Extensive myocardial infarction associated with intraventricular conduction disturbance

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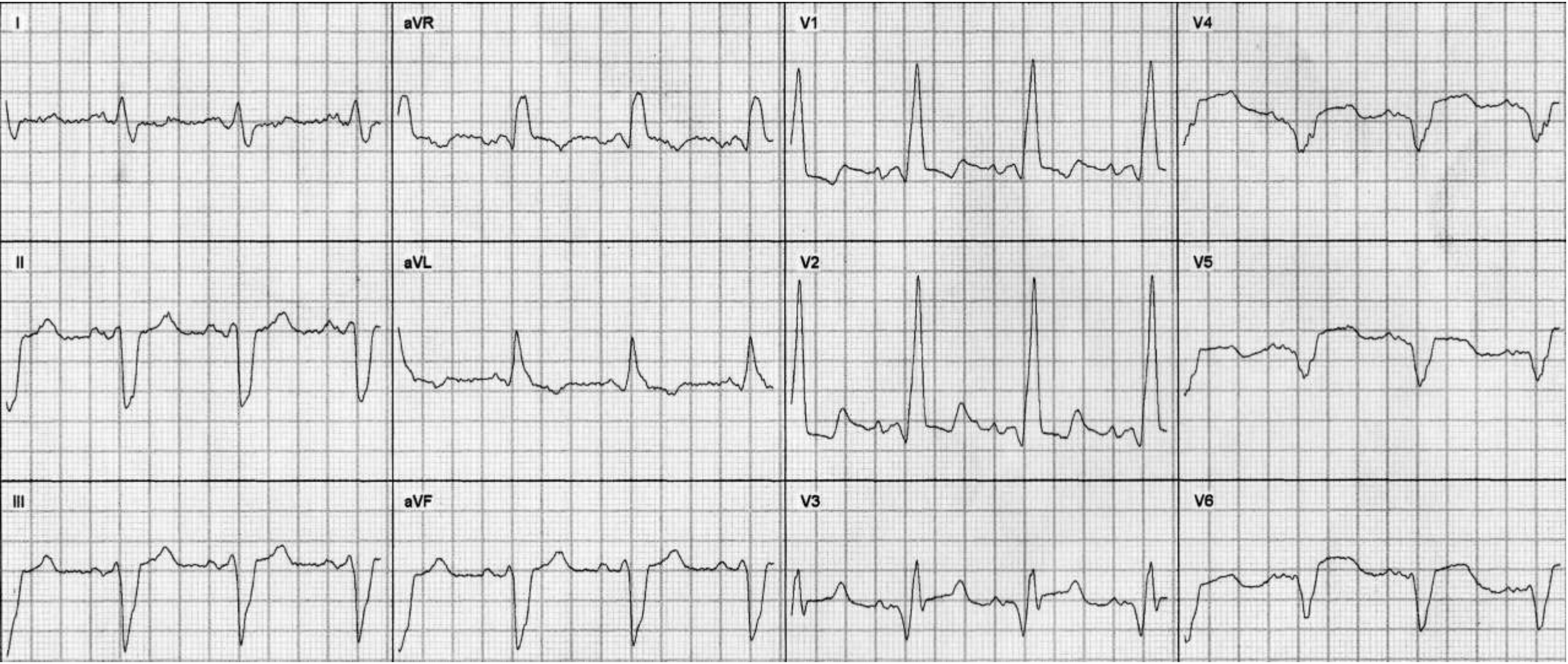
Portuguese
Relato de caso
Sexo masculino, branco, 65 anos. Eupneico, angina e cansaço aos esforços progressivos nos últimos 3 dias. Hipertenso, dislipidêmico
História de IAM há 2 anos com implante de 2 stents na artéria descendente anterior
Medicação: losartana, sinvastatina, aspirina, bisoprolol, furosemida
PA=130/70
RCR 2T B4 SS++/4 foco mitral segunda bulha desdobrada
Pulmões limpos
Sem edema periférico

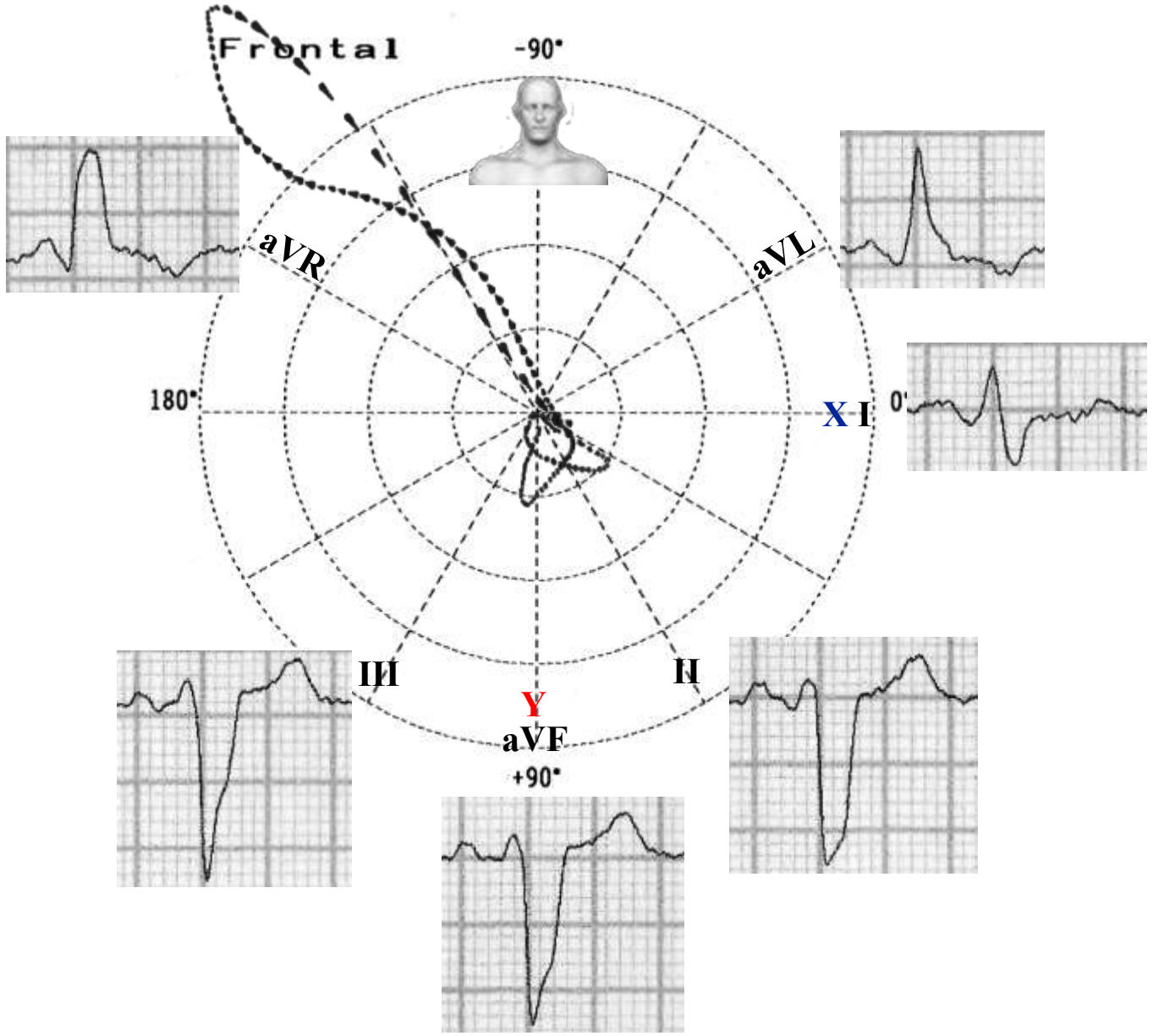
Perguntas:
1. Qual o diagnóstico ECG/VCG?
2. Qual a conduta adequada?

English
Case report
Caucasian, male, 65 years. Angina and tiredness to progressive efforts in the last 3 days. Hypertensive, dyslipidemic, CAD.
History of AMI 2 years ago with implantation of 2 stents in the anterior descending artery
Medication: losartan, simvastatin, aspirin, bisoprolol, furosemide
BP = 130/70 eupneic
RCR 2T B4 Systolic murmur ++ / 4 mitral focus. Second unfolded sound
Clean lungs
Without peripheral edema

Questions:
Which is the ECG / VCG diagnosis?
Which is the proper approach?


