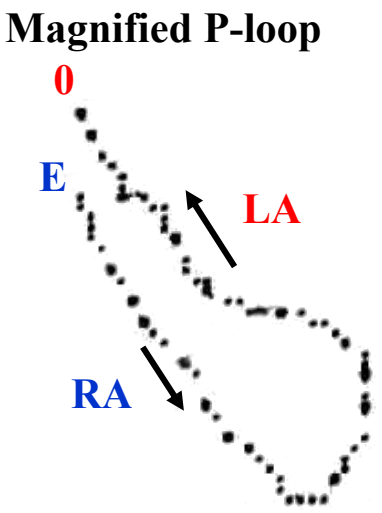


Clinical diagnosis: severe dilated ischemic cardiomyopathy, sequelae of extensive infarctions. LVEF = 18%; Diastolic diameter of LV = 70 mm

ECG/VCG correlation in the Frontal Plane

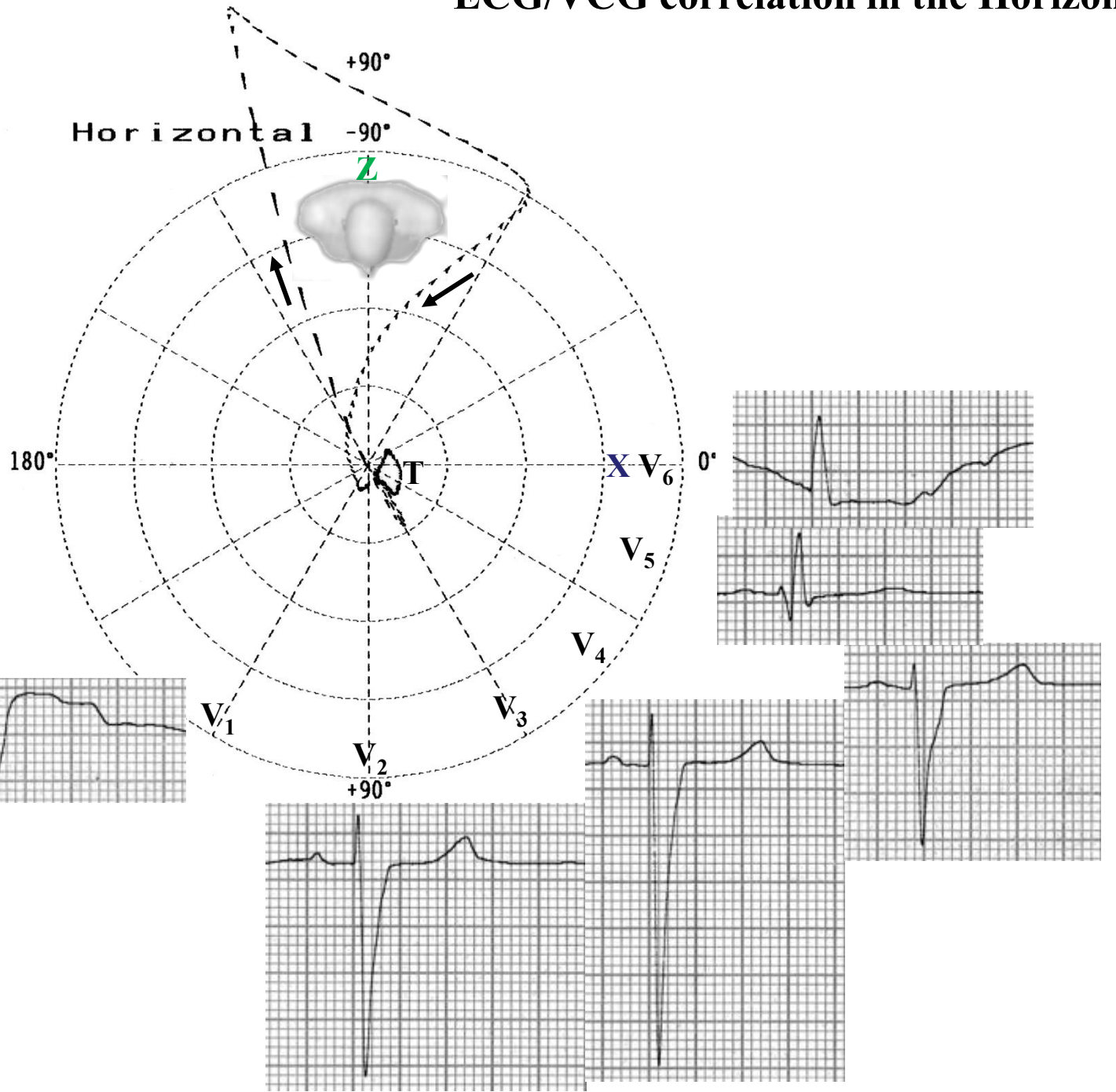
Sensi.	4
Timer	2 msec
Loop	All Loop
Sagittal	Right
Z Axis	Front
Filter	Hum
	Muscle
	Drift



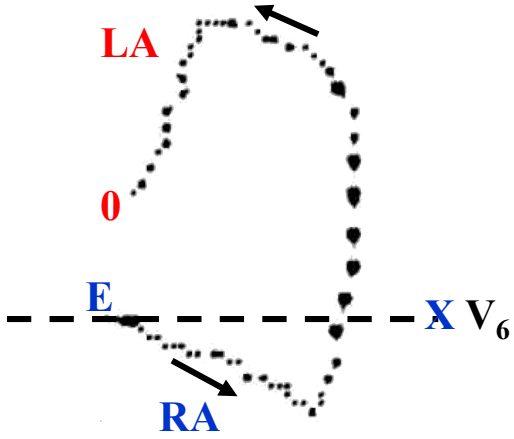


SÂP +62°

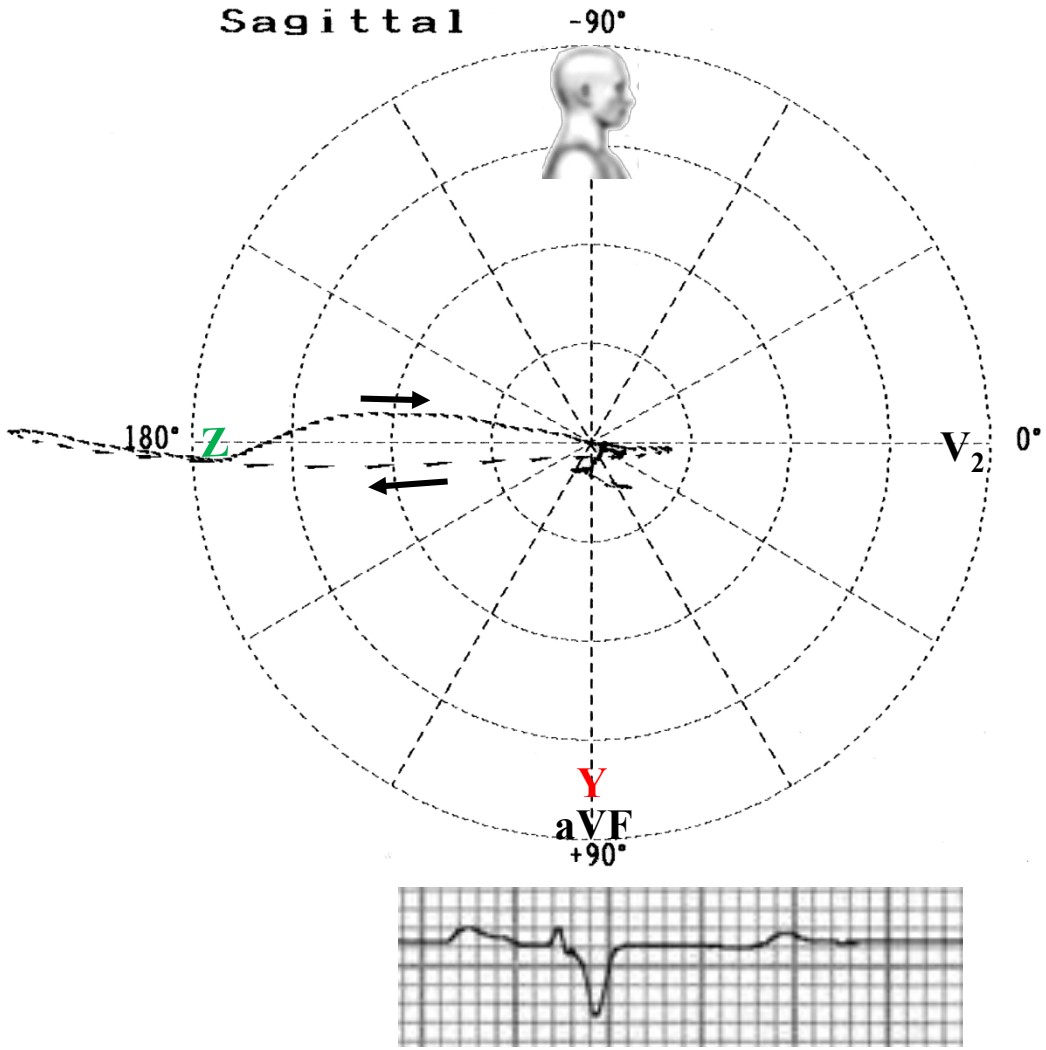
ECG/VCG correlation in the Horizontal Plane

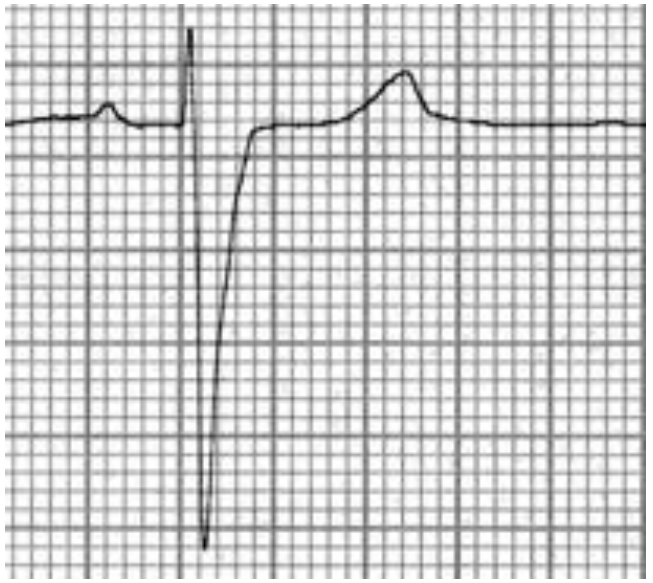


Magnified P-loop

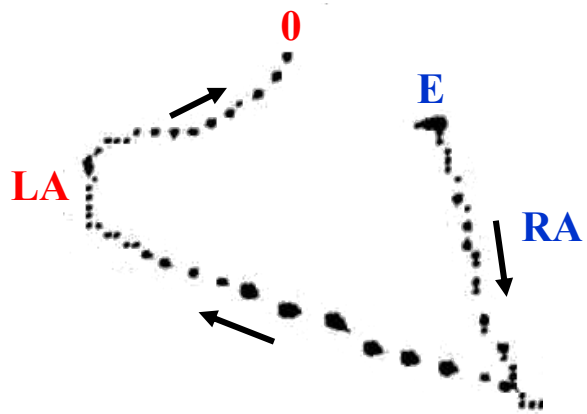


ECG/VCG correlation in the Right Sagittal Plane





Magnified P-loop



Colleagues opinions

Old inferior and Anterior MI.

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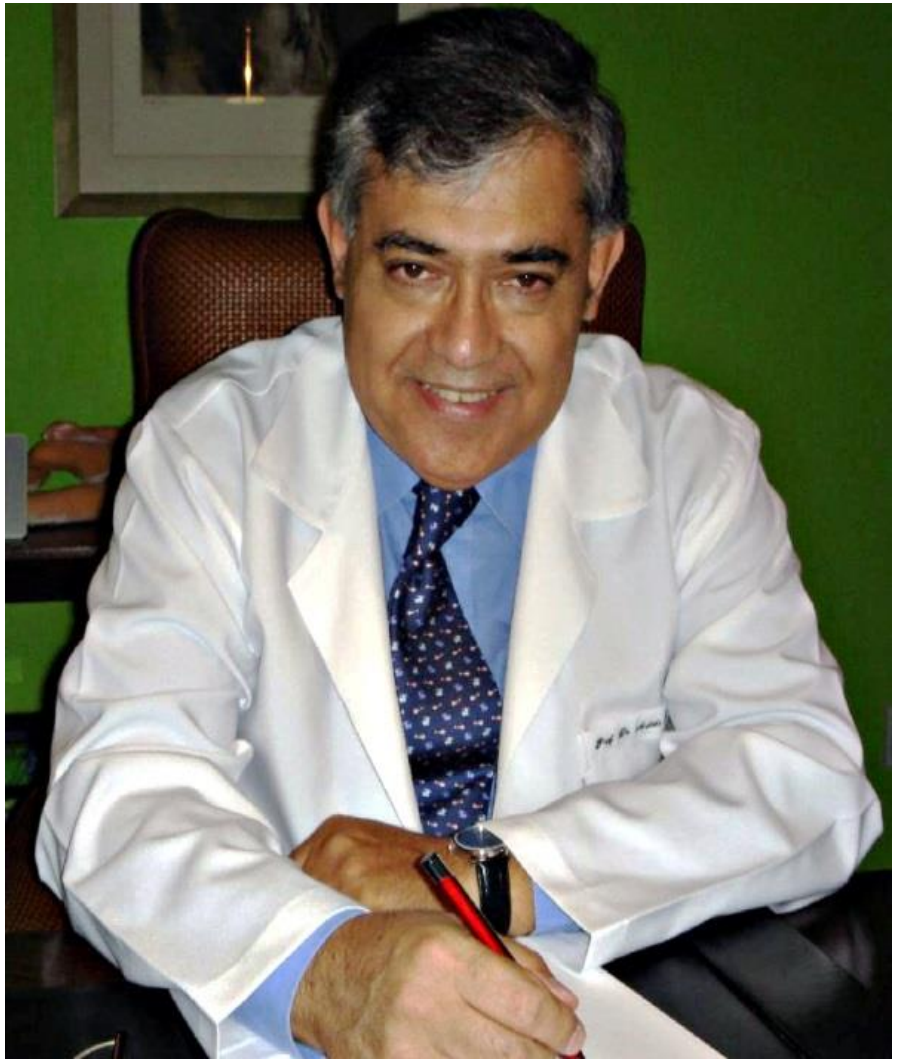
Final comments



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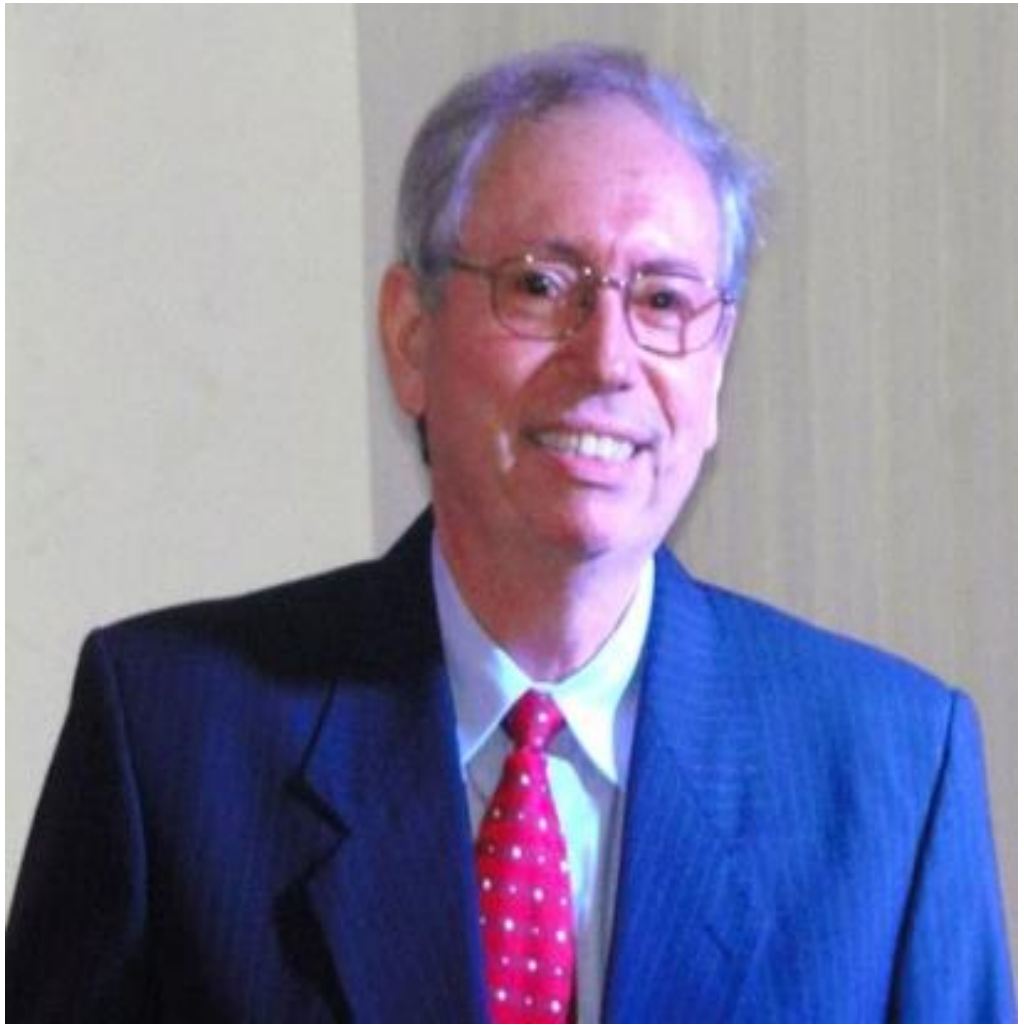