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

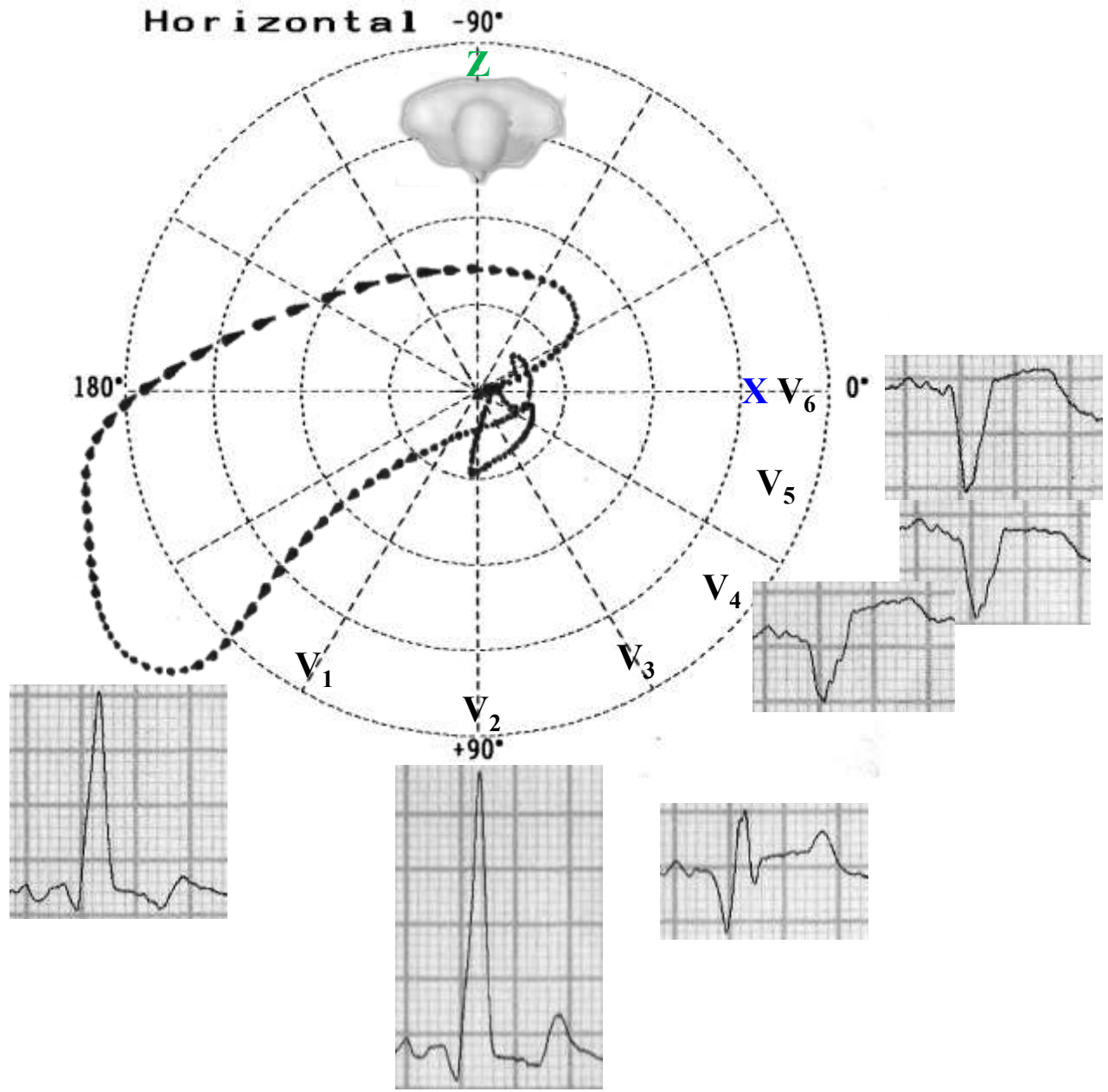
ECG/VCG correlation in the frontal plane



Magnified T-loop

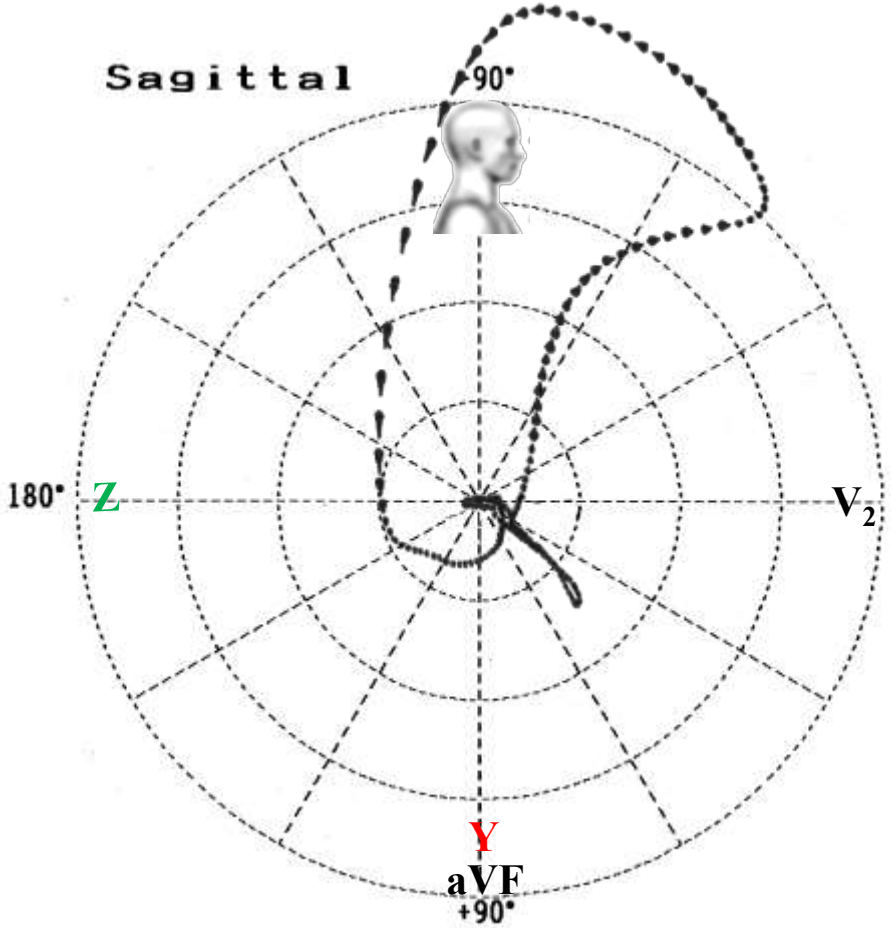
Magnified P-loop

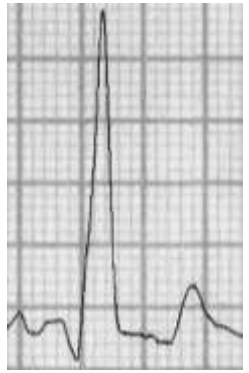
ECG/VCG correlation in the horizontal plane

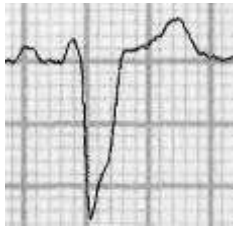


Magnified P-loop

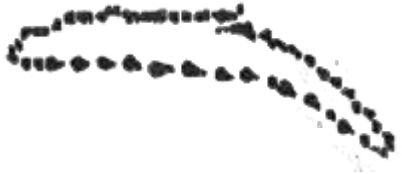
ECG/VCG correlation in the Sagital plane







Magnified P-loop



	Normal values				Normal values
Weight	67 kg		Right ventricle diameter	31 mm	
Height	162 cm		Ejection fraction	24%	>53%
Body surface	1.715 m^2		Left ventricle mass	312 g	94 a 276 g
End diastolic diameter of the left ventricle	77 mm	35 a 56 mm	Percentage of cavity shortening	12%	-
End systolic diameter	68 mm	25 a 40 mm	End diastolic volume	316 ml	73 a 156 ml
Diastolic thickness of the septum	8 mm	07 a 11 mm	Ejected volume	77 ml	54 a 99 ml
Diastolic thickness of the posterior wall of the LV	9 mm	07 a 11 mm	Volume/mass ratio	1.46 ml/g	0.45 a 0.90 ml/g
Aorta	35 mm	20 a 37 mm	Systolic volume	239 ml	18 a 57 ml
Left atrium	55 mm	20 a 40 mm	Mass index of the left ventricle	81.78 g/m^2	

Colleagues Opinion

Dear friends.
Looks like an old extensive anterior Q-wave MI. In addition, there could also be a more recent lateral Q-wave MI. RBBB (probably not LSFB?) and LAFB. ST elevations in V4-V6 could indicate an aneurysm. Severely depressed LV systolic function. PTF as a sign of increased LV filling pressure (diastolic dysfunction).
Coronary angiography. Prophylactic ICD. ACE-inhibitor/ATR-blocker, betablocker, spironolactone possibly.
Best regards
Kjell Nikus
Tampere Area, Finland - Professor in Cardiology at Heart Center, Tampere University Hospital

The ECG shows sinus rhythm. Left atrial abnormality. RBBB + Left anterior. Probably RVH. There are Q wavesV1-V6. There is ST elevation V3-V6.
I suspect that this is ischemic cardiomyopathy with aneurysm and eccentric hypertrophy. Based on the murmur, I suspect significant MR with pulmonary hypertension. I think that the last slide (echo? MRI?) support it.
Regards

Yochai Birnbaum, MD, FACC, FAHA
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Dear Raimundo and Andrés,
Right bundle branch block, left QRS axis, normal PR interval, Intramural conduction delay, Lateral myocardial infarction. Intra atrial block.
Associated degenerative cardiomyopathy.
What about a new coronarography. After volume depletion (high pulmonary cap. wedge pressure) I would suggest exercise stress test and holter monitoring. A CRT seems not indicated (lateral necrosis, RBBB).
An ICD could be discussed.
Kind regards,
Philippe Chevalier Lion France
philippe.chevalier@chu-lyon.fr
Service de Rythmologie, Hôpital Louis Pradel. Hospices Civils de Lyon. Université de Lyon
Claude Bernard University Lyon 1
Villeurbanne, France

Pr. Philippe Chevalier, MD PhD is the head of the Rhythmology unit of HCL and the coordinator of the National Reference Center for inherited arrhythmia. He is an internationally recognized clinical expert in the field of cardiac arrhythmias. He has been implicated in more than 10 clinical studies. He is the principal investigator and coordinator of a European multicenter study on molecular markers of sudden death. He develops fundamental studies on the pathophysiology of AF. He organized every year the national congress of “les journées de rythmologie” in Lyon. Regularly invited to international cardiology congress, he is also a member of several societies (French Society of Cardiology, American Heart Association). His research led him to be present in more than 358 publications pubmed indexed.

Portuguese

Jose Grindler

Ritmo Sinusal. SAE, BRD. BDAS, Área Inativa anterior Provável SVD.
Conduta: Terapeutica Otimizada, Implante Apenas de CDI AV para prevenção primária de morte súbita.

José **Grindler** e Acácio F. **Cardoso**

Faculdade De Medicina USP- ECG HC

English

Sinus rhythm. Left Atrial Enlargement(LAE), RBBB. LAFB, Previous anterior Inactive Area, a probable RVH.
Conduct: Optimized Therapy, Implant Only ICD for AV for primary prevention of sudden death.