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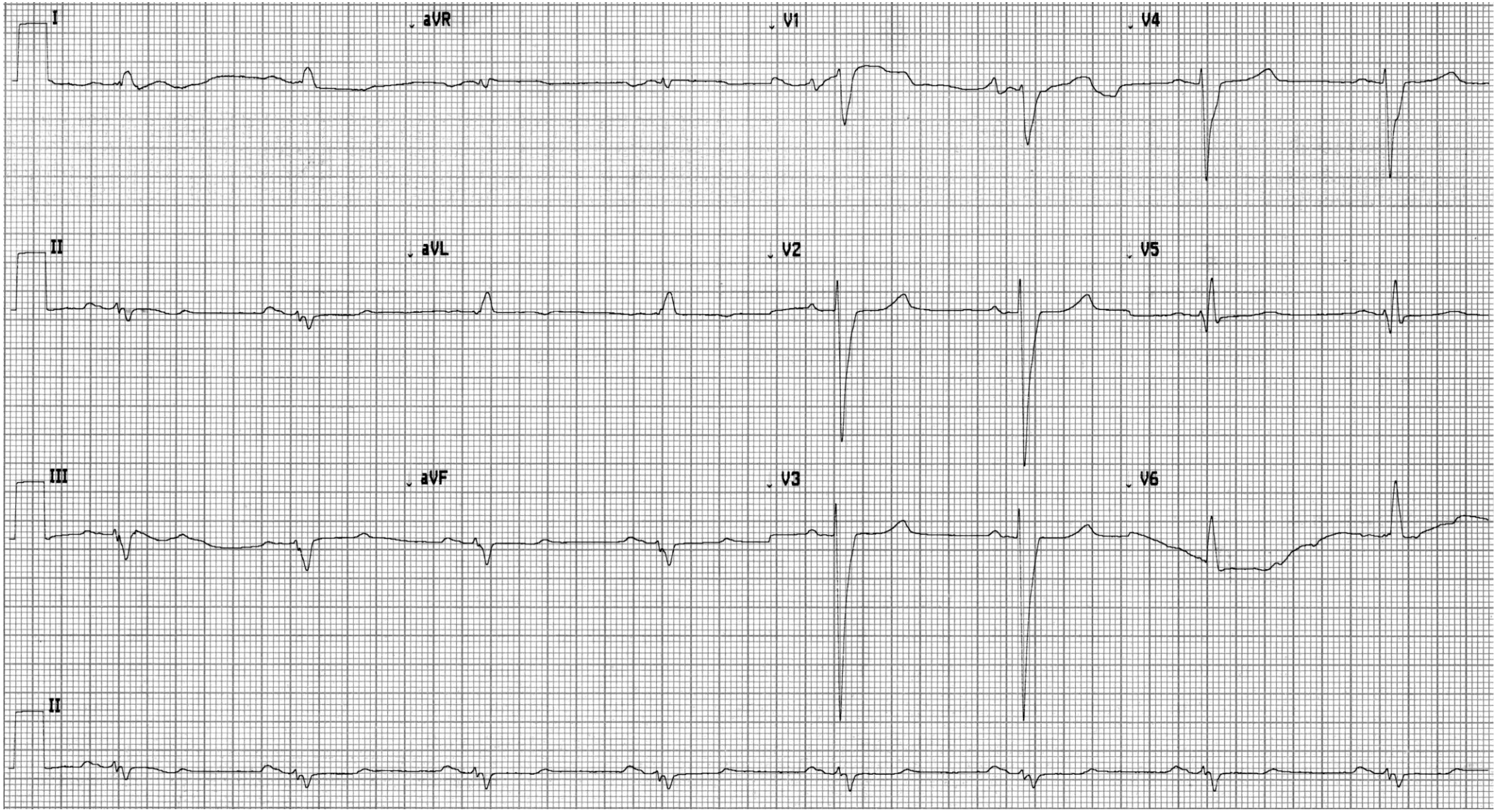
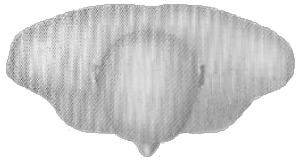


ECG diagnosis: SÂP +62°; PR interval 210 ms; QRS duration 156 ms; QRS axis -53°; QT/QTc 551/514 ms; SÂT +80°. Please see next slides….

Normal P-loops shape in the three planes

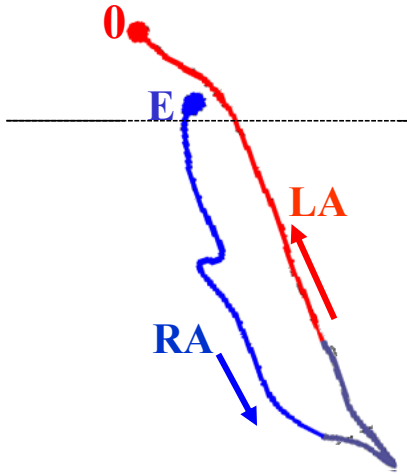
Normal atrial T vector (Ta vector): atrial repolarization (Ta vector) is responsible for the P loop being open and not closed (0 point does not coincide with initial E point). A straight line from the onset of the P loop (E point or null point) to the end of it (0 point) indicates the magnitude and direction of the Ta or Tp vector.

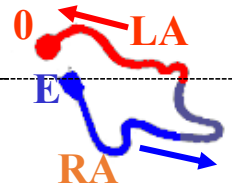
Frontal Plane

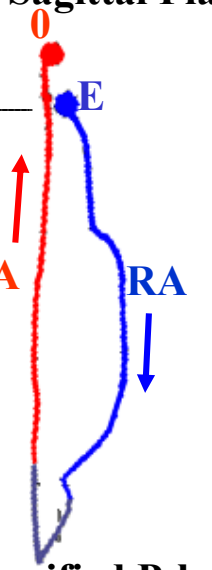


Horizontal Plane

Right Sagittal Plane

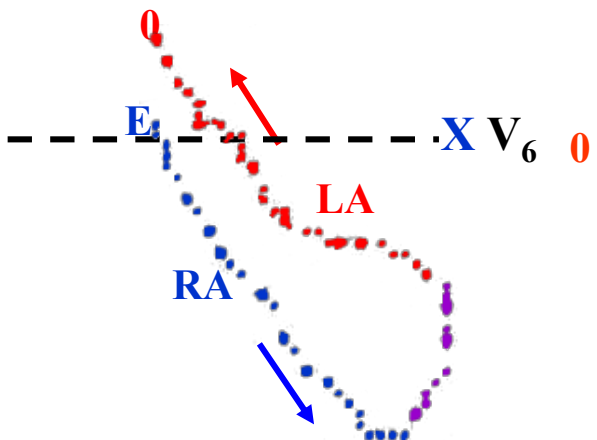






P-loops shape in the present case

Magnified P-loop



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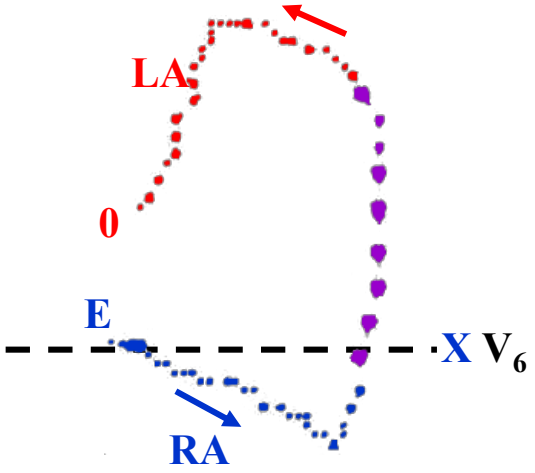
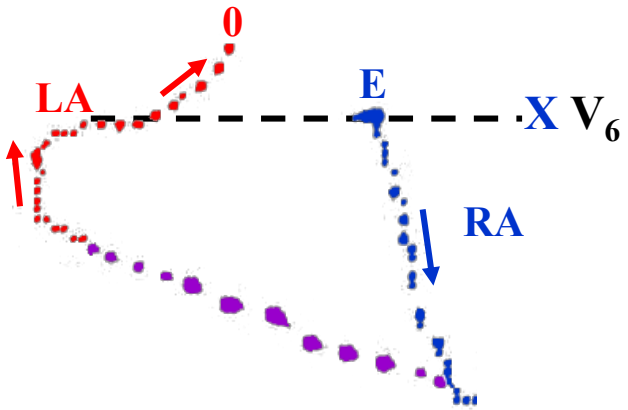
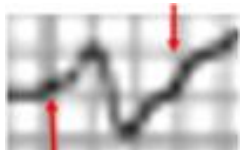
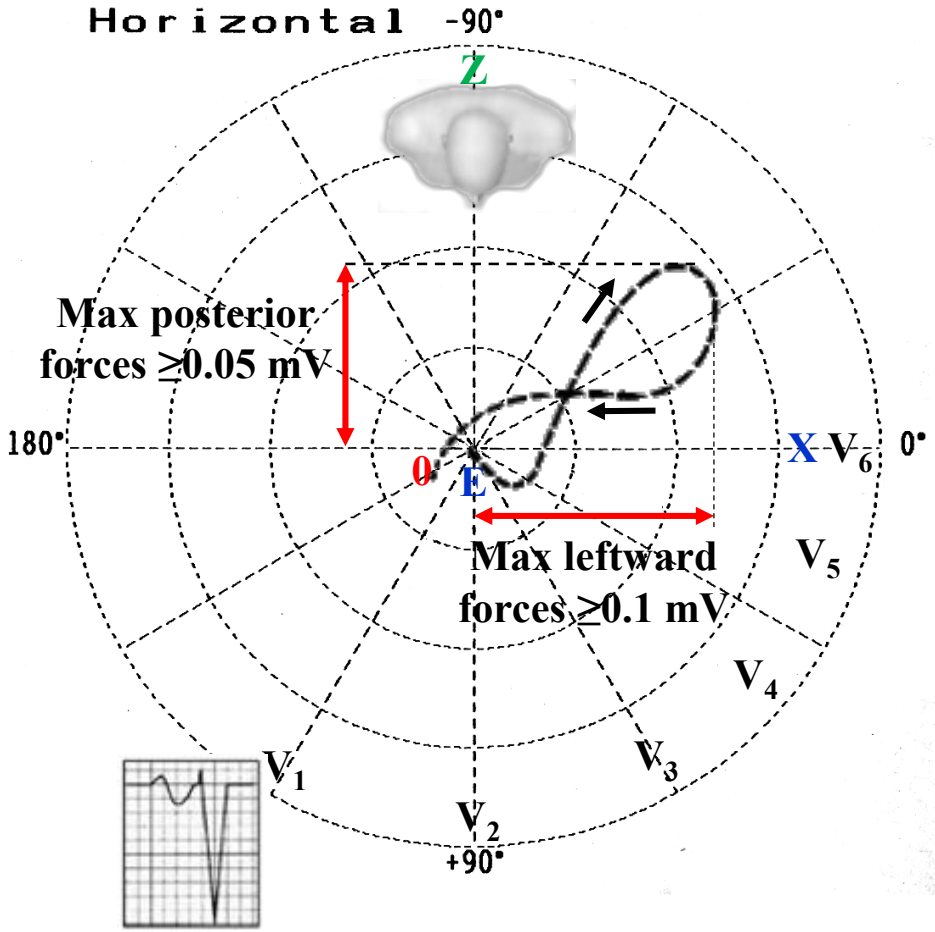


Magnified P-loop

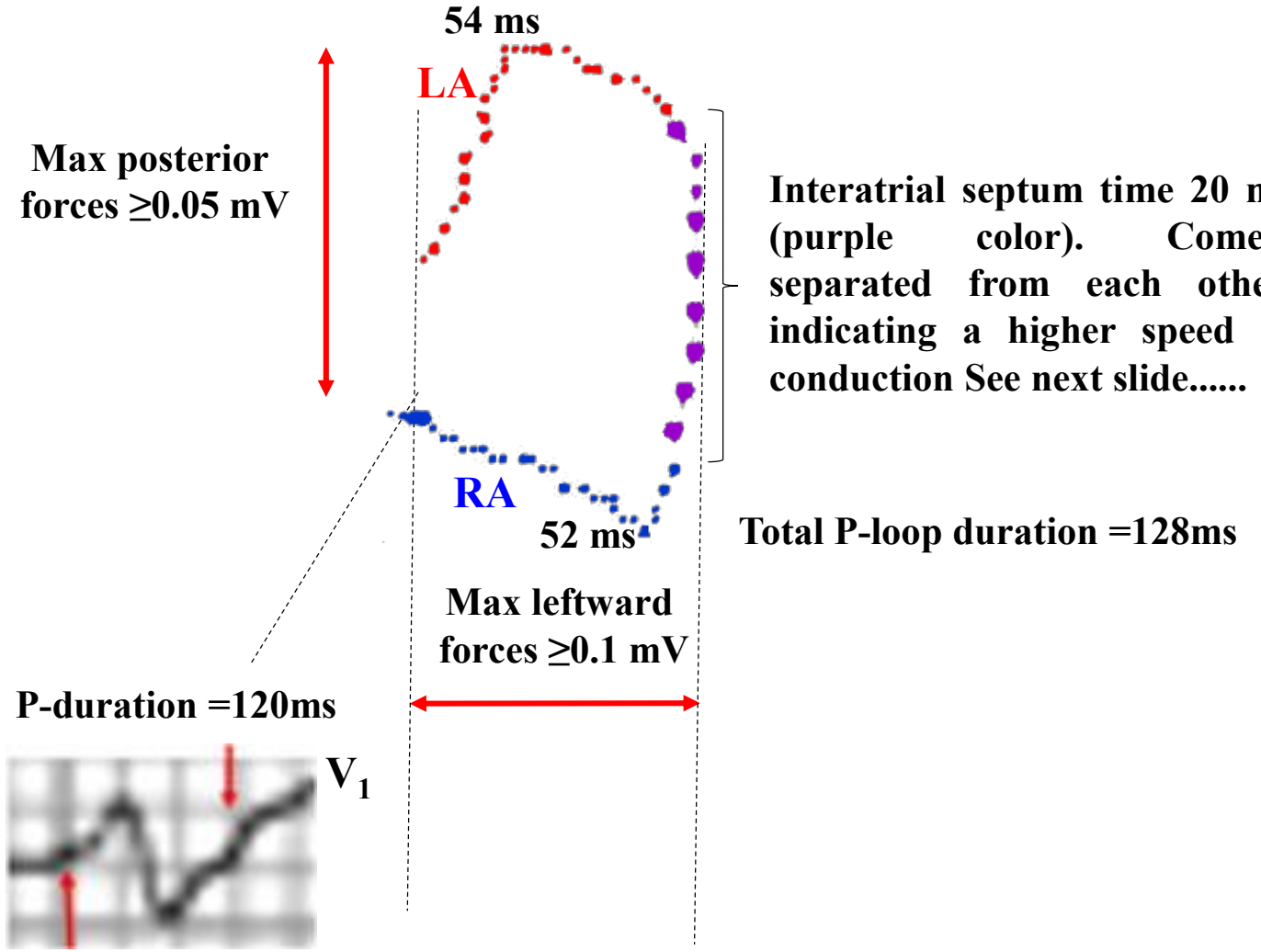




P-loop morphology and rotation in the HP in cases of isolated Left Atrial Enlargement (LAE)



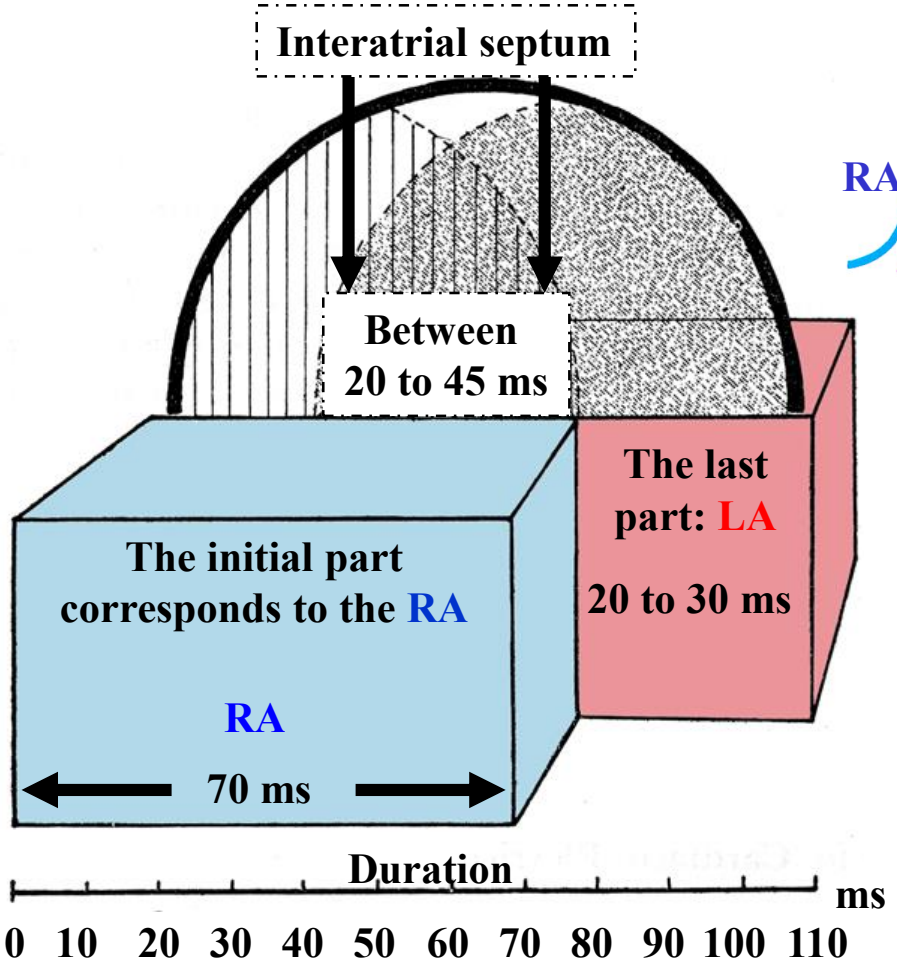
Magnified P-loop/P-wave in the present case



P-loop criteria of Left Atrial Enlargement (LAE) on Horizontal Plane or Transversal Plane

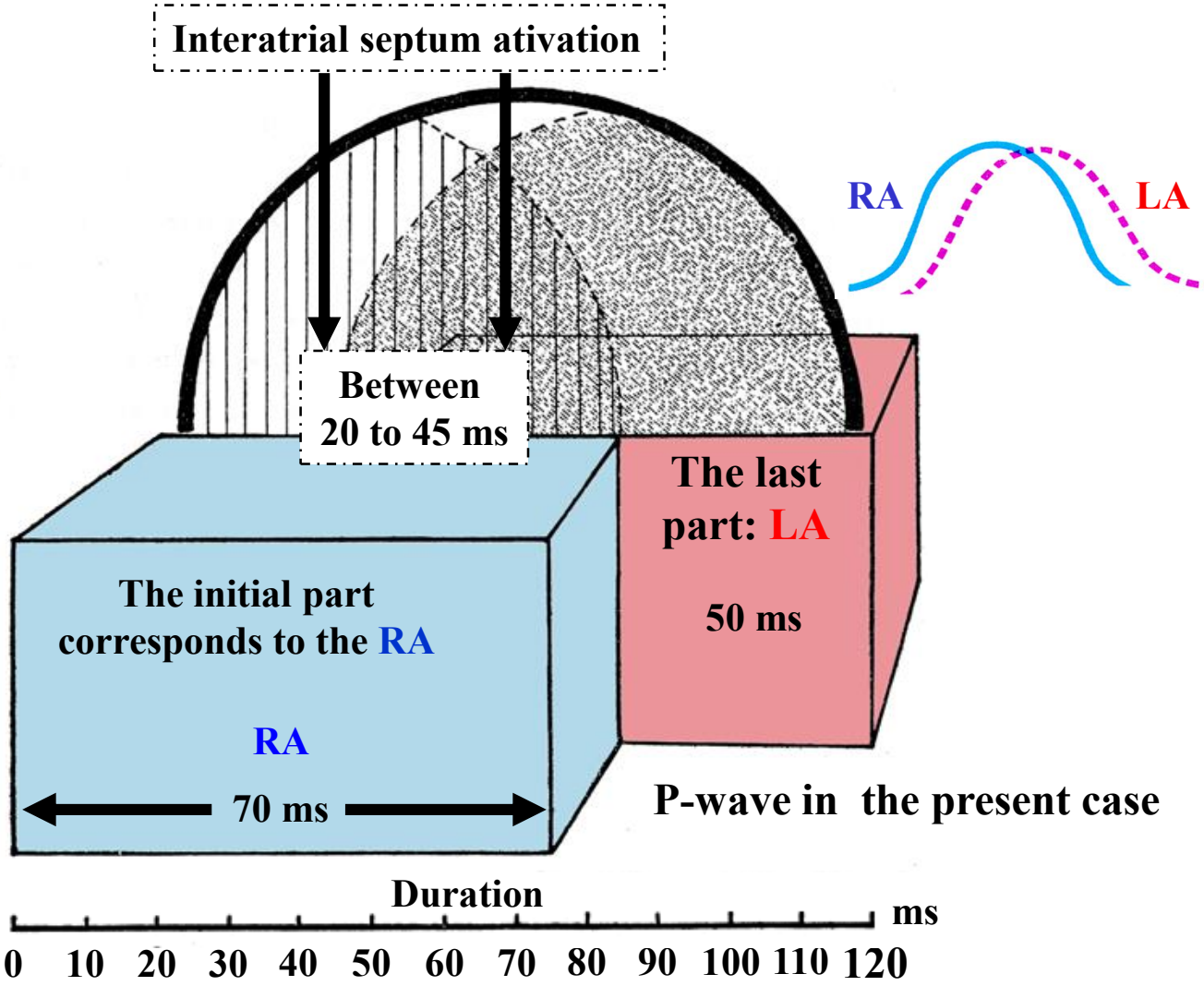
- The maximal posterior forces of the P-vector located to the left posterior quadrant: ≥0.10 mV in adults and ≥0.14 mV in <16 years old
- Max leftward forces ≥0.1 mV
- P-loop with frequent figure in 8 ("Bow Tie" morphology) with the last portion **(LA)** of the P-loop dislocated to back ≥0.05 mV

Normal activation of biatrial chamber

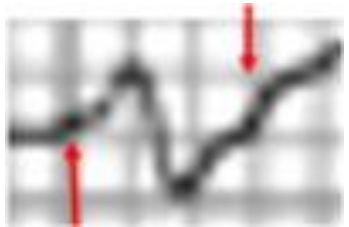


Normal duration of the P wave = 80 to 110 ms

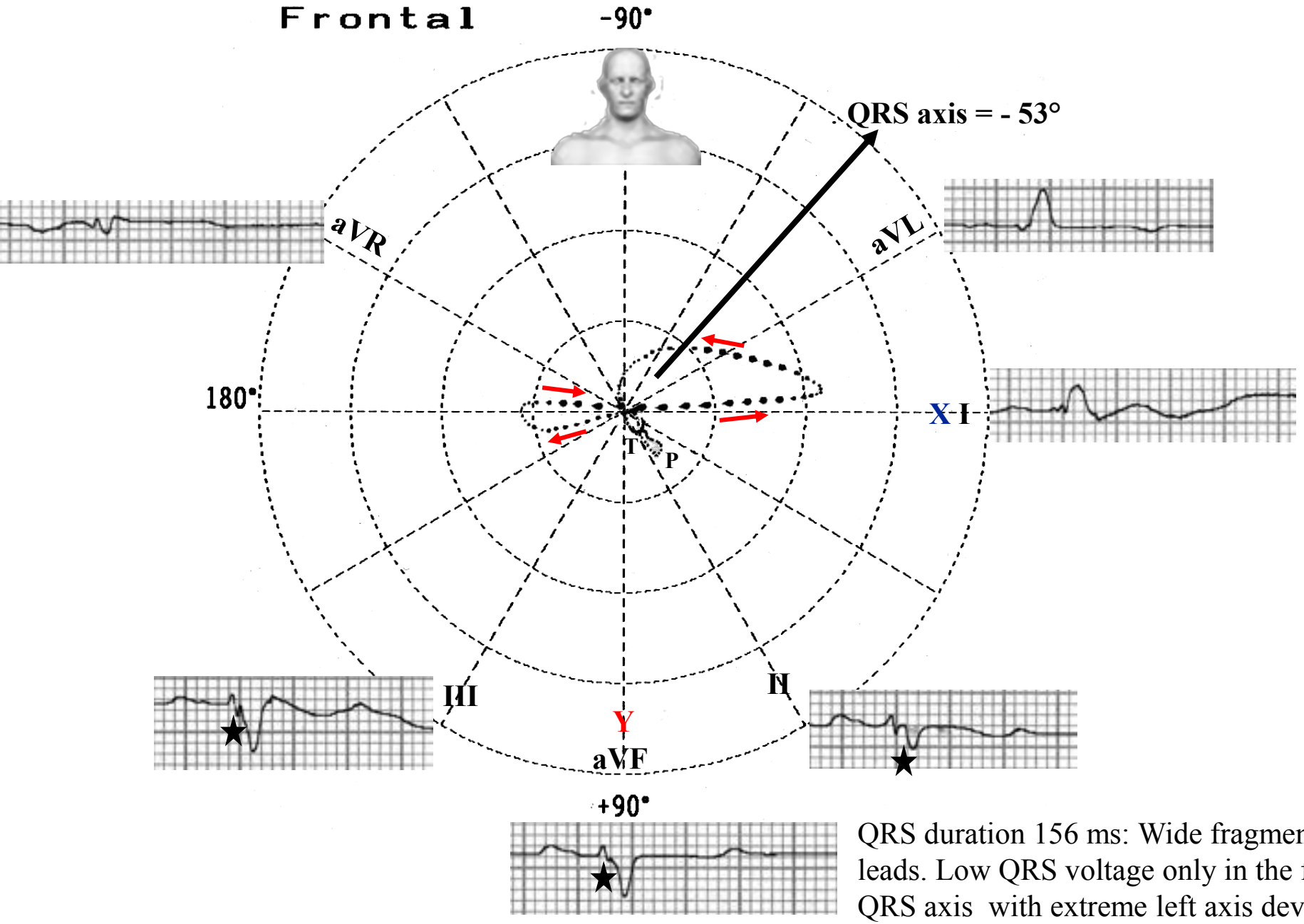
Prolonged P-wave duration in the present case



P-duration =120ms

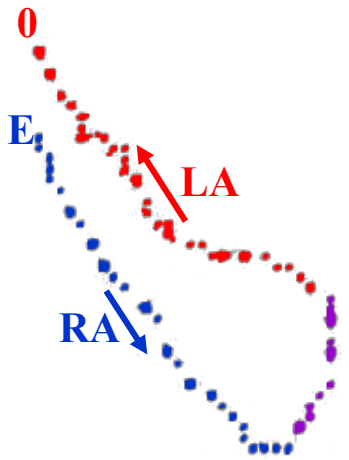


ECG/VCG correlation in the Frontal Plane



Sensi.	4
Timer	2 msec
Loop	All Loop
Sagittal	Right
Z Axis	Front
Filter	Hum
	Muscle
	Drift

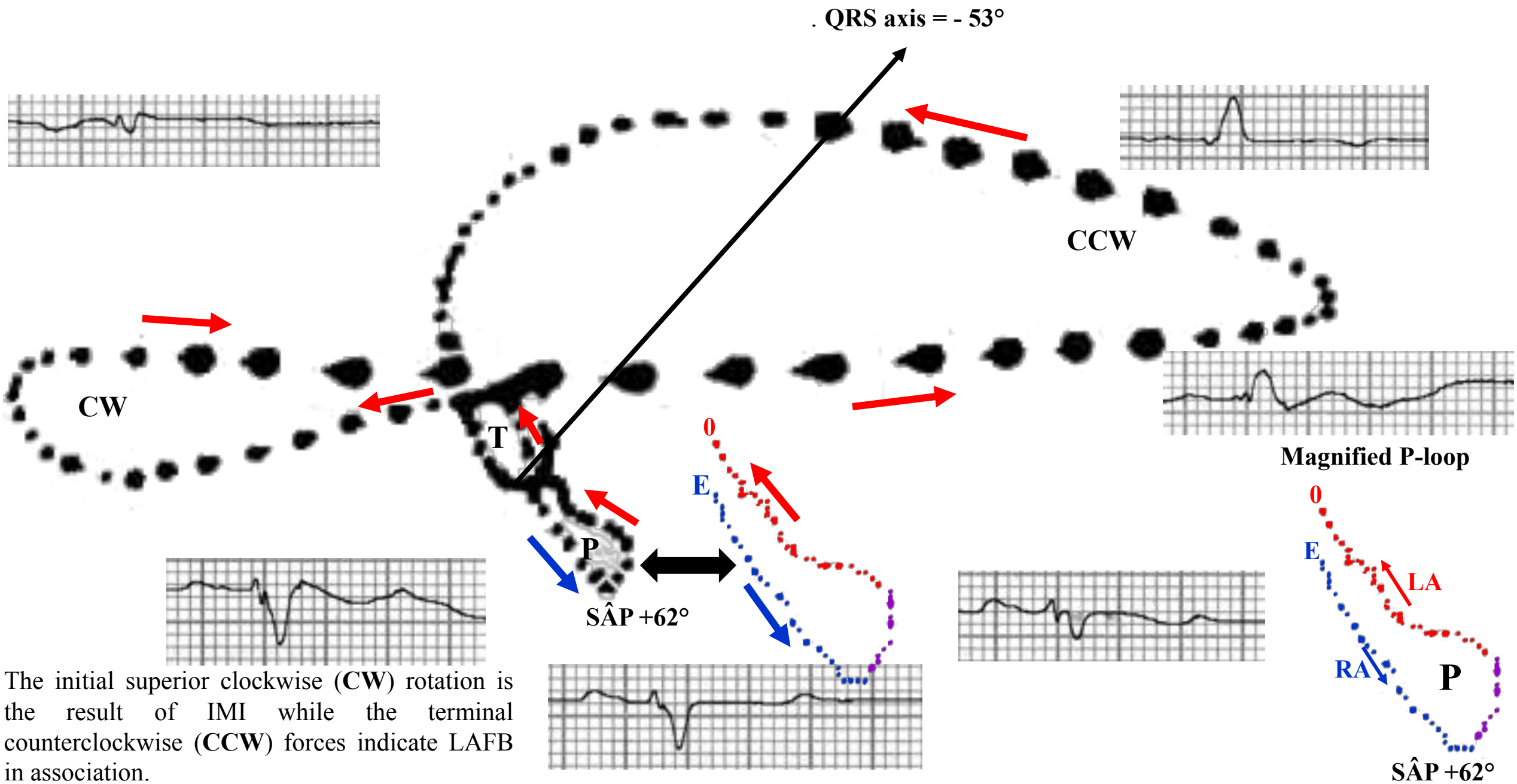
Magnified P-loop



SÂP +62°

QRS duration 156 ms: Wide fragmented QRS (W-f-QRS★) in at least two leads. Low QRS voltage only in the frontal plane.
QRS axis with extreme left axis deviation = -53° consequence of inferior inactive area associated with LAFB?