José Grindler and Acácio F. Cardoso

Faculty of Medicine USP- ECG HC

Spanish

Estimado *Andrés*, interesante caso con miocardiopatía dilata e insuficiencia cardiaca de origen isquémico, con importantes defectos de conducción: bloqueo inteauricular con onda P de 0.16 seg, ascenso lento en DII y V1, bloqueo fascicular anterior en el plano frontal, bloqueo fascicular medio en el plano horizontal, trastorno de conducción intraventricular por necrosis miocárdica anterior con ondas q profundas anteriores y probable zona de necrosis lateral del VI (V4-V6), con un QRS de 0.20 seg, con eje de QRS hacia aVR, en el vectocardiograma en plano frontal entre - 120 a - 150 º. Relacionado con importante daño miocárdico confirmado por el ecocardiograma con dilatación severa del VI y crecimiento de AI. Tiene alto riesgo de muerte súbita y también alto riesgo de muerte por progresión de la falla cardiaca

Saludos

Dr. Humberto **Rodriguez-Reyes** (FACC, FHRS y AHA Member) Mexico.Cardiologia, Electrofisiologia (Arritmias), Medicina Interna

Instructor BLS, ACLS y ACLS-EP de la AHA. Presidente Sociedad Cardiovascular y del Metabolismo Presidente SOMEEC 2013-2014 • Coordinador del capítulo de Reanimación Cardio Pulmonar del Consejo de la Alianza contra la Muerte Súbita de la SIAC.

English: Dear Andrés, interesting case with dilated myocardiopathy and heart failure of ischemic origin, with important conduction defects: P with interatrial block, (P wave duration of 0.16 sec, slow ascent in II and V1, left anterior fascicular block in the frontal plane, left septal fascicular block in the Horizontal plane, intraventricular conduction disorder due to anterior myocardial necrosis with anterior deep q-waves and probable lateral necrosis (V4-V6), with a very broad QRS(0.20 sec) with QRS axis towards aVR, in frontal plane vectorcardiogram Between - 120° to - 150°. Associated with significant myocardial damage confirmed by the echocardiogram with severe LV dilatation and AI growth. It has a high risk of sudden death and also a high risk of death due to progression of heart failure.

Dr. Humberto **Rodriguez-Reyes** (FACC, FHRS y AHA Member) Mexico.

HOSPITAL CARDIOLÓGICA AGUASCALIENTES

Escudo

Spanish

Hola Intentaré hacer el diagnóstico electrocardiográfico y correlacionarlo con el VCG

ECG

Agrandamiento de aurícula izquierda
Bloqueo de fascículo anterior izquierdo (BFAI)
Bloqueo de rama derecha
Fuerzas anteriores prominentes(FAP) qR en V1 V2 con QR en V3 y QS en V4 V5 V6 por necrosis lateral de VI que" simula " BFMS

VCG

1. **Plano frontal** el bucle de P evoca agrandamiento de aurícula izquierda. El bucle del QRS se localiza predominantemente en cuadrante superior derecho por bloqueo del fascículo antero-superior izquierdo asociado a hipertrofia ventricular izquierda. El bucle de T muestra los puntos 0 y J no coincidentes que explica el segmento ST desnivelado
2. **Plano Horizontal:** El bucle de P sugiere sobrecarga biauricular. Bucle de QRS predominantemente localizado en cuadrante anterior derecho por bloqueo de rama derecha asociado con fuerzas anteriores prominentes (FAP) por falta de oposición de las fuerzas de la pared lateral por infarto /necrosis de dicha cara (antiguo cara posterior) simulando un bloqueo del fascículo medio
3. **Plano Sagital Derecho:** El bucle P indica agrandamiento auricular izquierdo. Bucle QRS orientado en los cuadrante superiores e anteriores por la mencionada falta de oposición de fuerzas de la cara lateral

Conducta

- Optimización de terapia con fármacos
- Terapia de resincronización cardiaca: la fibrosis de cara lateral invalidaría umbrales eléctricos aceptables No indicada
- CDI
- Transplante futuro?

Abrazos
Juan José **Sirena MD** Santiago del Estero Argentina

Hi I will try to make the electrocardiographic diagnosis and correlate it with the VCG

ECG

- Enlargement of the left atrium
- Left anterior fascicular block (LAFB)
- Right Bundle Branch Block(RBBB)
- Prominent Anterior Forces (FAP) qR in V1-V2 with QR in V3 and QS from V4 to V6 by lateral necrosis of VI that "simulates" LSFB.

VCG

1. **Frontal plane:** the P loop evokes left atrial enlargement. The QRS loop is predominantly located in the upper right quadrant due to LAFB associated with left ventricular hypertrophy. The T loop shows the mismatched 0 and J points explaining the uneven ST segment elevation.

2. **Horizontal plane:** The P loop suggests biauricular overload. The QRS loop predominantly located in right anterior quadrant due to right bundle branch block associated with prominent anterior forces (FAP) due to lack of opposition of side wall forces due to infarction /lateral necrosis of (old posterior infarction) simulating a LSFB.

3. **Right Sagittal Plane:** The P loop indicates left atrial enlargement. QRS loop oriented in the upper and anterior quadrant by the aforementioned lack of opposition of lateral side forces.

Approach

- Optimization of drug therapy
- Cardiac resynchronization therapy: lateral-side fibrosis would invalidate acceptable electrical thresholds Not indicated
- CDI
- Future transplant?

Hugs

Juan José **Sirena MD** Santiago del Estero Argentina

Interesting case, not common in the era of early reperfusion. Extensive anterior wall remote myocardial infarction complicated with right bundle branch block and left anterior fascicular block. Modified Selvester score predicts myocardial scar ~ 30% as shown in cardiac magnetic resonance

Conduct: Coronary angiography in view of angina. Revascularization if sizable territory at risk/viable.

ICD is indicated regardless of revascularization.

I doubt His bundle pacing would be able to normalize the QRS, but I would try it anyway at time of implant.

Otherwise, biventricular pacing, although benefit in this setting is borderline

Optimize heart failure treatment, consider sacubitril/valsartan

Cordially

Sergio **Pinski, M.D**

Dr. Sergio Pinski is a cardiologist in Weston, Florida and is affiliated with Cleveland Clinic Weston Florida USA. Specialized in Cardiovascular Disease.

Dear Andres,
This ECG shows:
1. Sinus rhythm with notched P waves
2. Complete right bundle branch block
3. left axis deviation
4. Q waves in lead I through V6 with loss of R wave in V4 to V6, consistent with old anteroseptal myocardial infarction complicated with RBBB and left axis deviation.
5. ST-T wave changes consistent with remote anteroseptal myocardial infarction and RBBB

Questions about next steps includes what were the echocardiogram findings and the LVEF?
Looking forward to hearing the correct answer.

Very best,

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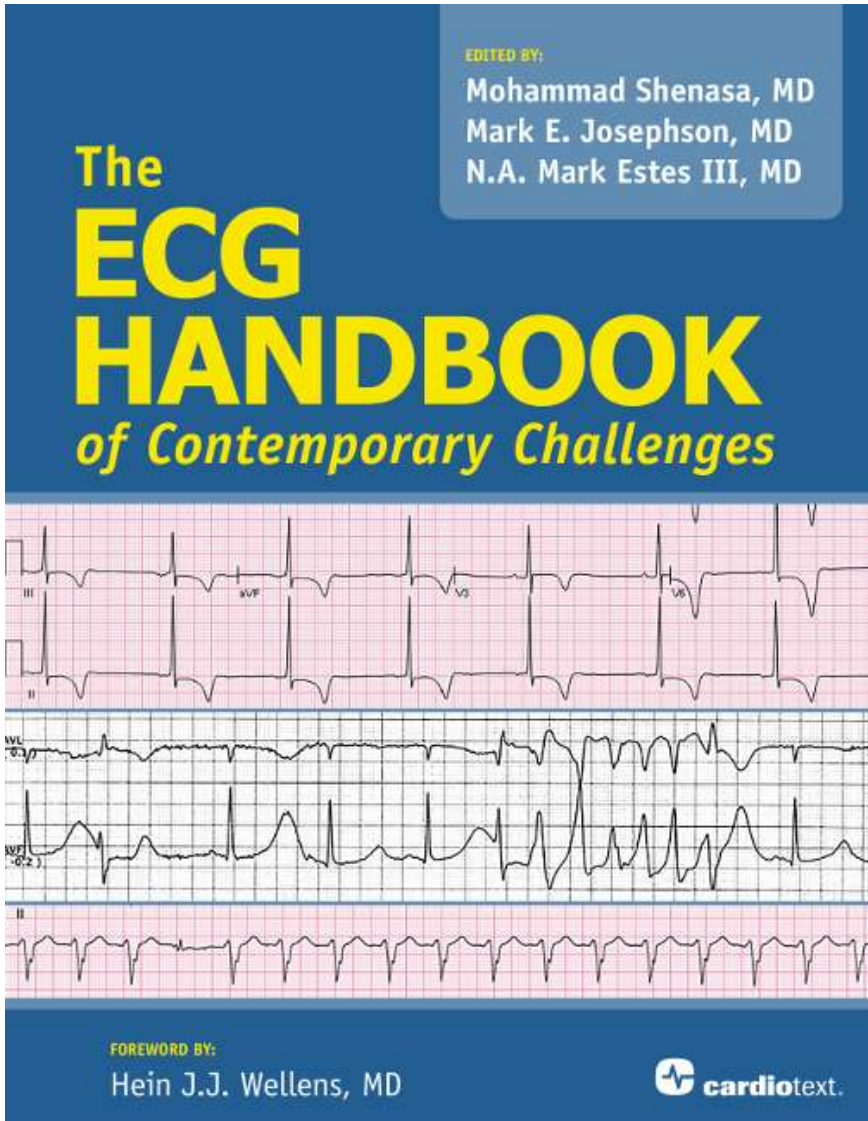
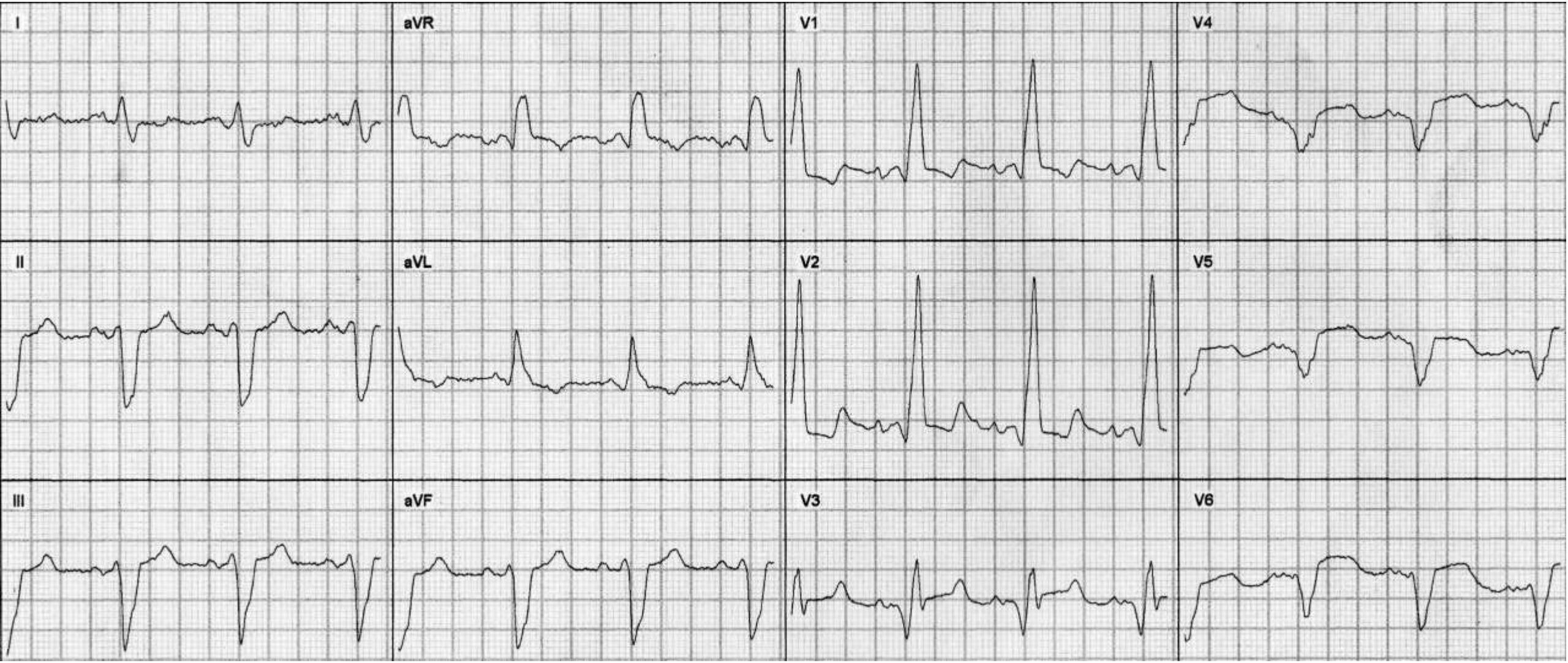


Final conclusions by
Andrés Ricardo Pérez-Riera & Raimundo Barbosa-Barros

“But there is no possible knowledge which arrives not from a pre-existent knowledge.”

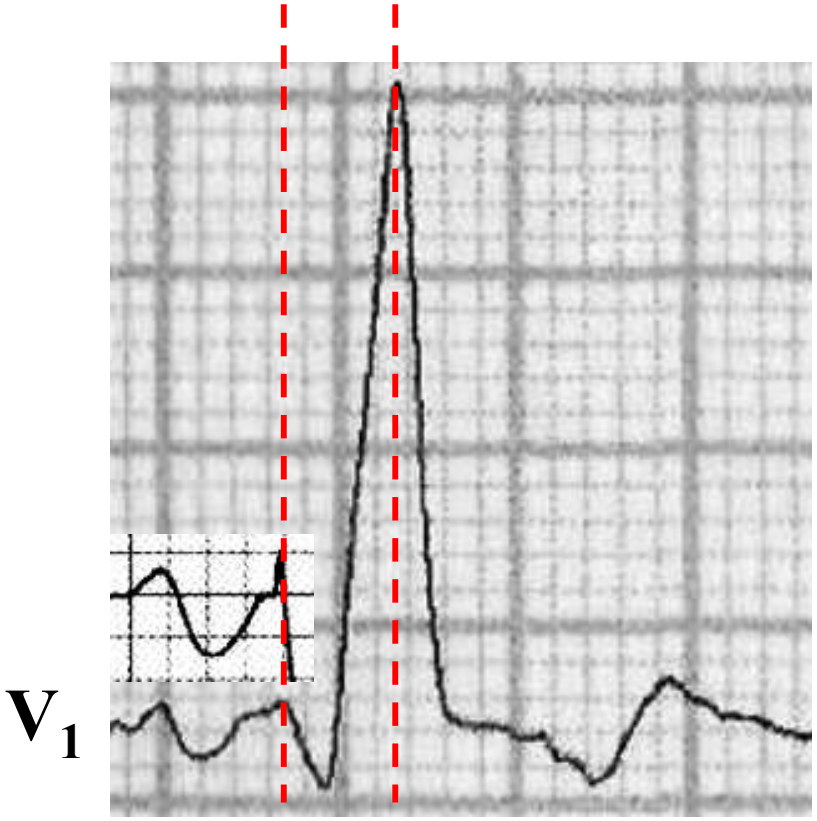
William Harvey(1578-1657)



ECG diagnosis: sinus rhythm, SÂP +35°, P duration 120 ms, P axis (SÂP) + 35° with plus-minus pattern in V_1 (deep terminal negativity of P wave in V1 (DTNPV1), normal PR interval, very broad QRS duration (196 ms), very prolonged R-wave peak time in V_1, QRS axis (**SÂQRS -105°**) located in the upper right quadrant (-105°), SIII>SII, qR in right precordial leads and QS from V3 to V6 and persistent ST segment elevation form V4 to V6 suggesting LV aneurism.

Conclusion: left atrial enlargement (LAE), left anterior fascicular block (LAFB), complete right bundle branch block (CRBBB), transmural anterolateral MI+ probable LV aneurysm in LV lateral wall(persistent ST segment elevation from V4 to V6.

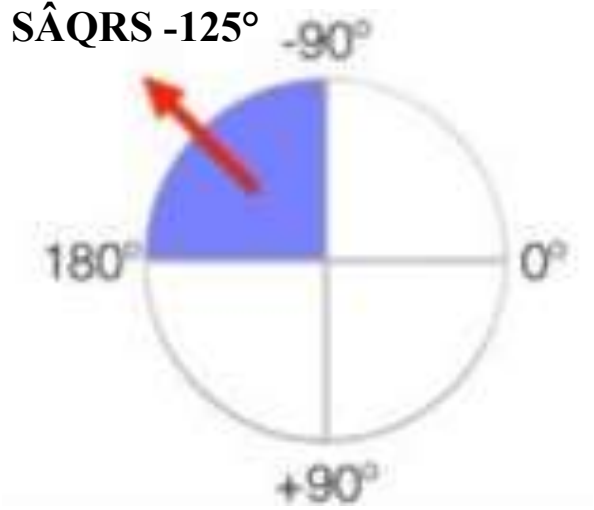
Very prolonged R-wave peak time in V1: 125 ms!!! In normal conditions, the R-peak time for the thinner-walled right ventricle is measured from lead V_1 or V_2 and its upper limit of normal is 35 ms (**Pérez-Riera 2016a**)