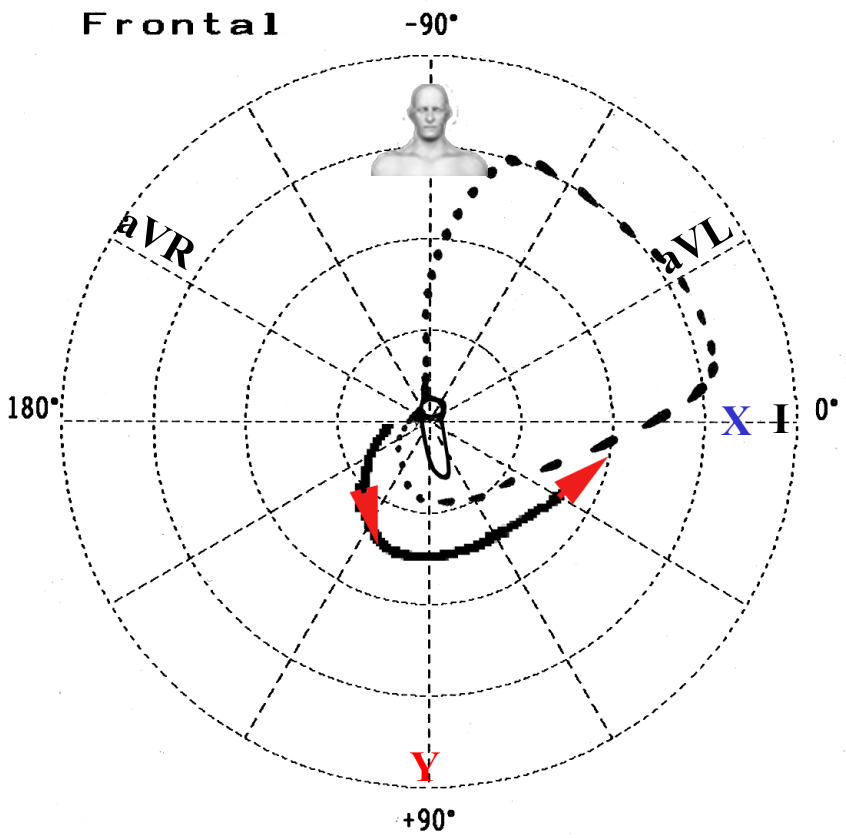
Magnified ECG/VCG correlation in the Frontal Plane



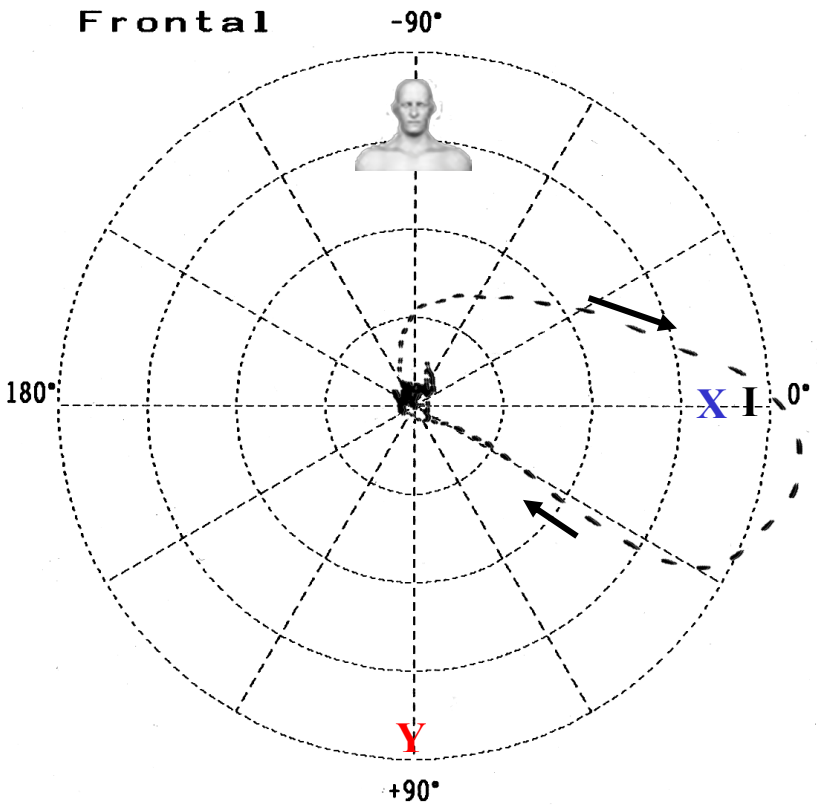
The initial superior clockwise (**CW**) rotation is the result of IMI while the terminal counterclockwise (**CCW**) forces indicate LAFB in association.

QRS loop in LAFB; isolated inferior MI and LAFB associated with inferior MI

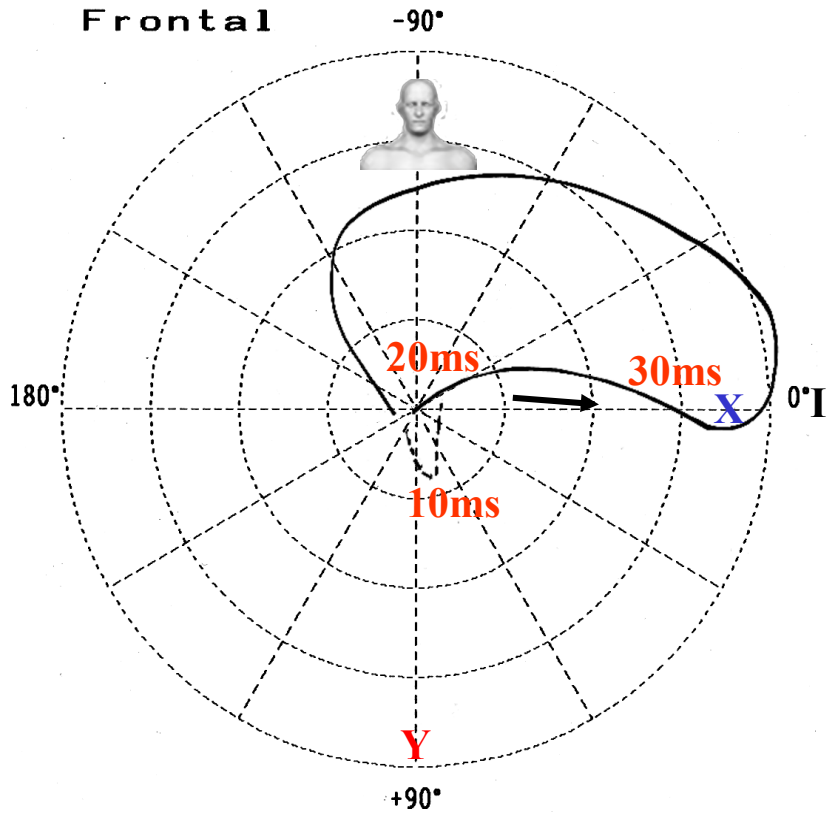
LAFB



Isolated inferior MI

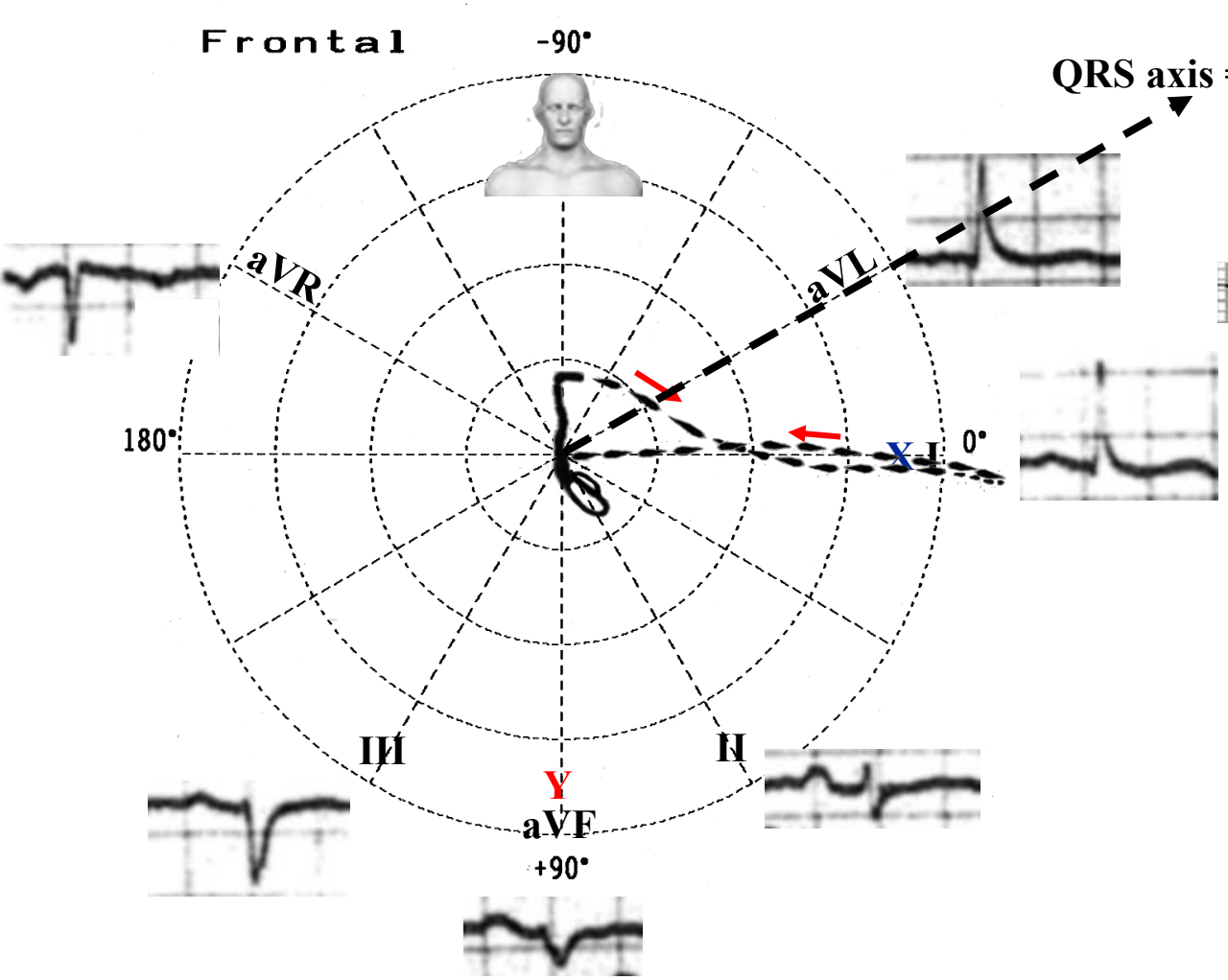


LAFB + inferior MI

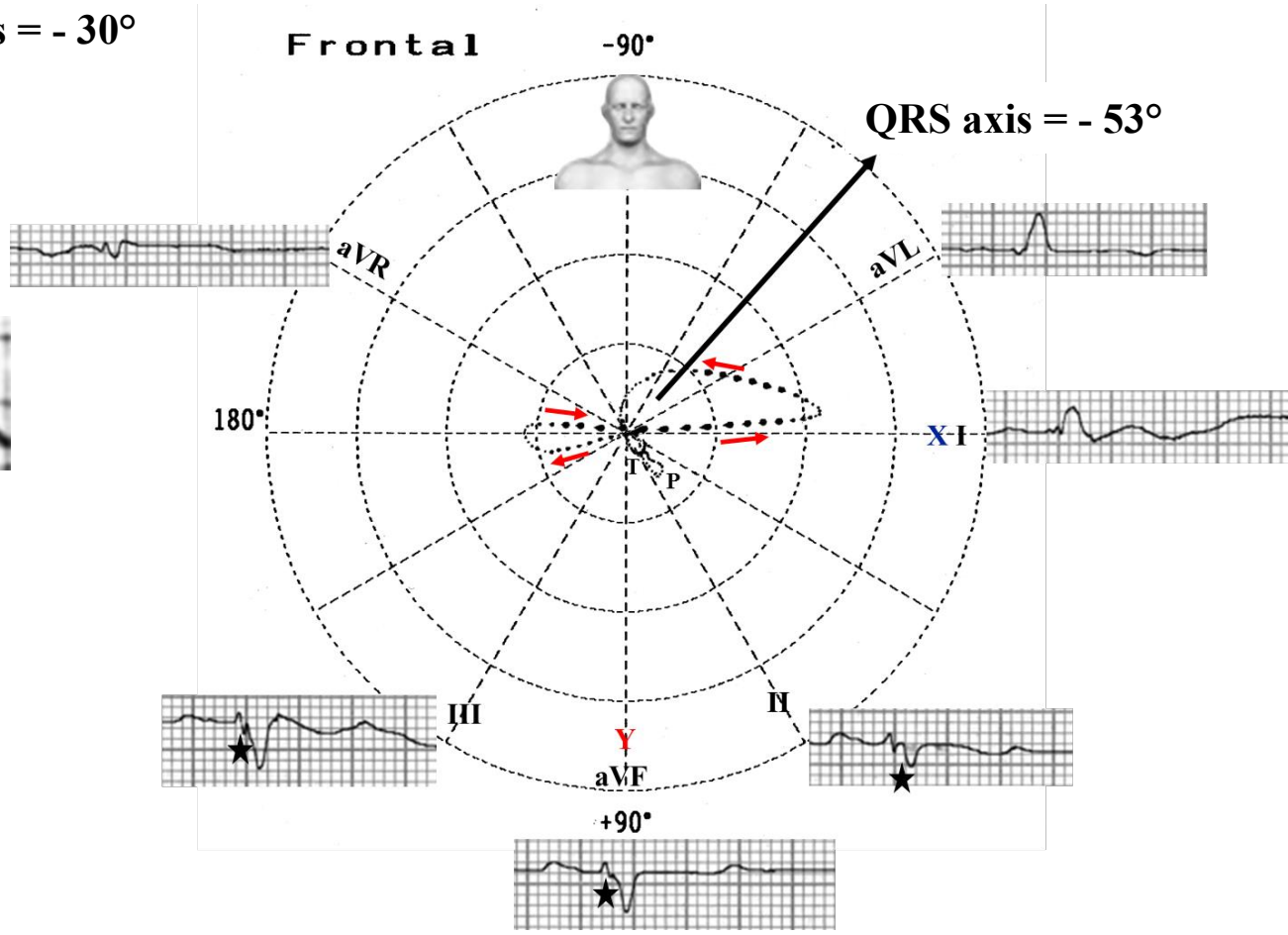


The initial 20 to 30ms forces are displayed superiorly and rotated CW. The remainder of the loop is also displaced superiorly but is rotated CCW. Occasionally the very early QRS vectors (10ms) may be directed inferiorly producing tiny r waves in inferior leads, but the reminder of the loop is displaced superiorly.

Inferior MI + LAFB ECG/VCG correlation in the Frontal Plane (Louridas 1981)



ECG/VCG correlation in the Frontal Plane in the present case: Inferior MI + LAFB



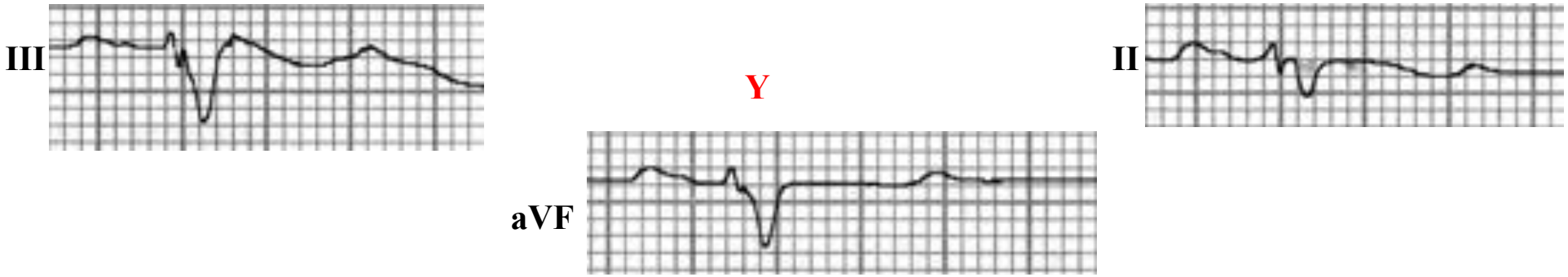
The initial superior clockwise rotation is the result of IMI while the terminal counterclockwise forces indicate LAFB.

LAFB associated with inferior myocardial infarction

- In the frontal plane we observe LAFB associated with inferior myocardial infarction (MI) when the initial 20 to 30 ms vectors are displayed superiorly and rotated CW.

- The remaining QRS loop is also displaced superiorly but is rotated CCW. Sometimes, the first 10 ms vector may be directed inferiorly producing an initial r wave in inferior leads, (as this case).

- When this occurs, an rS pattern may be seen in the inferior leads. So, inferior infarction is masked by preexisting LAFB by causing an initial r wave in the inferior leads .