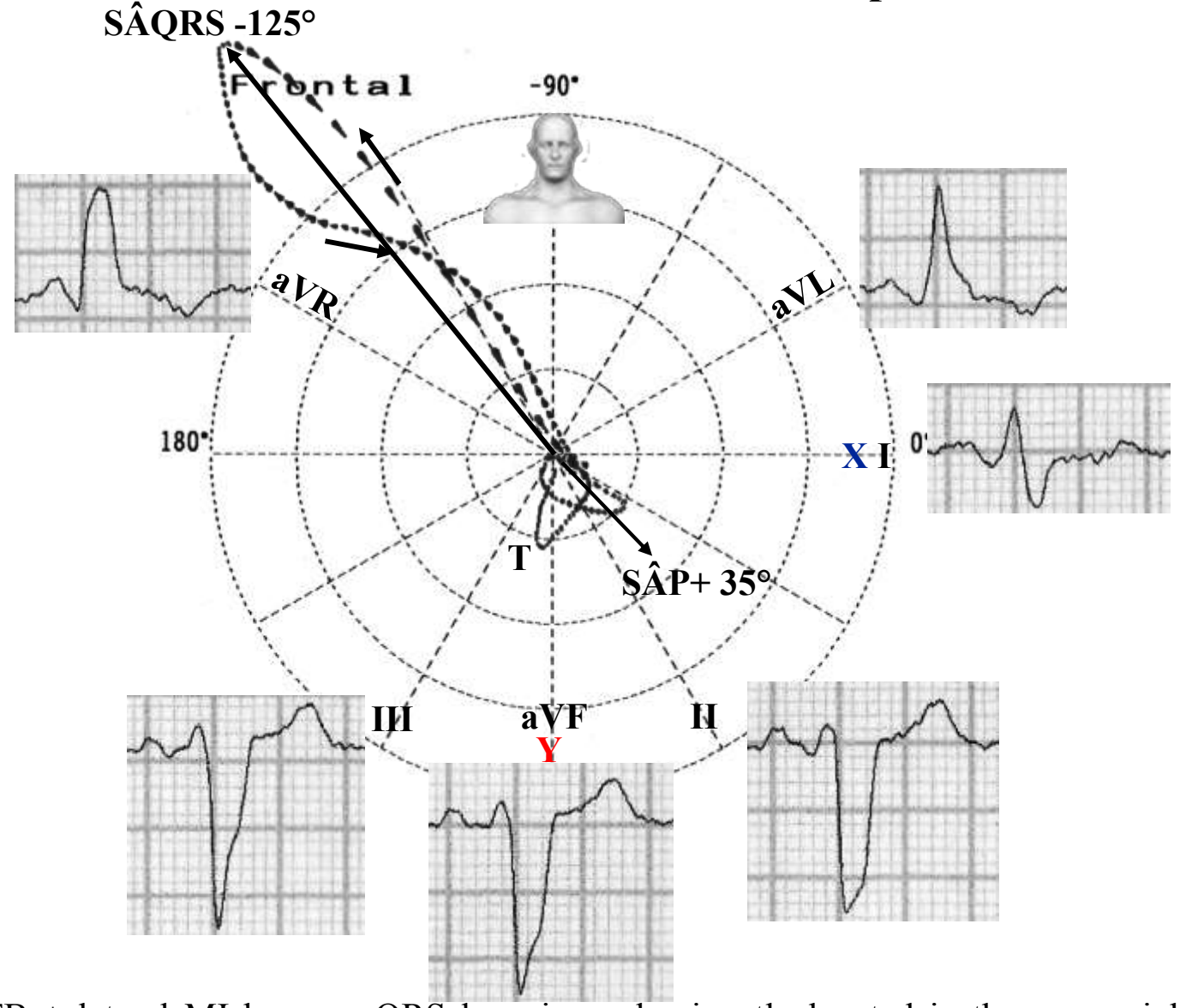


The R-wave peak-time (RWPT) extends from the beginning of the QRS complex to the apex of the R-wave, and corresponds to the initial and middle activation of the ventricular depolarization wall. In the right precordial leads, RWPT can not exceed a small square (40 ms). In this case, the RWPT value is more than 3 times the normal limit, signaling the Q wave duration of necrosis + transmural conduction disturbance.
There are 71 ms from the apex of R-wave to the end of QRS, consequently, 125 ms + 71 ms = 196 ms. This value is the QRS duration.

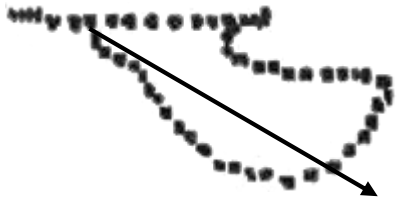
P-wave polarity in V_1: plus-minus with deep P-terminal force (PTF- V_1) or deep terminal negativity of P wave in V1 (DTNPV1) exceeding 0.04 mm/s. This is the terminal, negative part of the P wave in lead V_1 expressed as the multiplication of its depth in millimeters and width in seconds (mm/s). The normal PTF- V_1 does not exceed 0.04 s wide and 1mm deep, i.e., 0.04 mm/s. Morris index (**Morris 1964**) DTNPV1 is predictive of SCD suggesting its potential utility in risk stratification in the general population (**Tereshchenko 2014**).

ECG/VCG correlation in the frontal plane

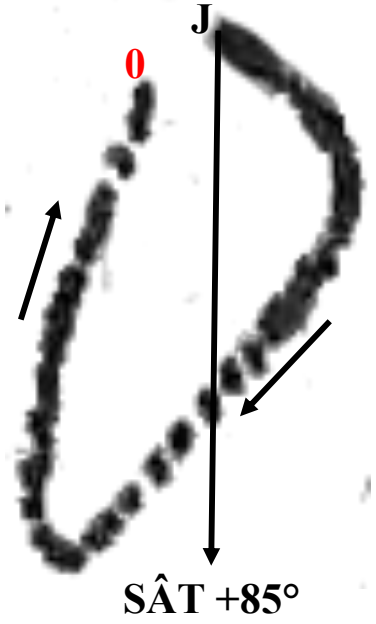


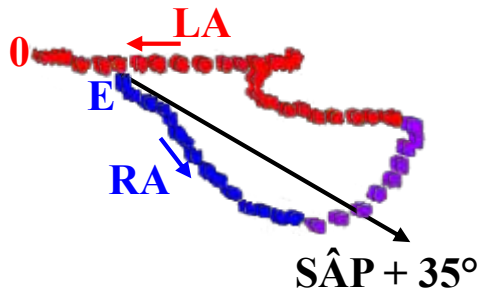


Magnified P-loop



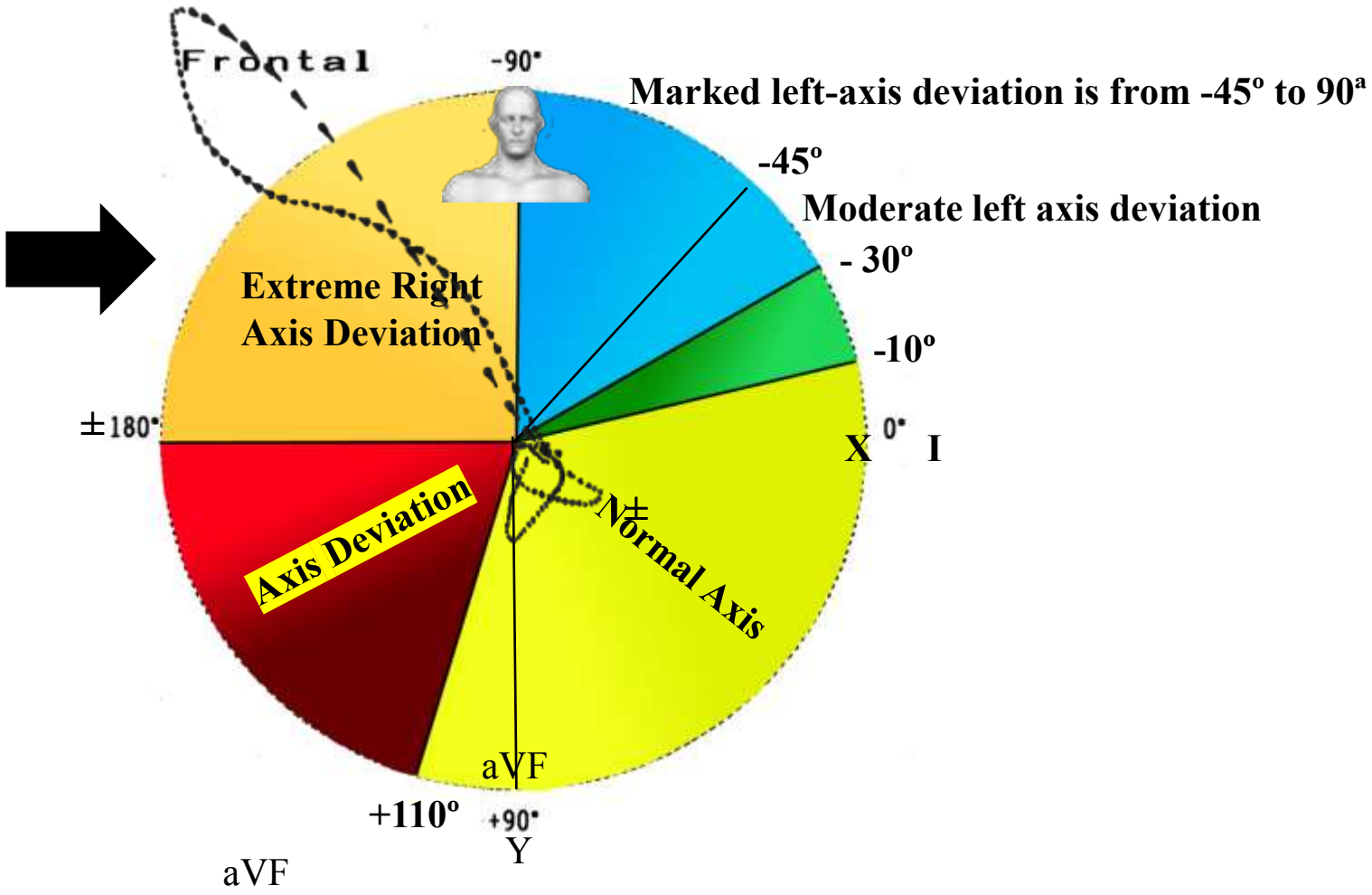
Magnified T-loop





Conclusion: LAE + atypical LAFB + lateral MI because QRS loop is predominantly located in the upper right quadrant: extreme right axis deviation = QRS axis between -90° and 180° (AKA “Northwest Axis”) or “no-man's-land” QRS axis (i.e., "N-M-L".). "N-M-L" is not synonymous with an "indeterminate" axis which occurs when the QRS is essentially isodiphasic or equiphasic in all 6 limb leads and therefore the polarity of the QRS cannot be discerned in leads I and aVF.

QRS axis (SÂQRS) in adults in the Frontal Plane

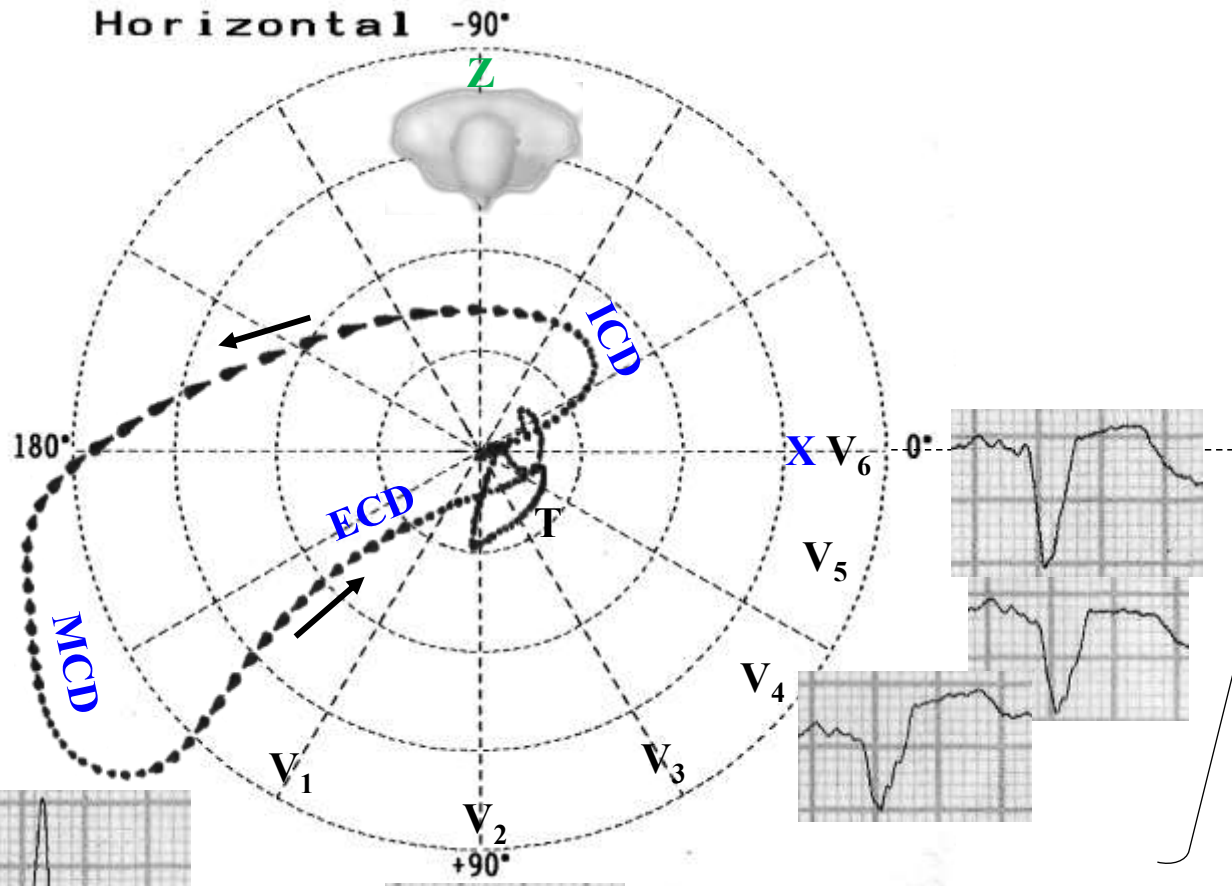


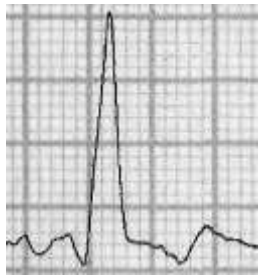
Normal QRS axis(SÂQRS) = between -10º to -110º
Moderate left axis deviation between -30º to -45º
Marked left-axis deviation is from -45º to -90°
Right axis deviation from +90 to +180º
Marked right-axis deviation is from -90º to ±180º (AKA “Northwest Axis”) or "no-man's-land“ QRS axis (i.e., "N-M-L".).

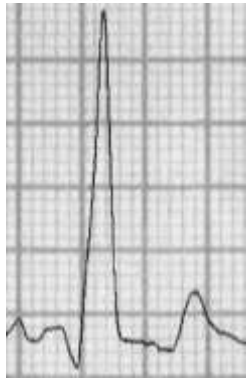
ECG/VCG correlation in the horizontal plane

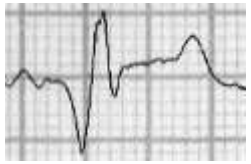
QRS loop with CCW rotation and located predominantly in the right quadrants. Initial, middle and final forces with delay inscription is observed. The initial delay forces are consequence of transmural lateral MI and the final forces located in the anterior right quadrant are consequence of RBBB. Finally, the middle conduction delay is consequence of intrainfarction block. The ST segment elevation observed from V3 to V6 is indicative of left ventricle aneurysm in the lateral wall.

Conclusion: LAE, transmural anterolateral MI associated with RBBB and possible anterolateral aneurysm.

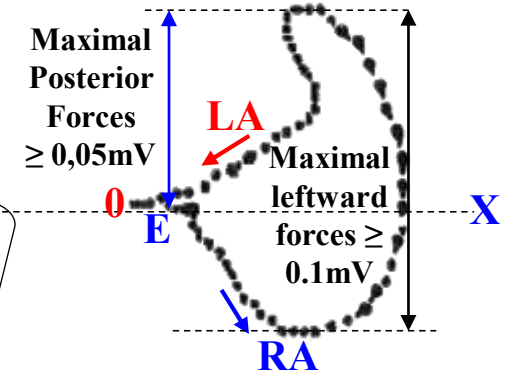



qR


qR


QRs

QS



Magnified P-loop

LAE

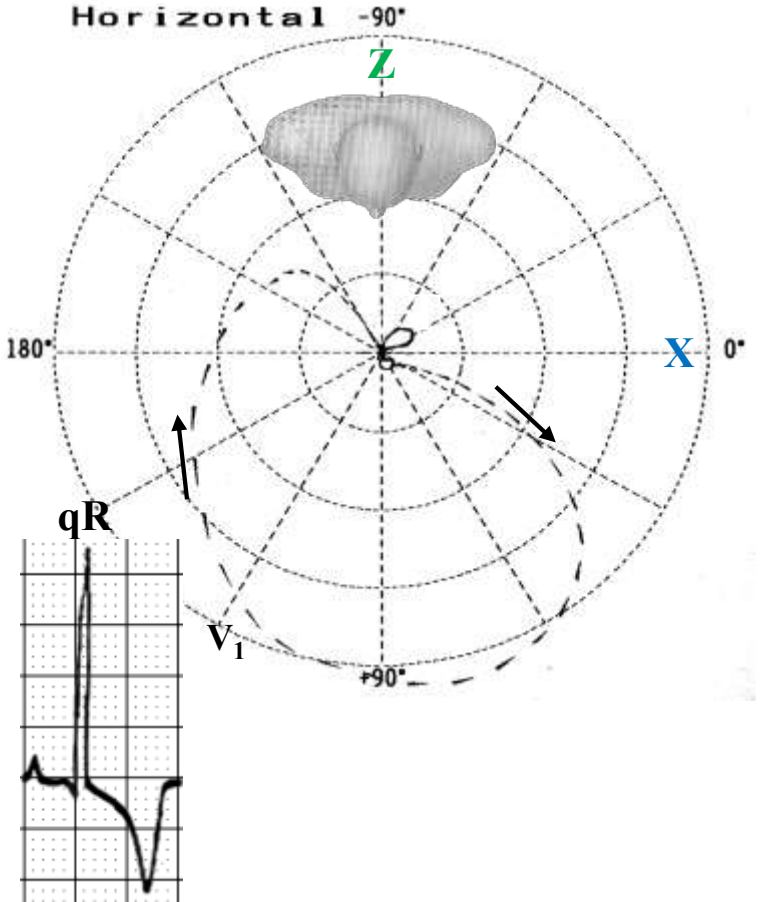
ICD: Initial Conduction Delay
MCD: Middle Conduction Delay: intramural conduction delay
ECD: End Conduction Delay

Possible causes of QR/qR pattern in right precordial leads

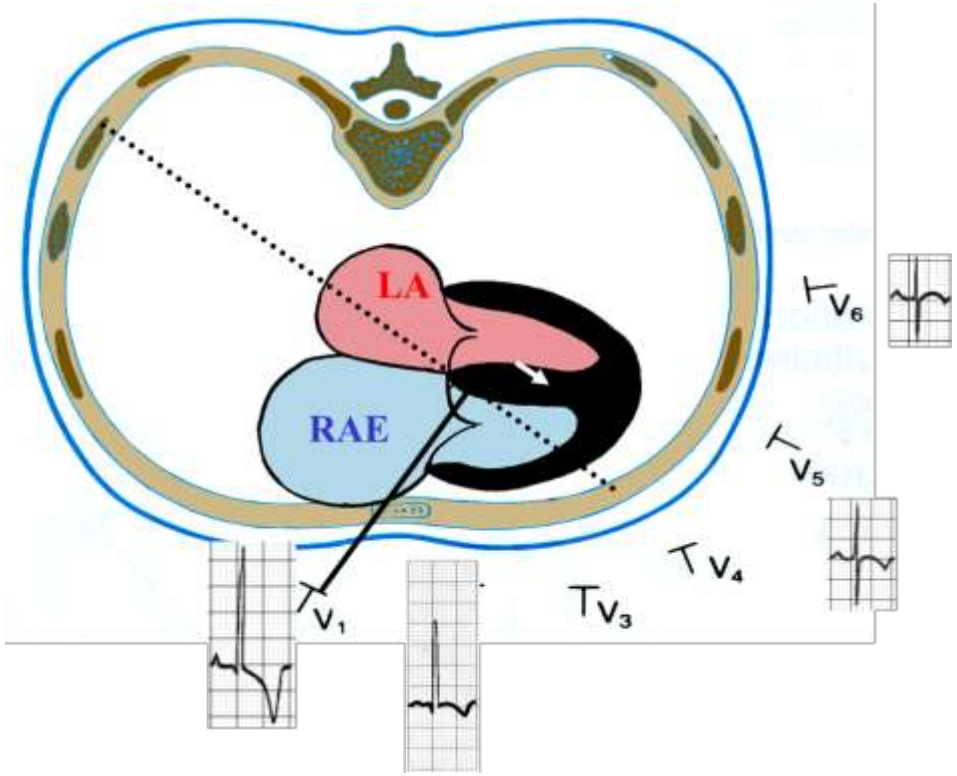
1. *Severe-extreme systolic RVE/RVH (**Gandhi 1962**) (supra-systemic intraventricular pressure inside right ventricle)* with strain pattern of repolarization: Ex critical Pulmonary valve stenosis and intact ventricular septum. The direction of the initial QRS vector on the X axis is helpful in predicting severity. With X initial vector to the left and located in negative hemifield of V1. The right intraventricular pressure is frequently but not necessarily suprasystemic. (**Mehran-Pour 1979**) **(A)**