Others clues for the ECG diagnosis of LAFB associated with inferior MI

- A qrS complex or tiny, bifid or notched initial R wave in lead II is a strong evidence of the diagnosis of LAFB in association with inferior MI

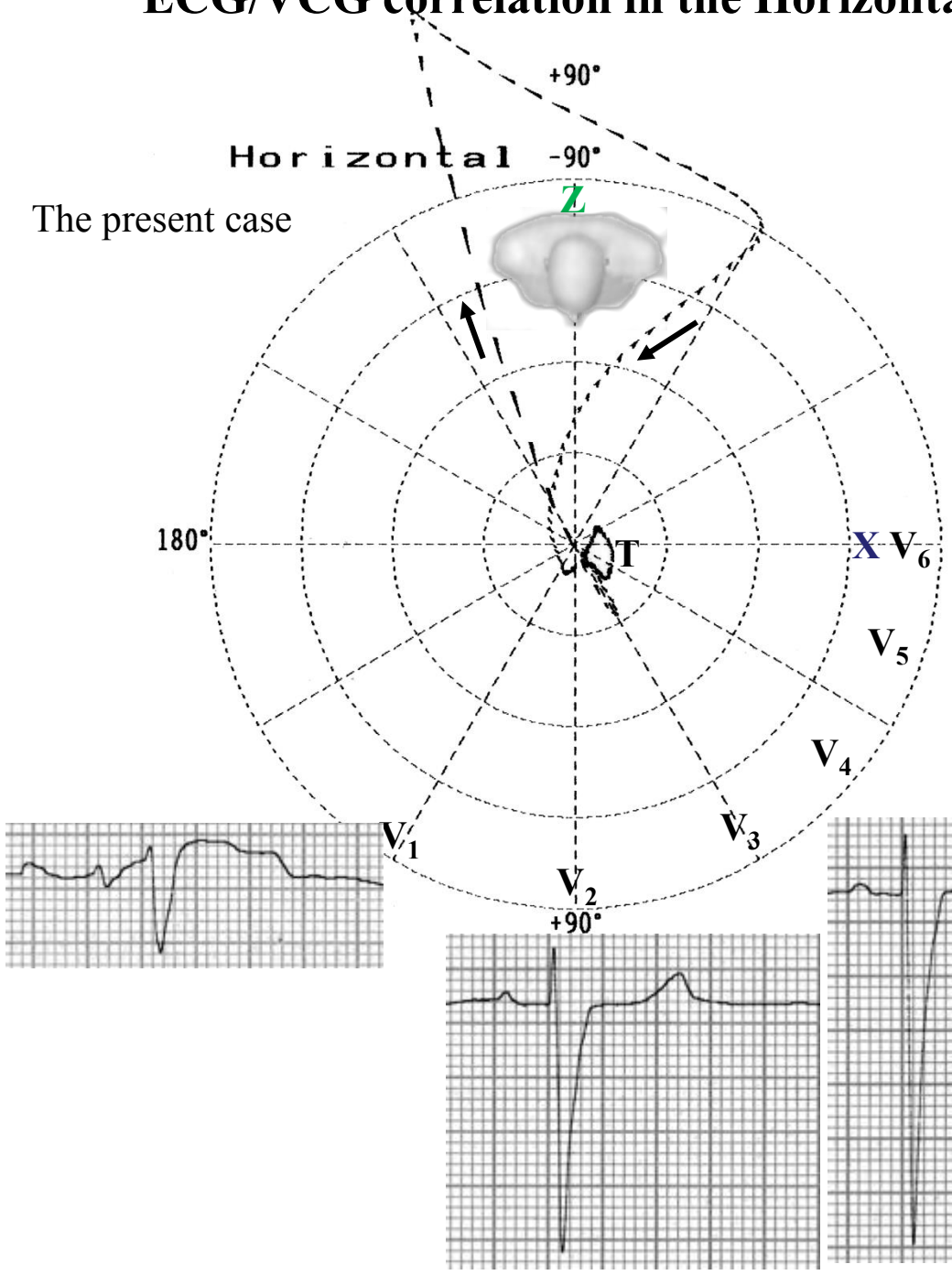
- Small Q waves preceding the rS complexes in leads III and aVF

- The presence of late R wave in inferior leads is strong evidence against the diagnosis of LAFB in association with inferior MI

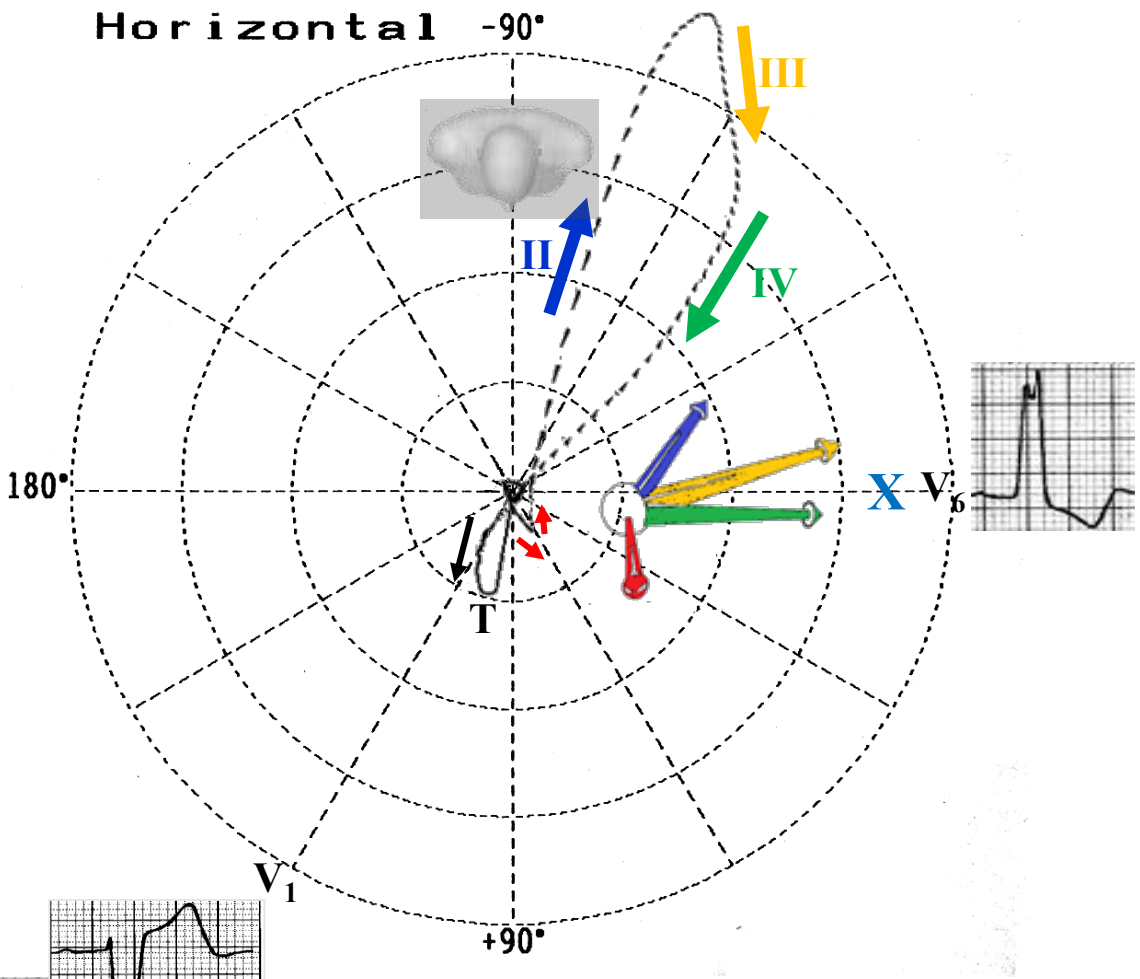
- Inferior MI with LAFB may be mimicked by chronic obstructive pulmonary disease .

ECG/VCG correlation in the Horizontal Plane comparative with truly Left Bundle Branch Block

The present case



truly Left Bundle Branch Block



See next slide.....

Sequence of septal ventricular activation/depolarization in uncomplicated LBBB and R-Wave Peak Time

There are six major components in ventricular activation processes in uncomplicated LBBB:

A. **Initial activation of the apicoanterior right ventricular wall:** Right inferior septal activation on the subendocardial region of the anterior papillary tricuspid muscle: **Vector I**: Directed anteriorly, inferiorly and to the left.

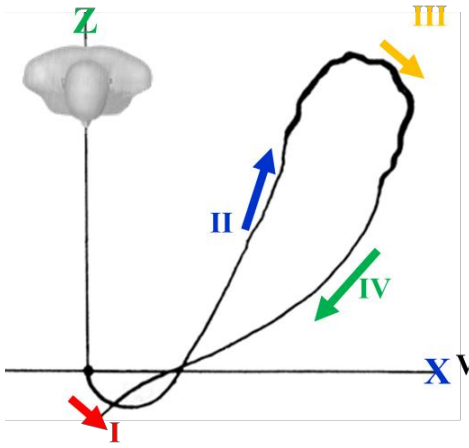
B. **Right-to-left septal activation and activation of right ventricular free wall:** Delayed and anomalous left septal activation: **Vectors II** and **III**: Directed posteriorly, superiorly and to the left.

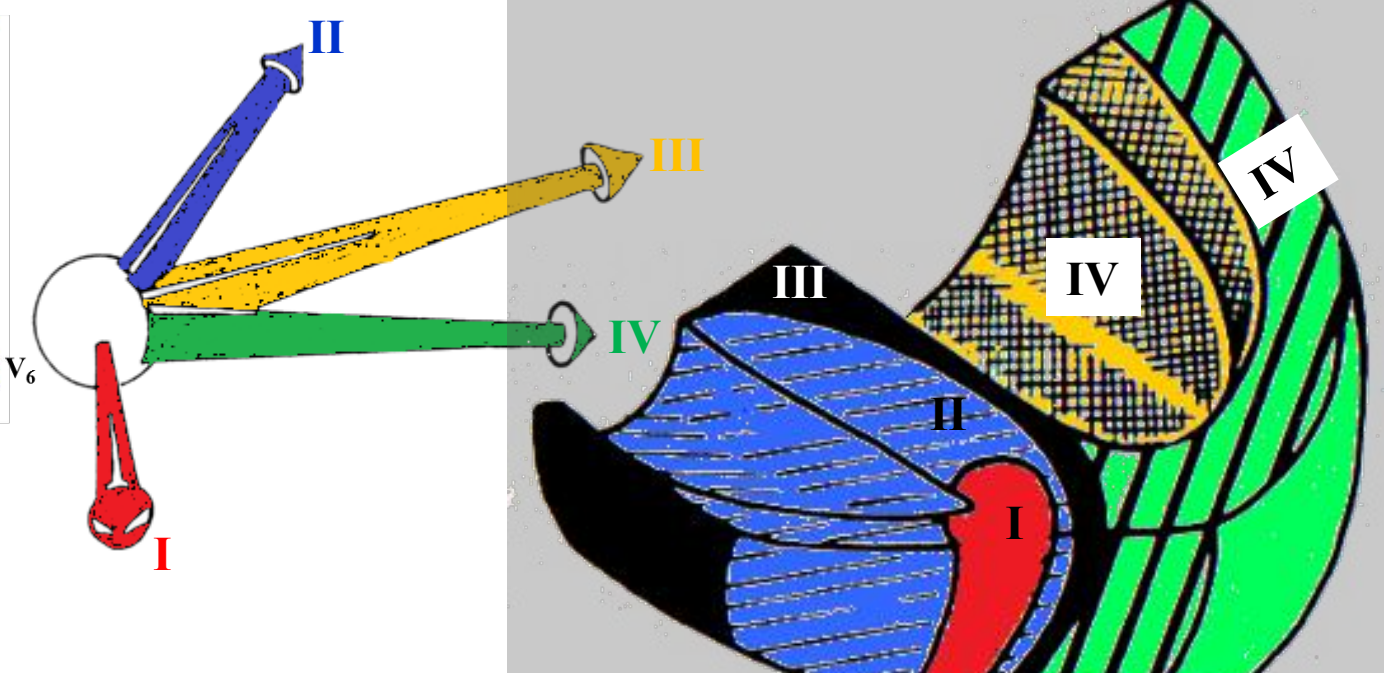
C. **Complementation of the septal and right ventricular activation** **III**

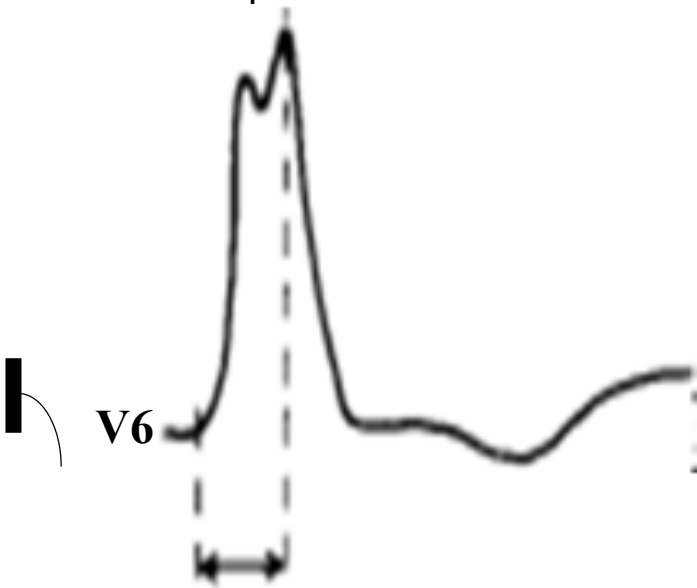
D. **Initial aberrant activation of basal left ventricular wall**

E. **Activation of the posterior, lateral and anterior left ventricular wall: Vector IV**

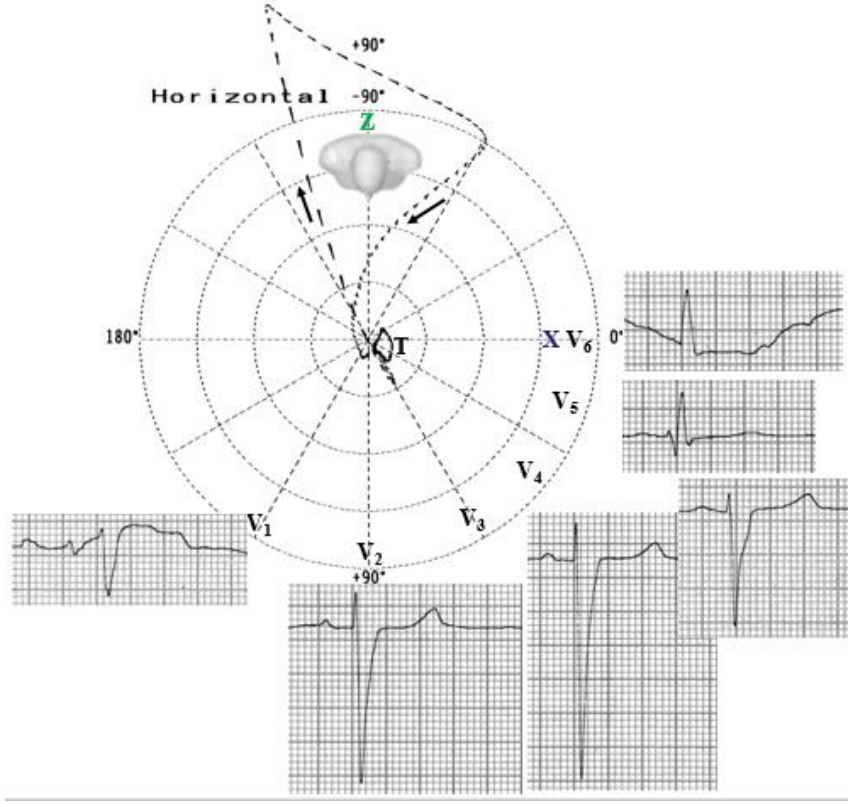
F. **Completion of the activation of the anterior wall of ventricle**: Delayed and anomalous activation of the free left ventricular wall: **Vector IV:** Directed posteriorly, superiorly and to the left. The last 50 ms is made up of activation fronts in the posterolateral wall of the left ventricle.



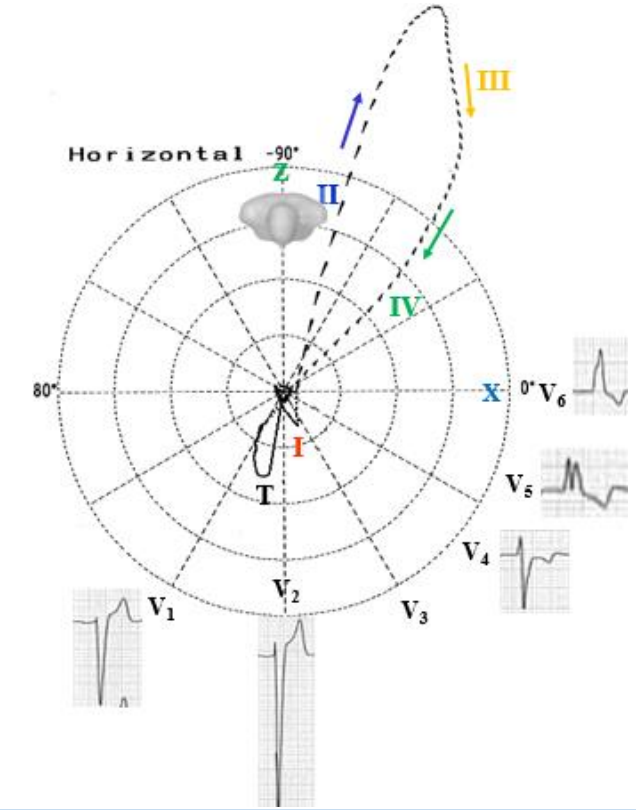




ECG/VCG correlation in the Horizontal Plane in the present case



ECG/VCG correlation in "genuine" LBBB in the HP

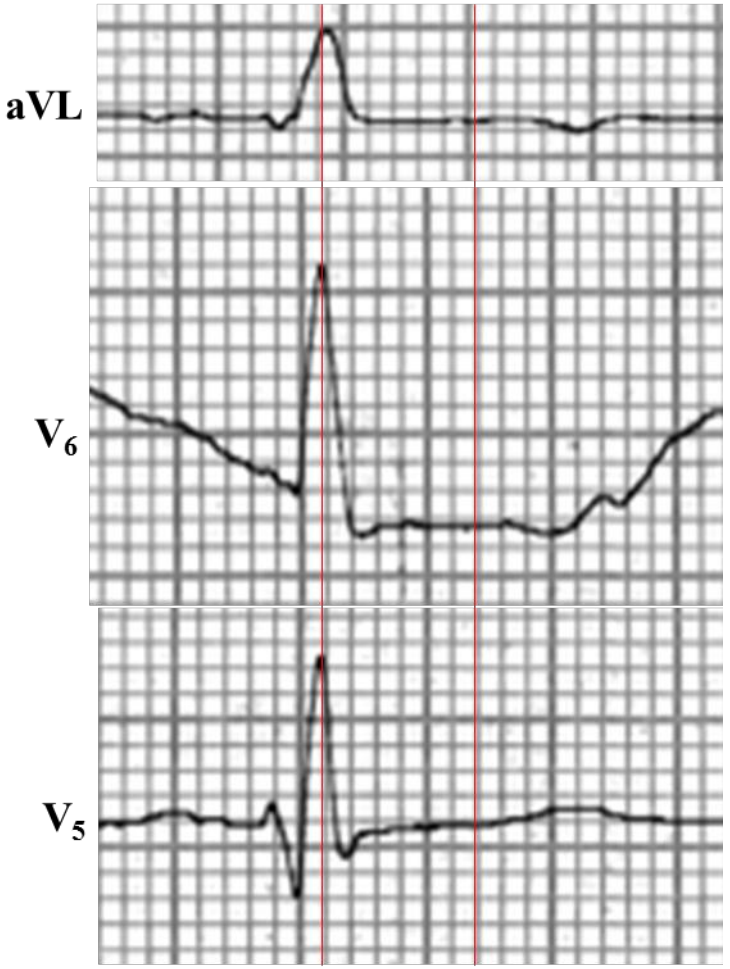


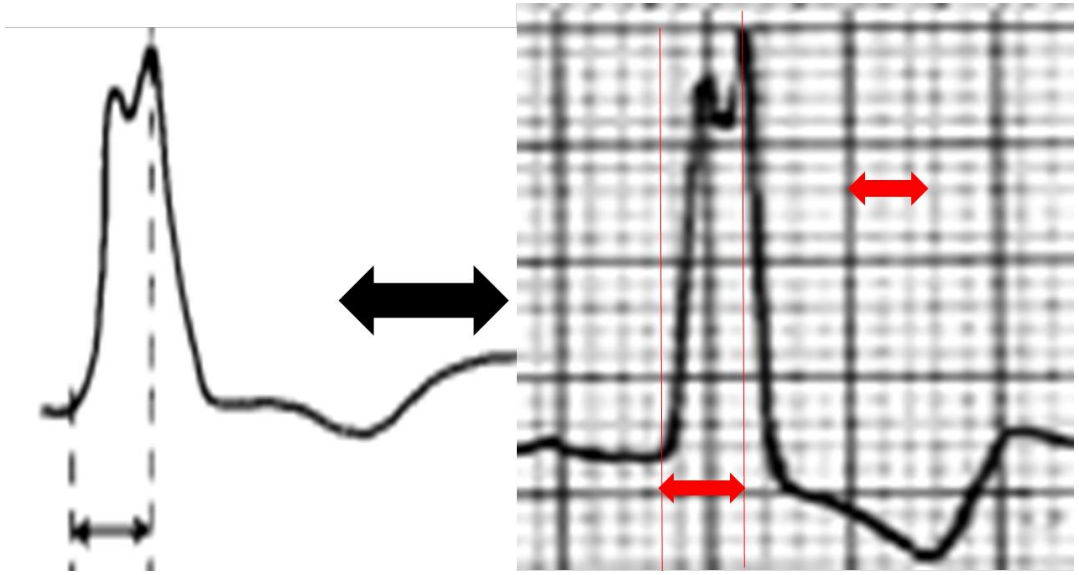
	The present case: Nonspecific intraventricular conduction delay (NSIVCD)	Truly or genuine LBBB
QRS-loop rotation	Main portions of QRS loop of clockwise rotation.	Main portions of QRS loop of clockwise rotation.
QRS duration	156 ms	≥ 120ms (following the conventional ECG criteria). Stricter criteria for complete LBBB that include a QRS duration ≥ 140 ms for men and ≥ 130 ms for women (**Strauss 2011**)
QRS- loop shape	Wide, triangular.	Narrow and elongated and with morphology usually in 8.
QRS Location	≈50% on right posterior quadrant and ≈50% on left posterior quadrant	On left posterior quadrant (on right posterior quadrant when associated with RVH)

	The present case: Nonspecific intraventricular conduction delay (IVCD or NSIVCD)	Truly or genuine LBBB
Maximal vector of QRS location	In the **right** posterior quadrant (≈ -105º) and of increased magnitude (>2 mV).	In the **left** posterior quadrant (between –40º to -80º) and of increased magnitude (>2 mV).
Efferent/afferent lib relationship	The efferent limb located to right related afferent limb.	The efferent limb (**II**) located to right related afferent limb (**III** and **IV**).
Conduction delay inside of the QRS loop	In the middle and terminal portion (comets closest to each other).	In the middle and terminal portion (comets closest to each other).
ST and T-loop vector	Directed leftward and anteriorly. Small T-loop with symmetric afferent/efferent limbs: **Primary T-loop**.	Directed rightward and anteriorly. T-loop of counterclockwise rotation and with asymmetrical afferent/efferent limbs: **Secondary T-loop**.
LV Electrical Interval (Q-LV): Represents the time taken for excitation to spread from the endocardial to the epicardial surface of the LV. The time from the onset of the earliest Q or R wave to the peak of the R wave in the lateral leads aVL, V5-V6.	Wide range. Only prolonged if: ➢ Mid-QRS notching/slurring in lateral leads or ➢ QRSd >150ms; or ➢ R-Wave Peak Time > 60ms. R-wave peak time is said to be *prolonged* if > 45ms(in case of LBBB >60ms). Mid-QRS notching in lateral leads strongly predicts a longer Q-LV interval in L-IVCD patients.	Very prolonged Q-LV interval (**Pastore 2016**) always > 110ms. These patients are responders to CRT: defined as a reduction in NYHA functional class by ≥1 grade (favorable clinical response in 70% after 6 months of CRT), quality-of-life score, exercise capacity expressed as 6-min walking distance; a reduction in LV end-systolic volume ≥15% or reversed LV remodeling. Cardiac resynchronization therapy (CRT) is recommended for HF patients who remain in symptomatic NYHA class III or IV despite optimal medical treatment, with normal sinus rhythm, low LVEF (35%), LV dilation, and QRS duration > 120 ms (**Vardas 2007**).

	The present case: Nonspecific intraventricular conduction delay (IVCD or NSIVCD)	Truly or genuine LBBB
Comparison between NSIVCD and LBBB	12.5% (**Sandhu 2004**)	22%
R-Wave Peak Time in left leads	Variable broad range	Prolonged always >60 ms

R-Wave Peak Time = 100ms





R-Wave Peak Time

R-Wave Peak Time = 120ms

R-Wave Peak Time = 120ms. The time from the onset of the earliest Q or R wave to the peak of the R wave in the lateral leads aVL, V5-V6. R-Wave Peak Time is said to be prolonged if > 60ms in cases of LBBB. Represents the time taken for excitation to spread from the endocardial to the epicardial surface of the LV.

Atypical LBBB (Eschalier 2015)

In the NonSpecific Intraventricular Conduction Delay (NSICD) subgroup, one must include the appearance of atypical bundle branch block (BBB) after post–MI patients that corresponds to the probable existence of a true LBBB, in which the superposition of the electrical abnormality in relation to the necrotic area alters the typical LBBB pattern, characterized by:

1. Prolonged QRS (> 120 ms)

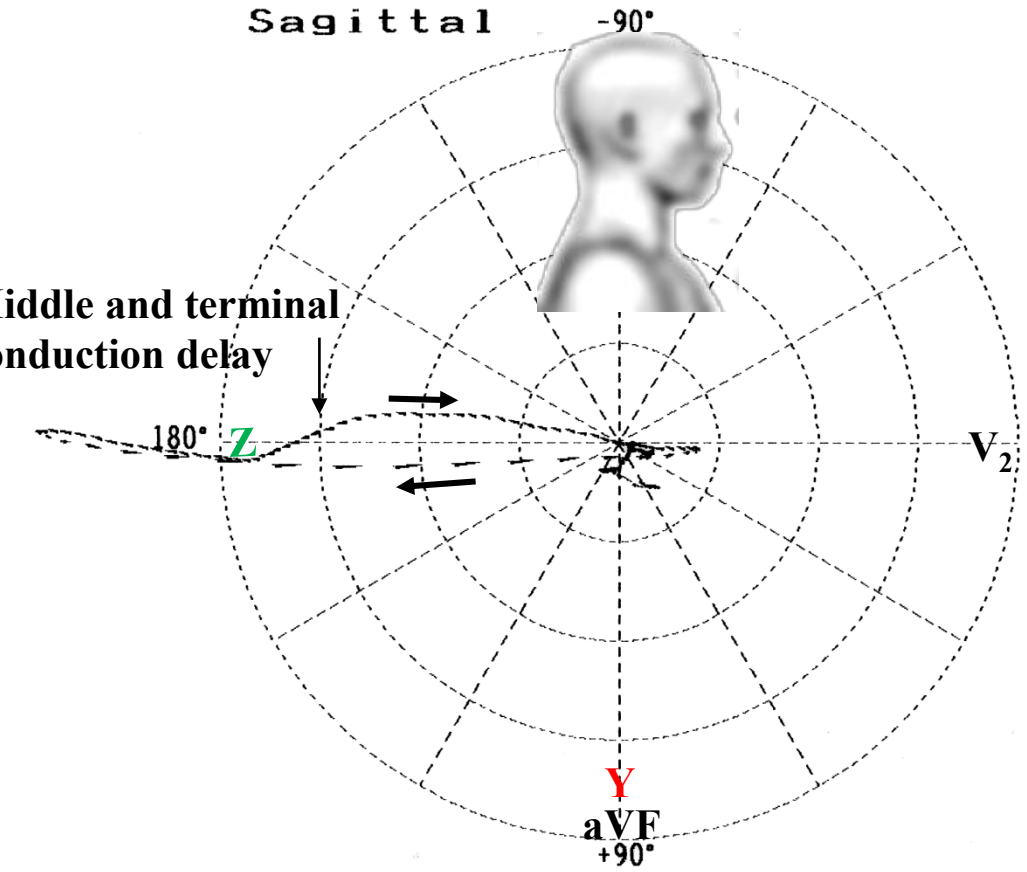
2. Deep Q waves in multiple leads in cases with massive MI or that affect multiple walls

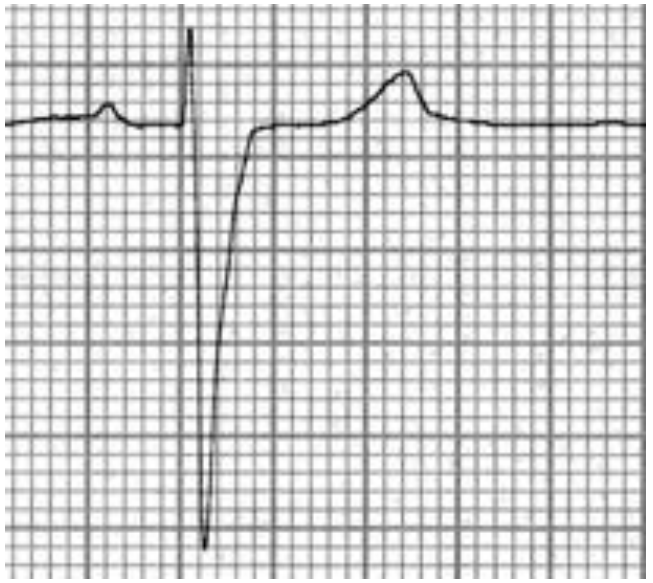
3. Wide fragmented QRS (Wf-QRS) related to scar in patients with CAD (**Das 2008**)

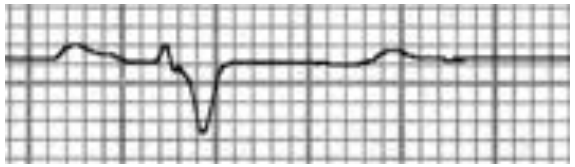
4. QS pattern in the anterior leads and QR wave in lateral leads after an anterior or lateral MI

5. The ventricular activation sequence should be very close to that observed in patients with genuine or typical LBBB such us the present case.

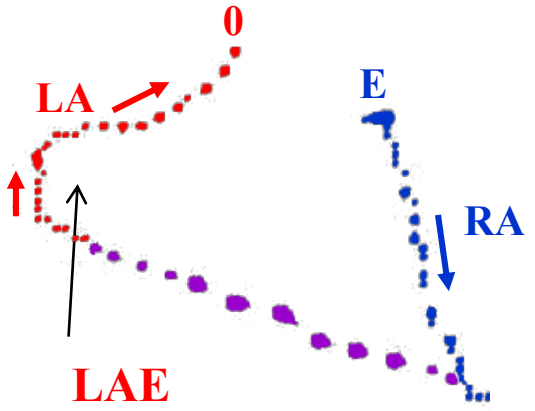
ECG/VCG correlation in the Right Sagittal Plane



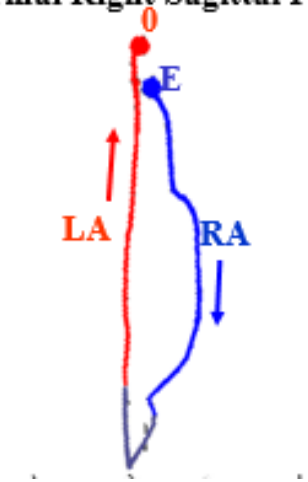




Magnified P-loop

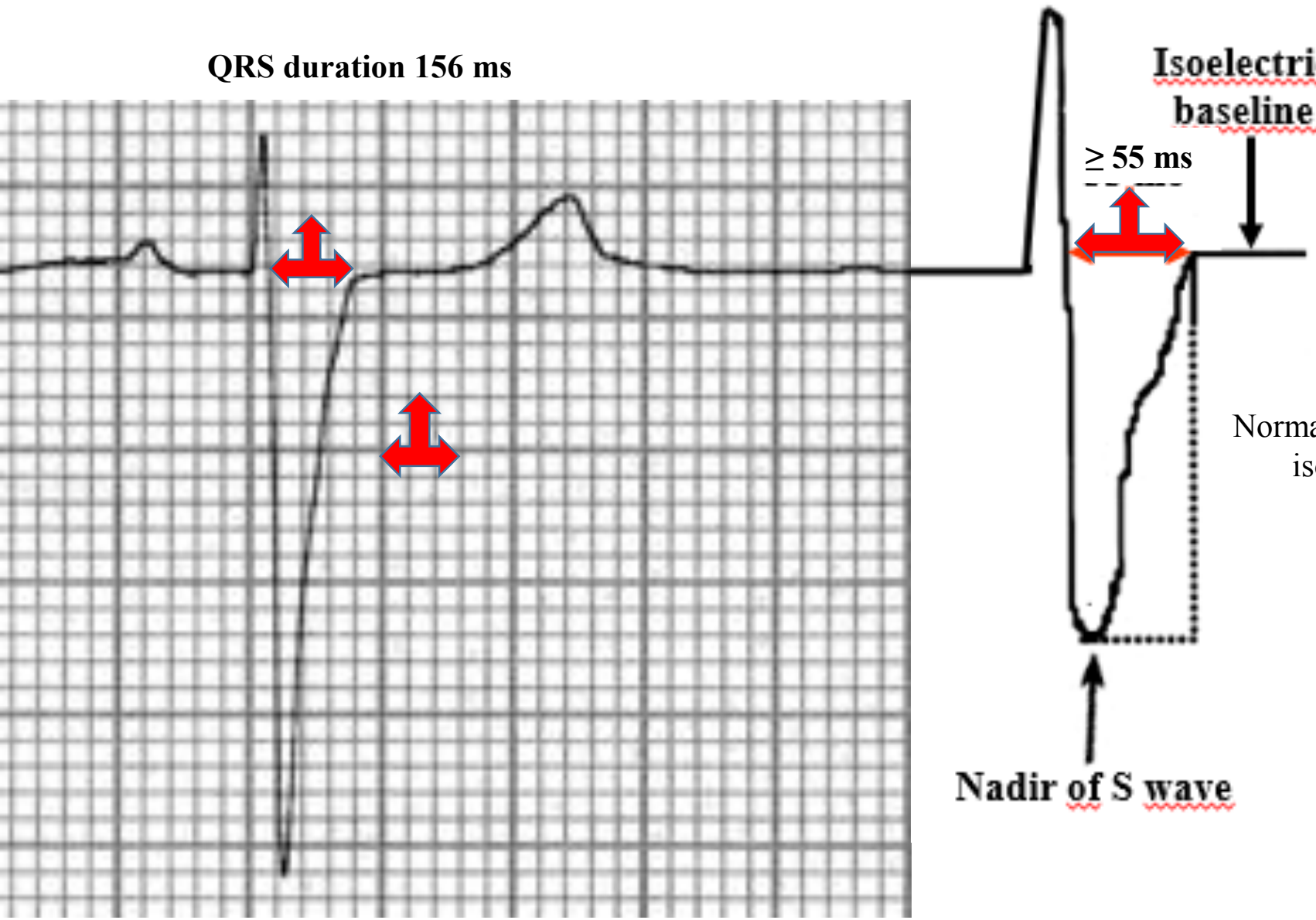


Normal Right Sagittal Plane



QRS perpendicular to frontal plane (Y orthogonal lead/ aVF) This QRS direction partially explain the low QRS voltage the leads of the Frontal Plane. rS pattern is suspected of inferior MI + LAFB

Very prolonged S-wave upstroke in V_1 through $V_3 \geq 55$ ms is correlated with disease severity and induction of VT on EPS.