2. *Right Atrial Enlargement: qR pattern in V_1 may be an indirect sign of RAE* **(Sodi signal)** This occurs because the electrode of V_1 registers the electrocardiographic pattern from within the right atrium (**Sodi-Pallares 1970**; **Sodi-Pallares 1959**). Important dilatation of the right atrium: E.g.: Ebstein´s anomaly (**Lowe 1968**), tricuspid insufficiency. The volumetric increase of the RA gets it closer to the exploring electrode of V_1, registering negatively initially in this lead, because the electrode records the epicardial morphology of the RA. **(B)**

A



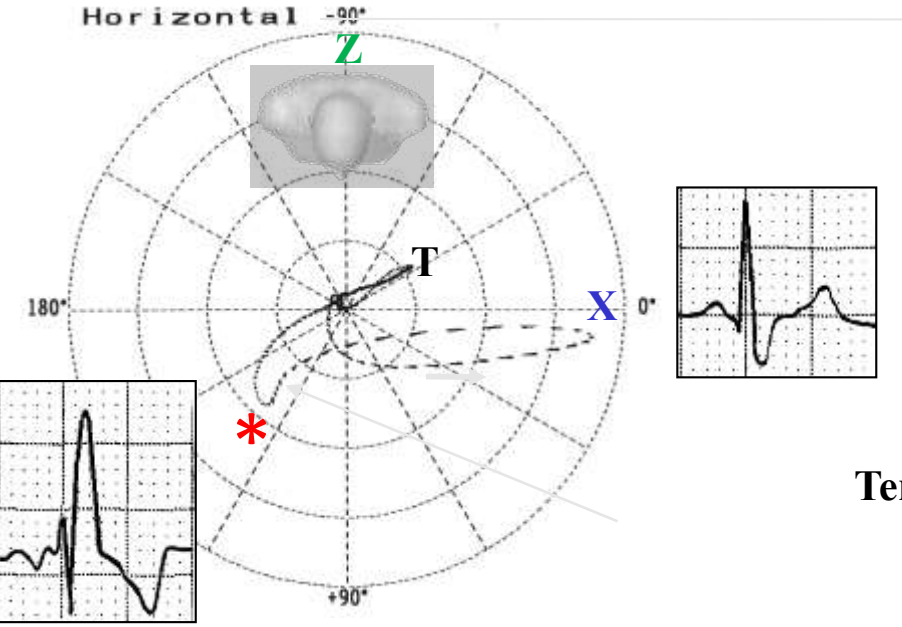
B



qR, QR or qRs in V_1 and V_2. It is an indirect sign of RAE

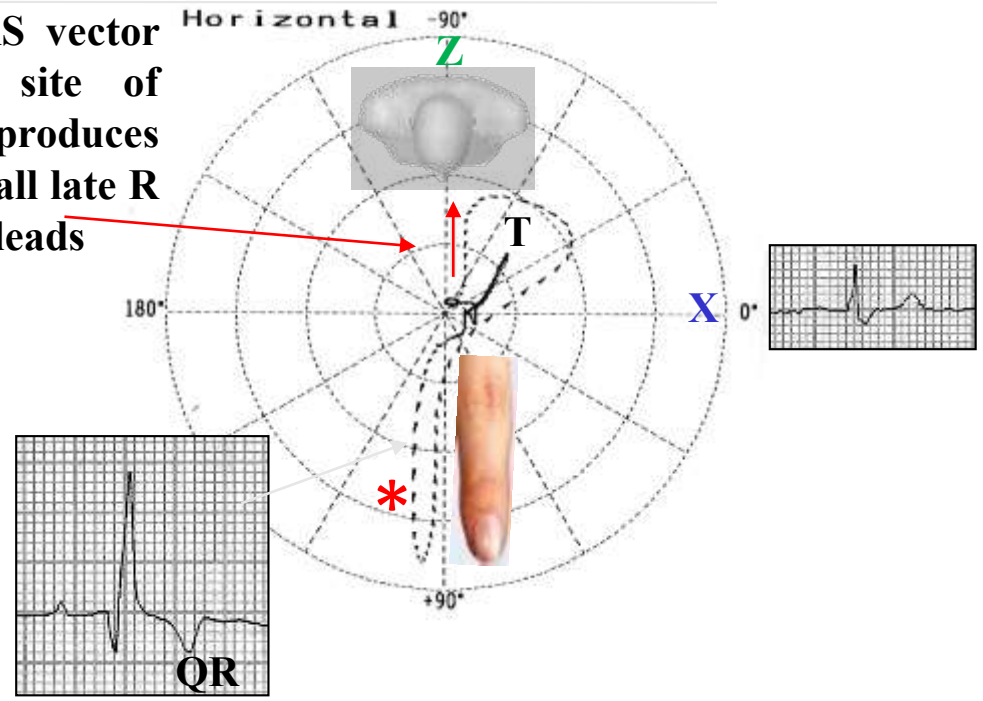
*3. Complete RBBB complicated with anterior or anteroseptal Myocardial Infarction (**Sodi-Pallares 1952; Rudiakov 1964**)*

Uncomplicated RBBB(Cabrera type VCG)



RBBB associated with anterior MI

The initial 40ms QRS vector points away from site of infarction and so produces abnormal Q waves. Tall late R wave appear in these leads



*

Terminal fingerlike appendage

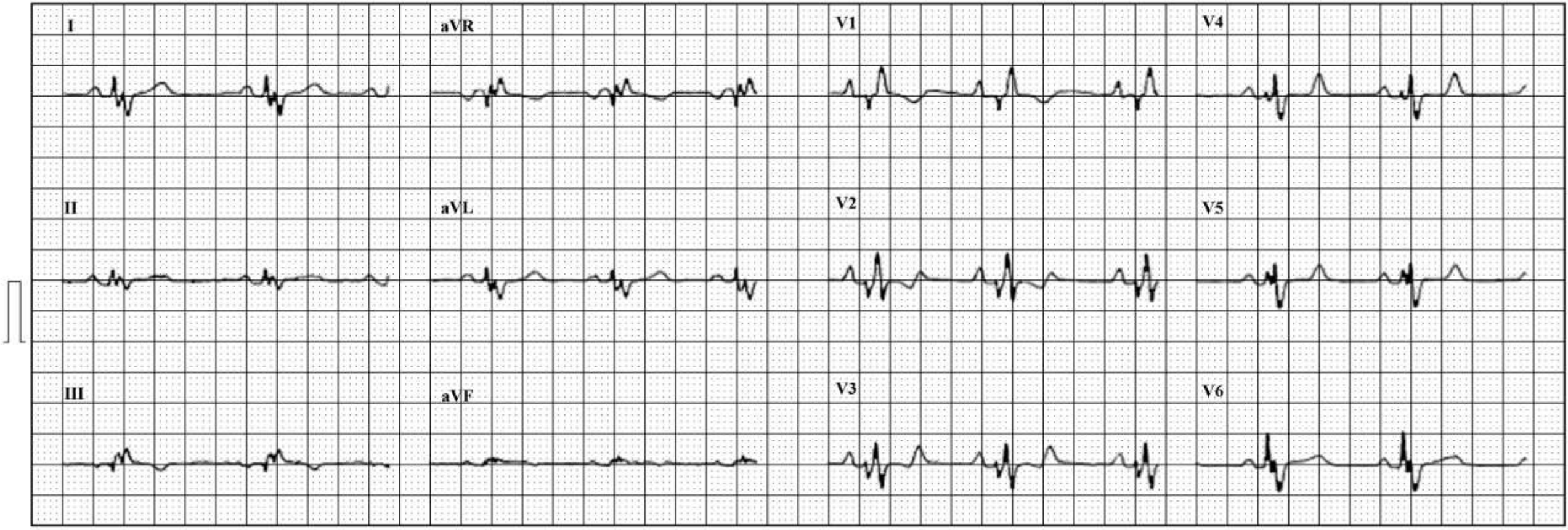
7% of patients presenting with NSTEMI and 4% of patients with STEMI have a RBBB. Patients with RBBB are older, more often have comorbidities, and less often receive short-term in hospital treatment according to guidelines. In NSTEMI, RBBB is not an independent predictor of mortality in multivariate analysis. This is in contrast to the findings in STEMI where RBBB is independently associated with increased inhospital and long-term mortality. (**Kleemann 2008**)

Anterior MI with RBBB: Conventional criteria

1) ***QRS duration ≥ 120ms;***
2) ***Lead I and the left precordial leads show wide final S wave;***
3) ***Final broad R wave in aVR;***
4) ***Prolonged R-wave peak time in V1-V2;***
5) ***The right side precordial leads from V1 to V3 or V4 show diagnostic signs of infarction. There is loos of the R waves in these leads. However, the QRS patterns are modified by the presence or RBBB, so that in addition to the abnormal Q waves, tall late R waves appear in these leads. When the infarct I more extensive , abnormal Q waves may also appear in leads I and aVL and the left precordial leads.***