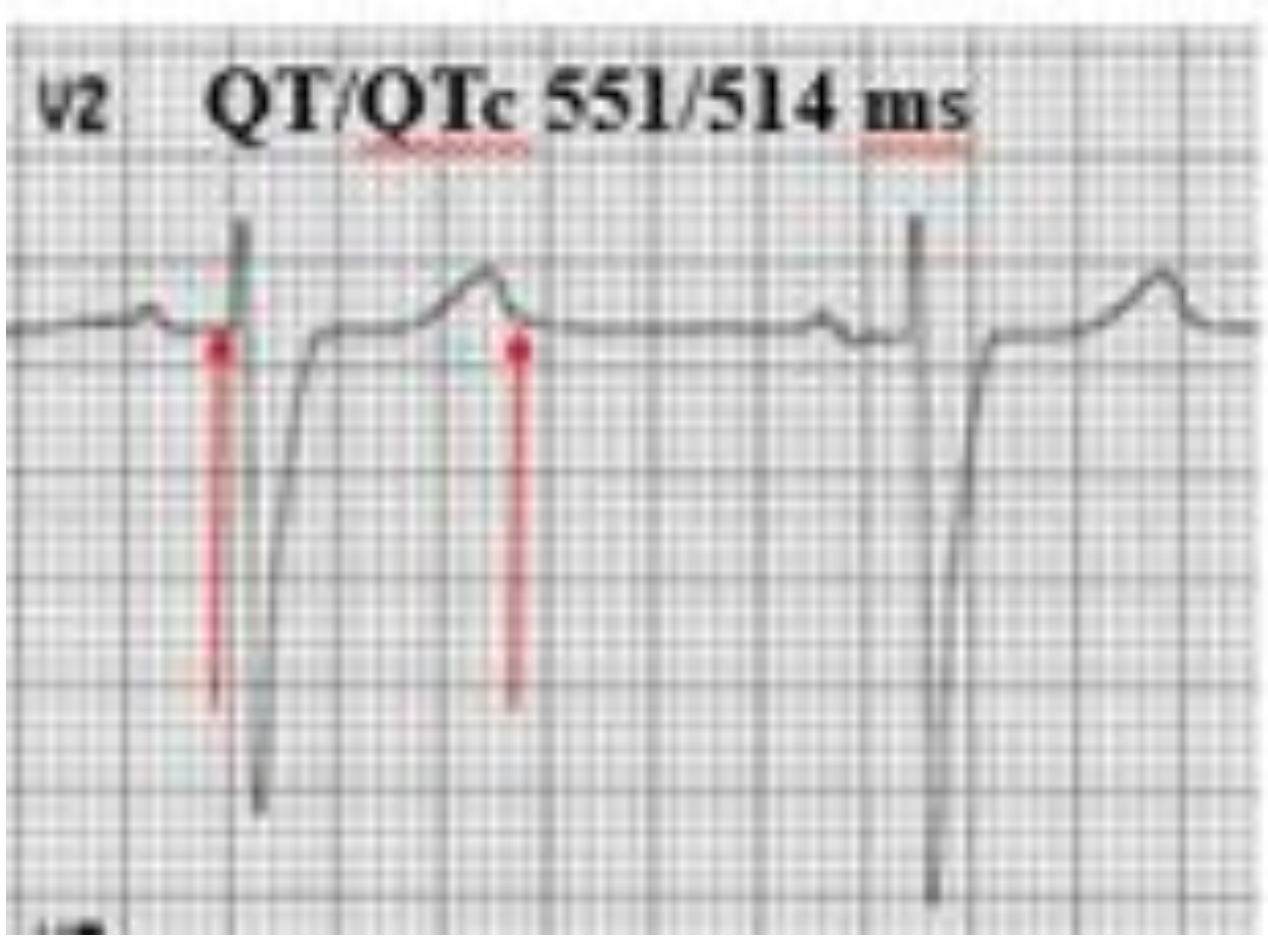


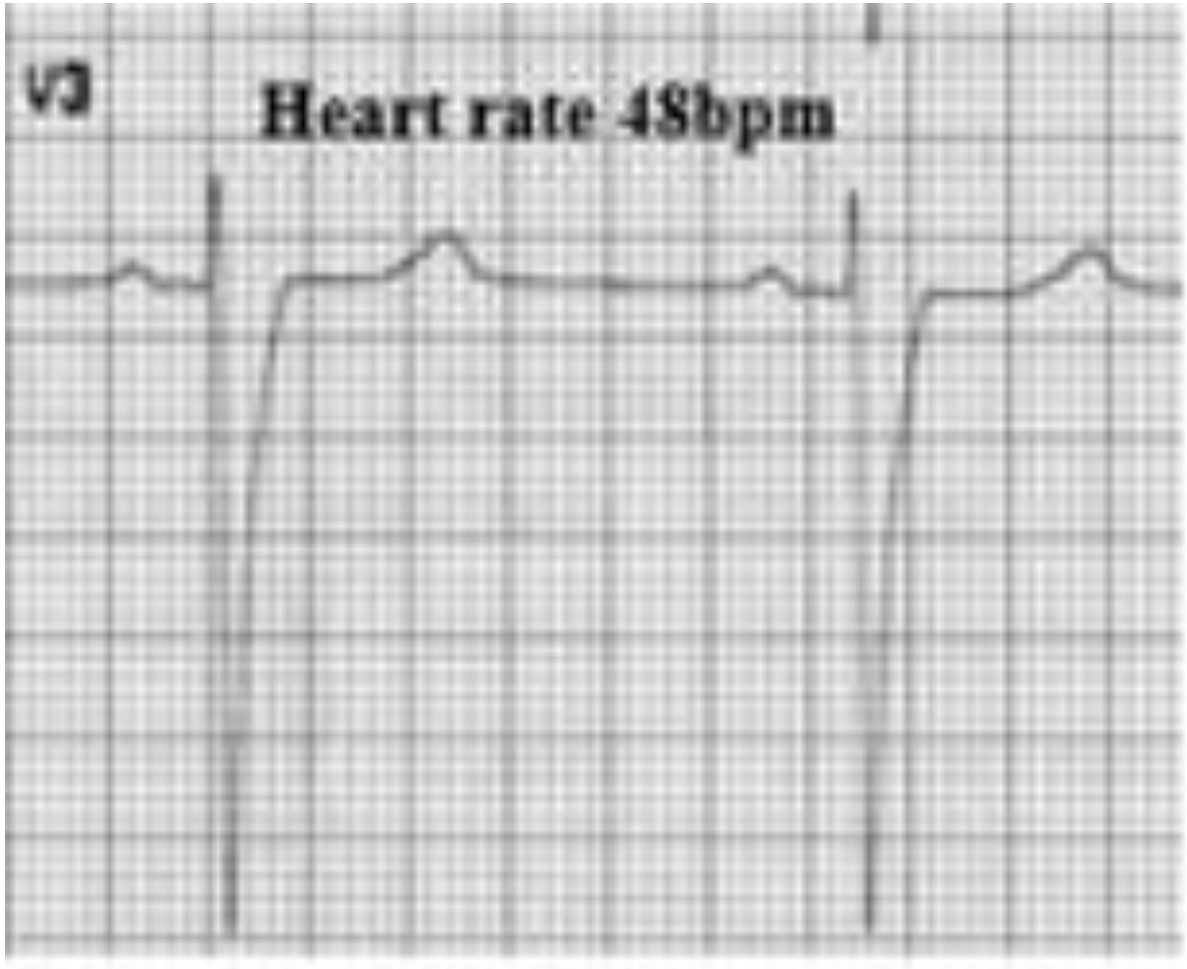
Prognosis prolonged QRS duration in a standard 12-lead ECG is associated with increased mortality in a general population, with intraventricular conduction delay being most strongly associated with an increased risk of arrhythmic death (**Aro. 2011**).

Normally, upstroke S wave at level of isoelectric baseline is < 55ms

Very prolonged S-wave upstroke in V_2 = 120ms indicative of severe **parietal block. Parietal block in right precordial leads is characteristic of arrhythmogenic ventricular cardiomyopathy, Brugada syndrome and atypical LBBB or NSIVCD.**

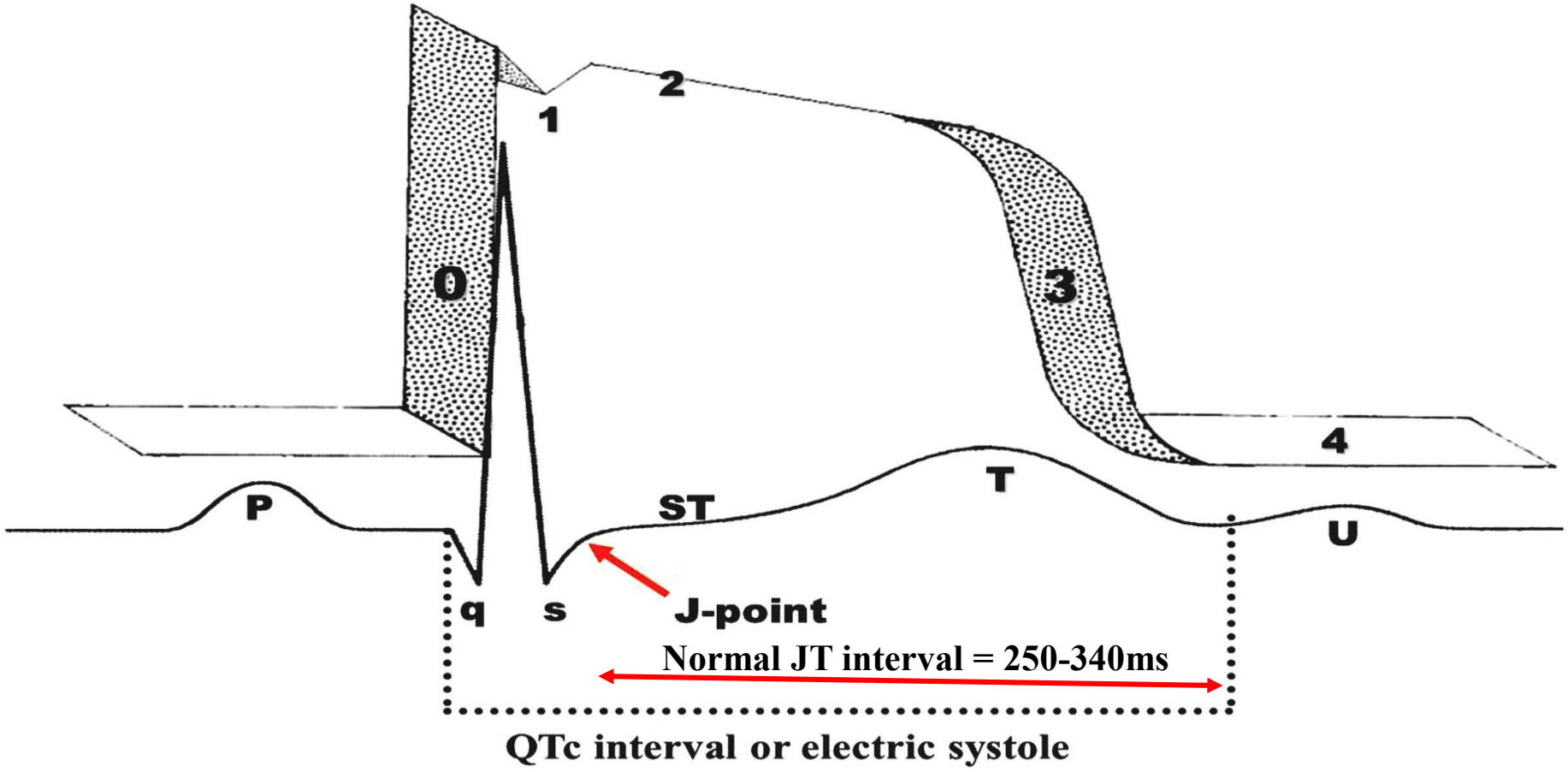
Prolonged QT interval QT/QTc 551/554ms





Mean Predicted QT Values at RR Cycle length Mean value 414 Lower limit 370ms Upper limit 458ms following Framingham study (**Sagie 1992**)

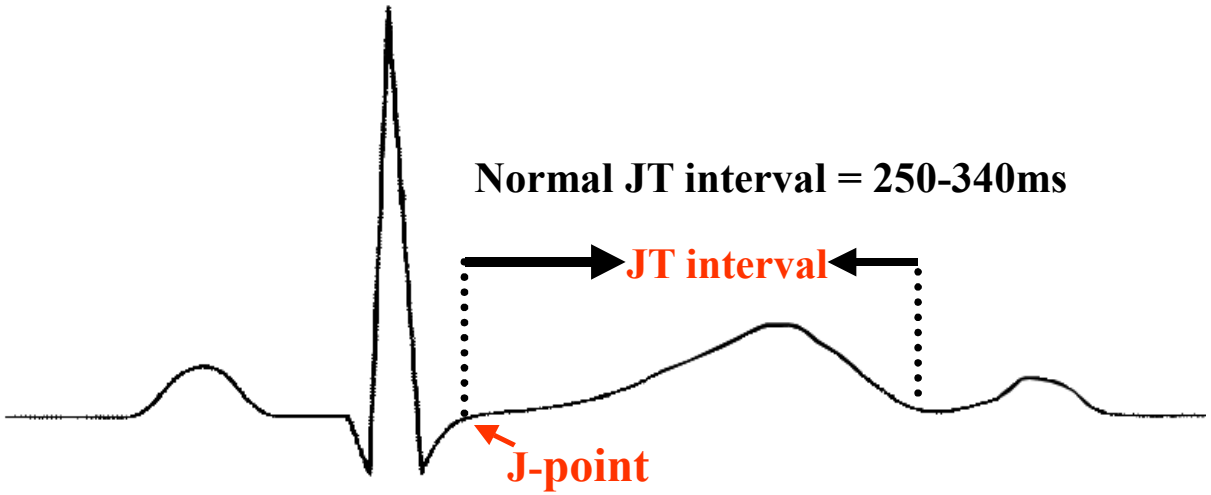
Representation of minimal and maximal normal values of QTc and JT intervals and its correlation with action potential



Normal values of QTc are between 350 to 440 ms or 446 + - 15%. QTc values <330 ms are considered short QT intervals. Values of QTc >450 ms are considered long QT intervals.

The JT interval extends from the **J point** to the end of the T wave. Normal JT interval = 250-340ms.

The value of meassurement of the JT and JTc intervals in the present case



JT and the rate-corrected JT interval (JTc):

The JT interval extends from the J point to the end of the T wave. Although the QTc has been the standard measurement of ventricular repolarization, it includes both depolarization and repolarization and may not always be a sensitive indicator of the type of repolarization abnormalities seen in LQTS. Intraventricular conduction disturbance complicate evaluation of the QTc interval. Thus, when there is **Left Bundle Branch Block, Right Bundle Branch Block, Nonspecific Intraventricular Conduction Delay (IVCD or NSICD) or Wolff-Parkinson White type of ventricular preexcitation**, the measurement of ventricular repolarization by QTc may be incorrect. In such cases, the measurement of JTc is more accurate than the QTc interval, because it excludes depolarization.

The measurement of JTc may be useful to identify LQTS cases with borderline values, where QTc interval could be normal in rest ECG.

We find an example in patients carriers of tetralogy of Fallot who underwent surgery, and as a consequence of RV ventriculotomy, developed CRBBB. In these cases, JTc interval measurement is more sensitive than the QTc interval to detect prolonged repolarization (**Berul 1994**).

QT interval

QTc interval is the value corrected according to HR, which represents the period between electric depolarization onset in the ventricles and the end of their repolarization. The end of T wave is defined as T wave return to the baseline, here called T-P segment.

For a proper measurement of the QT interval, we should be certain of not having included the U wave. To that end, it is advisable to perform the measurement in the aVL lead, because it is usually perpendicular to the U wave axis (SAU) (**Goldenberg 2006**). In the cases where there is R-R irregularity, we will conduct the measurement in three consecutive cycles, and then, the mean value is estimated. The normal maximal value that is accepted for the QT interval in males is 446 ms and in females 447 ms $\pm$ 15. If it exceeds 440 ms in males and 460 ms in females, the QT interval should be considered as prolonged. Values above 500 ms may cause a tendency to TdP. Patients with QTc intervals >600 ms are considered to be in high risk of arrhythmic SCD by TdP. In these cases, if the pharmacological treatment with beta blockers is insufficient to abolish TdP or in patients carriers of severe bronchial asthma or type 1 diabetes mellitus, where they are contraindicated, VATS ("Video-Assisted Thoracoscopic Sympathectomy") should be considered; or pacemaker at higher HR, when inappropriate bradycardia is observed as the main cause for TdP; and in special cases, ICD (**Garson 1993**). The QTc interval estimation is performed by applying the Bazett's formula proposed in 1920:

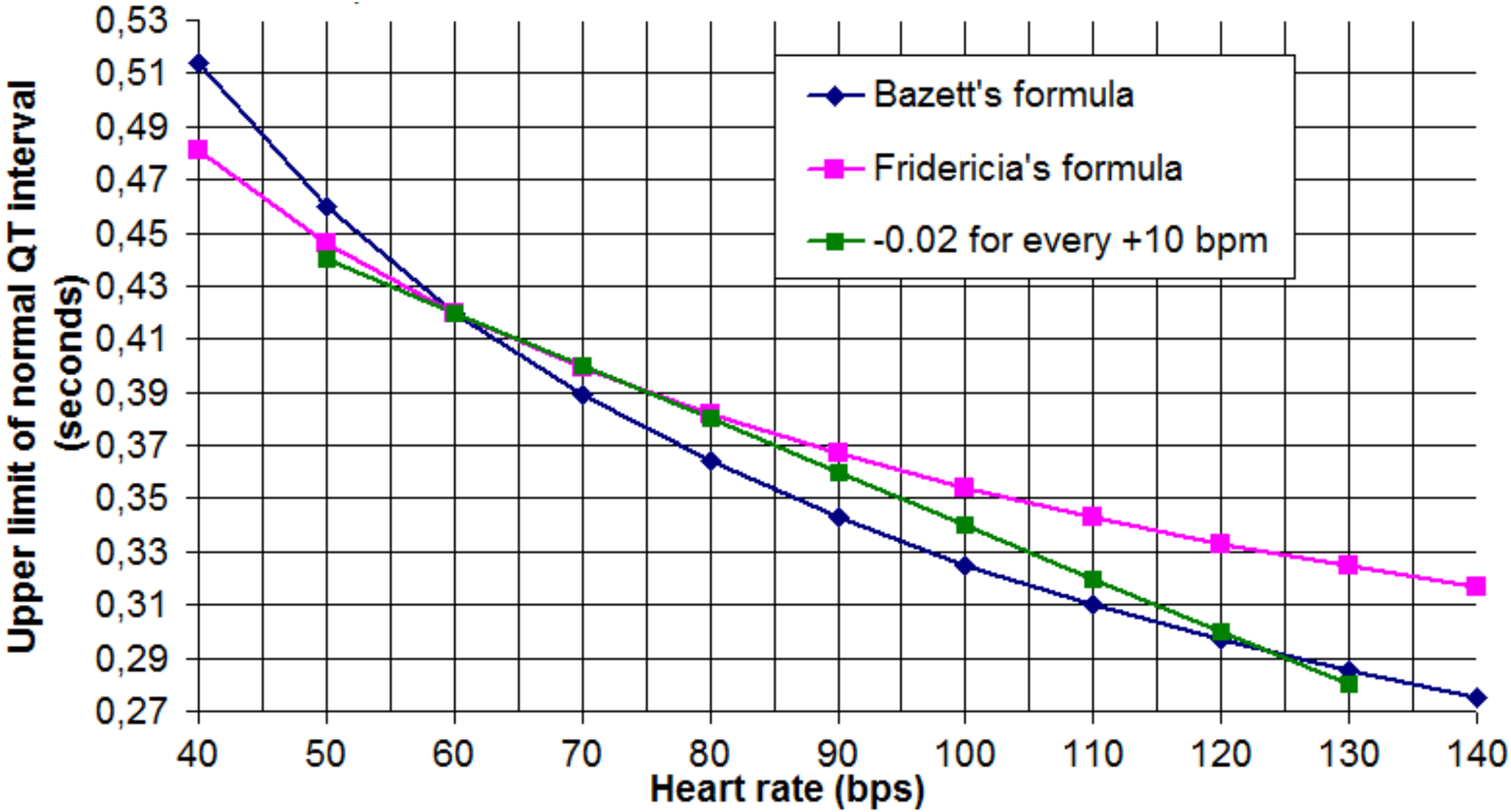
$$QTc = \frac{\text{Measured QT}}{\sqrt{RR}}$$

Bazett´s formula has been criticized because it tends to provide an inappropriately short QTc at slow rates and inappropriately long QTc at higher rates. Several competing methods have been developed: 1) (**Fridericia 1920**): **QTcF=QT/3√RR** published an alternative correction using the cuber root of RR cuberoot of RR. 2) Framingham: QTc = QT + 0.154 (1-RR) (**Sagie 1992**); 3) Hodges: QTc=QT+105(1+RR-1) (**Hodges 1983**)

None of the formulas has been shown to be clearly superior, so despite its obvious shortcomings.

Bazett's correction is used for automated analysis and large clinical trials.

QT duration is inversely proportional to heart rate. The range of normality of QT interval in adults varies between 350 ms and 440 ms. Both short and long QT intervals can be susceptible to life-threatening ventricular arrhythmias.



Upper limit of normal QT interval, corrected for heart rate according to Bazett's formula, Fridericia's formula and subtracting 0.02s from QT for every 10bpm increase in heart rate. Up to 0.42s (≤420ms) is chosen as normal QTc of QTf in this diagram.

Method for measurement of QT interval

When measuring the QT interval, the ECG is best recorded at a paper speed of 50 mm/s and at an amplitude of 0.5 mV/cm using a multichannel recorder capable of simultaneously recording all 12 leads. A tangent line to the steepest part of the descending portion of the T wave is then drawn. The intercept between the tangent line and the isoelectric line is defined as the end of the T wave.w3 The QT interval is measured from the beginning of the QRS complex to the end of the T wave on a standard ECG. There are no available data on which lead or leads to use for QT interval measurement. Traditionally, lead II has been used for QT interval measurement because in this lead, the vectors of repolarization usually result in a long single wave rather than discrete T and U waves (**Garson 1993**). Generally, QT prolongation is considered when the QTc interval is greater than 440 ms (men) and 460 ms (women), although arrhythmias are most often associated with values of 500 ms or more. The severity of pro-arrhythmia at a given QT interval varies from drug to drug and from patient to patient. Unfortunately, the extent of QT prolongation and risk of TdP with a given drug may not be linearly related to the dose or plasma concentration of the drug because patient and metabolic factors are also important (for example, sex, electrolyte concentrations, etc). Furthermore, there is not a simple relation between the degree of drug induced QT prolongation and the likelihood of the development of TdP, which can occasionally occur without any substantial prolongation of the QT interval.

The QT interval is influenced by heart rate. The RR interval preceding the QT interval should be measured for rate correction. Several formulae may be used to correct the QT interval for the biophysical effect of heart rate (QTc), but none is perfect. The most commonly used formulae are Fridericia's cube root formula (QTc = QT/RR1/3) and Bazett's square root formula (QTc = QT/RR1/2). Of the two, Bazett's formula is the more popular, but Fridericia's correction is preferred because it is more accurate at the extremes of physiological heart rate.w4 w5 Apart from heart rate, the duration of the QT interval is also subject to the techniques of recording and measurement error of the QT interval, sympathovagal activity, drugs, genetic abnormalities, electrolyte disorders, cardiac or metabolic diseases, changes of cardiac afterload, and diurnal variation which can be up to 75–100 ms. It is important to remember that for every individual there is a different relation between the QT interval and the heart rate. Although the rate–correction formulae are useful clinically, they may not be accurate enough, especially when assessing the minor changes of the QT interval induced by drugs. The suggested QTc values using the Bazett's formula for diagnosing QT prolongation are outlined in table (**Moss 1992**).

Table

Normal value (using the Bazett's formula)	350 to 440 ms or 446 ± 15%
Short QT/QTc interval	< 330 ms
Long QT/QTc interval	> 450 ms

Newer repolarization parameters such as QT dispersion (maximum – minimum QT intervals) on the 12 lead surface ECG, which is considered to be an indirect measure of spatial heterogeneity of repolarization, may be useful in assessing drug efficacy and safety. In one important study, patients who received class 1a antiarrhythmic drugs and developed TdP had significantly increased precordial QT interval dispersion.w6 In contrast, patients receiving amiodarone or class 1A antiarrhythmics without TdP did not have increased QT dispersion, although the QT interval was noticeably prolonged.w6 Thus, spatial heterogeneity/dispersion of the ventricular repolarization process may be required in addition to QT prolongation for the genesis of TdP. Although the use of QT dispersion in the assessment of drugs that prolong the QT interval needs further confirmation, it may provide information about the clinical significance of QT prolongation

Upper limit of normal QT interval, corrected for heart rate according to *Bazett's formula*, Fridericia's formula (**Fridericia 1920**) and subtracting 0.02s from QT for every 10bpm increase in heart rate (**Yanowitz 2010**). Up to 0.42s (≤420ms) is chosen as normal QTc of QT if in this diagram.

Definitions of normal QTc varies around being ≤400ms, ≤410ms, ≤420ms) or ≤440ms. For risk of sudden cardiac death "Borderline QTc" in males is 431-450 ms, and in females 451-470 ms.

An "abnormal" QTc in males is a QTc above 450 ms, and in females, above 470 ms. If there is not a very high or low heart rate, the upper limits of QT can roughly be estimated by taking QT=QTc at a heart rate of 60 beats per minute (bpm), and subtracting 0.02s from QT for every 10bpm increase in heart rate. For example, taking normal QTc ≤ 420ms, QT would be expected to be 420ms or less at a heart rate of 60bpm. For a heart rate of 70 bpm, QT would roughly be expected to be equal to or below 0.40s. Likewise, for 80 bpm, QT would roughly be expected to be equal to or below 380ms.

Blockers with slow association/dissociation, pronounced reduction in phase 0 slope; no effect on action potential duration or effective refractory period (flecainide, propafenone, and moricizine), hypothermia, left ventricular enlargement/hypertrophy and very elevated serum potassium levels.

The QRS duration prolongation secondary to drugs or electrolyte disturbances frequently have acute clinical presentation. If a drug like quinidine is being taken and can be related temporally to the NSIVCD then increasing dosage is not indicated, and in specific case of this drug, a QRS complex prolongation for values >140 ms or >35% of the baseline tracing, constitutes absolute indication of interruption of the drug (**Heissenbuttel 1970**).

NSIVCD can be a normal variant and is not always associated with cardiac pathology. Clinical correlation (history and physical examination) is needed to identify associated cardiac abnormalities.

If conduction delay is uniform, the QRS complex is uniformly widened and The QRS-T angle remains normal. Not all delays, however, can be explained by factors known to slow conduction uniformly in the ventricular muscle. Atypical QRS widening that does not fit the criteria for either

The prolongation of the QT interval induced by drugs and appearance of TdP could represent iatrogenic reproduction of hereditary-familial LQTS. In patients carriers of the silent form of hereditary-familial LQTS associated to IKr mutation arrhythmias are easily observed after prolonged exposition to these drugs (**Tamargo 2000**).

Factors should always be taken into account, which when coexisting, increase even more the possibility of prolonging QTc (**Haddad 2002**):

Presence of silent forms of LQTS associated to mutation in I_{Kr}; heart failure; and bradycardia;

Class Ia antiarrhythmic drugs of Vaughan-Williams classification - sodium channel blockers with intermediate association/dissociation, prolongation of action potential duration and effective refractory period - know as fast-channel blockers-affect QRS complex (quinidine, procainamide, and dysopiramide),

Quinidine: is a drug that increases the QTc interval, mainly as a consequence of the block of the different outward K+ channels in phase 3, and it may trigger early after-depolarizations (EADs) capable of causing triggered activity and this in turn, severe arrhythmias such as atypical PVT of the TdP type, having estimated prevalence in 1.5% to 8%.

In the cases of appearance of incessant salvoes of TdP with the use of the drug, isoproterenol may be used, or 5 mg EV verapamil at a velocity of 1 mg/minute**. In therapeutic** doses, quinidine causes discrete prolongation in QRSd (10-20%). The QRS complex prolongation may be diffuse or only in the final part (**Macfarlane 1989**). QRSd $\geq$140 ms or >35% of the baseline tracing, constitutes absolute indication of interruption of the drug. The increase in QRS complex duration is directly related to the plasmatic concentration of the drug. On the contrary, QTc interval prolongation is not related with such concentration (**Heissenbuttel 1970**).

T wave modifications: as a consequence of the effect on rectifier delayed outward potassium channels in phase 3 of action potential: it increases duration and it affects T wave polarity of ECG: **In leads where the T wave has positive polarity:** it initially decreases voltage and finally causes notches or discrete inversion. **In leads where T wave has negative polarity:** T waves decrease their depth. In general, the T vector has an orientation opposite to the final vector of the QRS complex.

On the JT interval: this interval is the distance existing between the J point and the onset of T wave. Quinidine causes prolongation in a degree somewhat greater than the QRS complex.

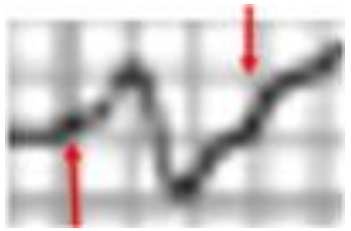
On the QTc interval: it causes prolongation of this parameter. If the QTc interval reaches 440 ms and the QT interval 600 ms, we consider that the drug caused acquired LQTS. QT interval prolongation is mainly due to the block of different delayed outward K^+ rectifier channels in phase 3. When the QTc interval is significantly prolonged, there is a greater possibility for early post-potentials to appear, capable of causing triggered activity, and in turn, polymorphic ventricular tachycardia of the "Torsades de pointes" type, which may degenerate into ventricular fibrillation and quinidine syncope (**Di Marco 1983**).

LBBB or RBBB may be caused by complex delays within the conduction system, regional conduction slowing within the myocardium, or a combination of both. No systematic exploration or classification of such patterns has been attempted (**Surawicz 1995**).

Conclusion

1) **Left Atrial Enlargement(LAE) or biatrial enlargement? Why RAD? Because normal P-axis in the FP is normal and first portion o P wave in V1 with deep slow final P-wave in V1**

P-duration =120ms



2) **Left Ventricular Enlargement(LVE)/Hypertrophy**
3) **Wide QRS fragmented QRS(W-QRS-f)**
4) **Severe parietal block**
5) **Anterior and inferior electrically inactive area associated with LAFB**
6) **Prolonged QT/QTc intervals/ and prolonged JT interval**
7) **Nonspecific or Unspecified Intraventricular Conduction Disturbance(NSIVCD):** QRS duration ≥120 ms in adults, greater than 90 ms in children 8 to 16 years of age, and greater than 80 ms in children less than 8 years of age without criteria for RBBB or LBBB. The definition may also be applied to a pattern with RBBB criteria in the precordial leads and LBBB criteria in the limb leads, and vice versa ("Masquerading bundle- branch block") (**Surawicz 2009**). NSIVCD is used when no characteristic pattern of RBBB or LBBB exists, although QRS duration is prolonged. Such abnormalities are frequently seen with previous MI and scar formation. If characteristics abnormalities of the initial QRS portion (Q waves) are present associated with abnormal terminal QRS forces directed toward the infarct, with an angle of ≥100º between the initial and the terminal QRS forces with little or no prolongation of the QRS complexes, these can be called peri-infarction block. (**Grant 1959; Vassallo 1986**). The term possible peri-infarction block is recommended when, in the presence of an abnormal Q wave generated by a myocardial infarction in the inferior or lateral leads, the terminal portion of the QRS complex is wide and directed opposite to the Q wave (ie, a QR complex in the inferior or lateral leads). Peri-ischemic block is term recommended when a transient increase in QRS duration accompanies the ST-segment deviation seen with acute injury. Reversible QRS changes during AMI are attributed to passive pull by the ST-segment shift and intraventricular conduction disturbance (**Surawicz 1998**). Prolonged QRS duration without a specific bundle branch block pattern can also occur due to intramyocardial conduction slowing by drug-induced block of the sodium channels such as tricyclic-antidepressants (TCAs) overdose (e. i. amitriptyline, nortriptyline, trimipramine, despiramine, protriptyline, and dothiepin poisoning), only observed in toxic doses as attempt of suicide and mostly secondary to QRS width prolongation. Imipramine (Tofranil) is the one that presents a greatest cardiovascular toxic power. The event occurs after high-degree or total block (**Roose 1987**). Imipramine and amitriptyline act mostly by inhibiting the HERG potassium channel (HERG defect of the K+ channel or human-ether-a-go-go-related gene) that affects the rapid outward K+ channel known as IKr ("delayed rectifier current") (**Witchel 2003**).

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Merry Christmas and Happy New Year for all!!!!!