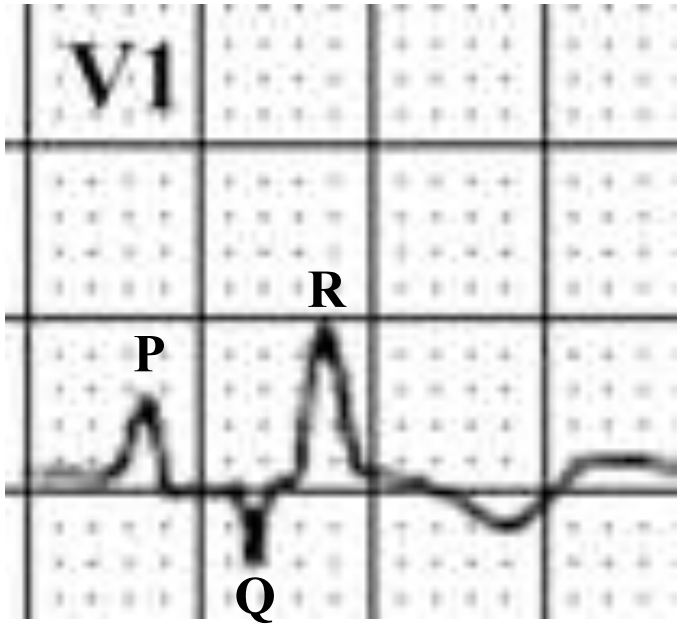
	Uncomplicated RBBB	RBBB associated with anterior MI
Initial 40 ms deflection	It is recorded to the right and anteriorly, just as normally.	It is directed to back.
Right precordial leads QRS pattern V2-V3:	Triphasic rSR`.	Biphasic QR or qR.
Final 60-80 ms QRS forces	In the right anterior quadrant, these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

4. *Ebstein's anomaly: bizarre and low voltage RBBB with initial q wave IN v1 (Kumar 1971)*

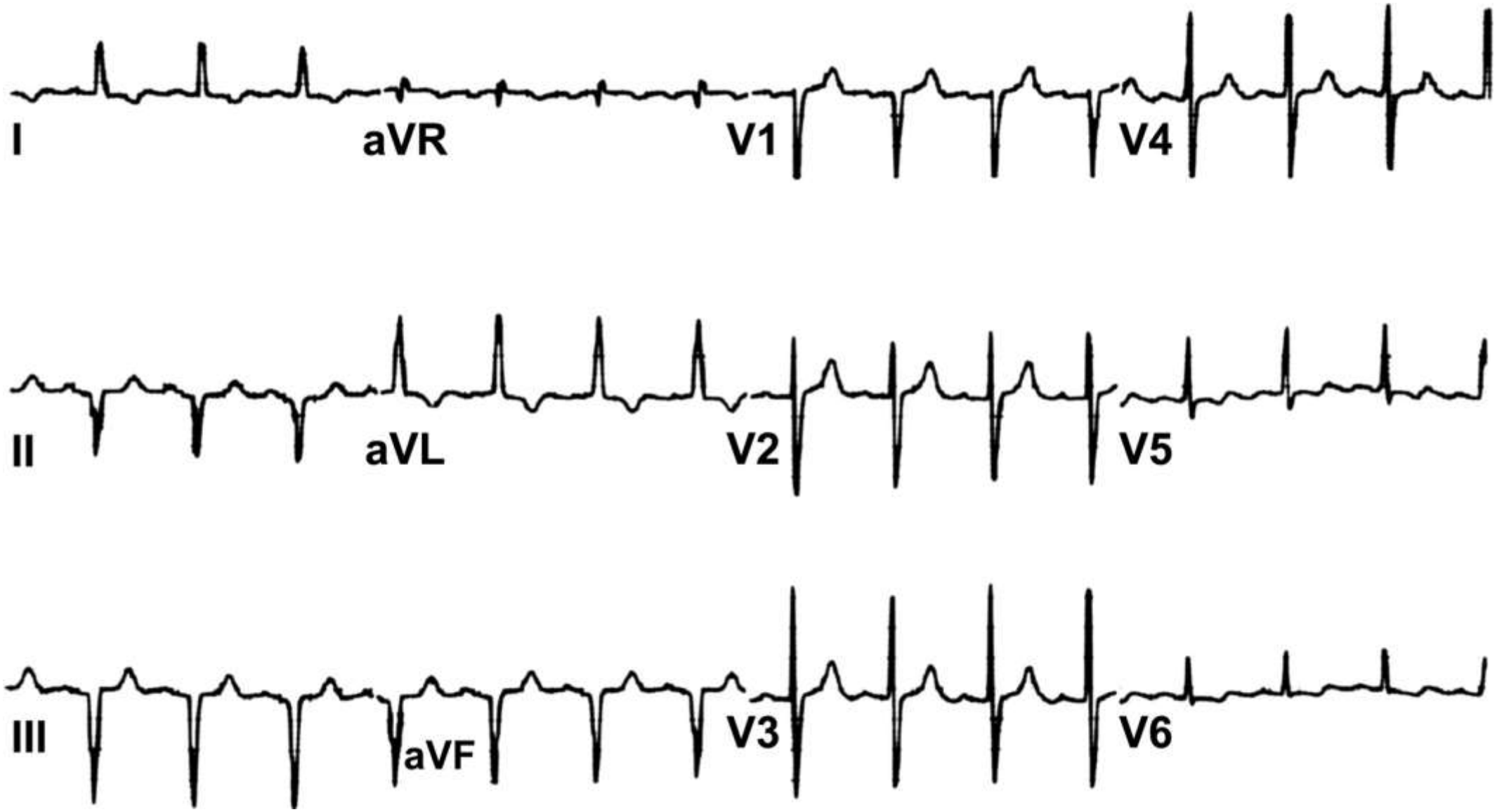


Sinus rhythm; HR: ± 60bpm; PRi = 180ms; indeterminate QRS axis in the frontal plane. qR in V1 with apiculate P wave in this derivation (right atrial enlargement). Intraventricular conduction disturbance characterized by a notched and bizarre QRS pattern, with two components (RV and LV).

Himalayan P-wave and bizarre QRS pattern with two components (RV and LV): QR pattern in V1



5. *Congenitally Corrected Transposition: Secondary to inversion of septal activation, RAE, by progressive tricuspid regurgitation that occurs with age and associated with deterioration of RV function (**Warnes 2006; Ruttenberg 1966**)*



ECG of a patient with C-TGA. Septal activation occurs from right to left, and therefore Q waves are seen in the right precordial leads II and III, but no Q waves are seen in V_5-V_6, I and aVL.

5. *Endomyocardiofibrosis (**Tobias 1992**)*

6. Acute pulmonary embolism (APE) (**Kukla 2011**)

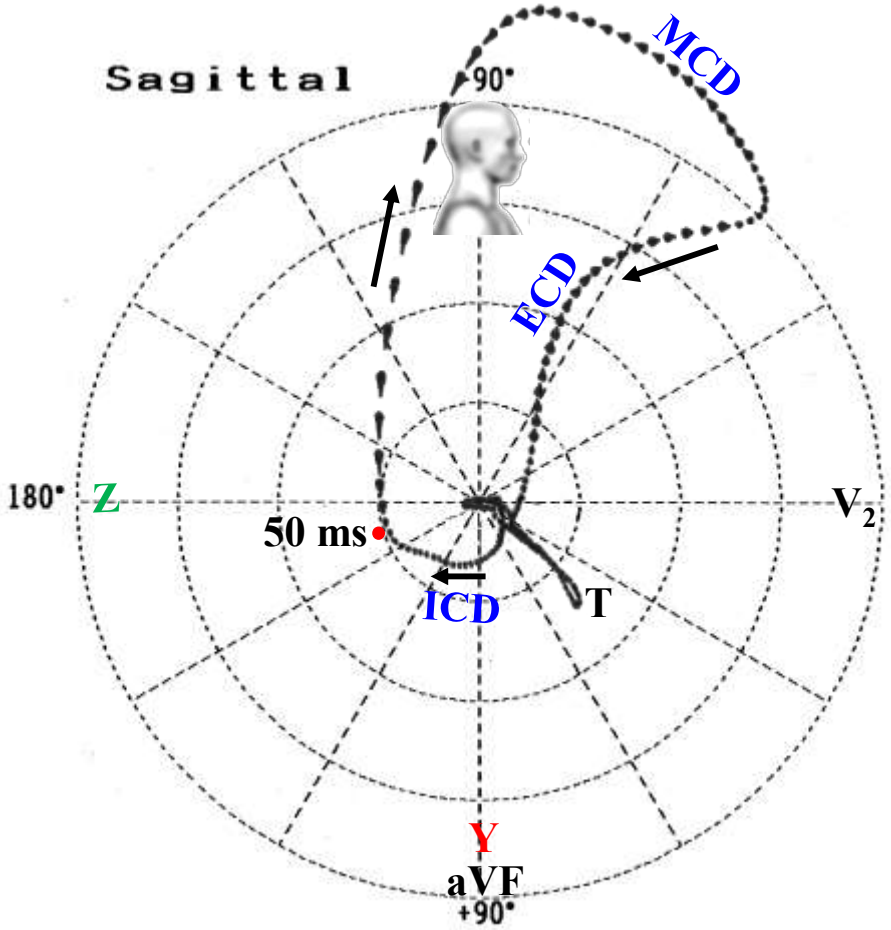
7. *MI or ischemia / injury associated with LSFB. S-T elevation and increase in R-wave voltage, "giant R waves", and concomitant shift of the frontal QRS axis toward the locus of injury is also displayed (**David 1982; Deanfield 1983; Pérez-Riera 2011; Pérez-Riera 2016b; Riera 2008a; Riera 2008b**)*

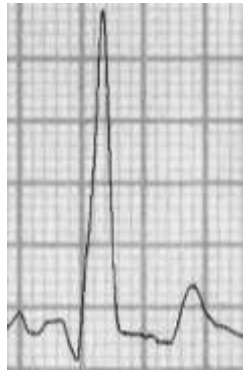
ECG/VCG correlation in the right sagittal plane

QRS loop with the initial 50 ms shows comets very close to each other (slow speed) and directed to down and backward (Q-wave >40 ms in V2: **ICD**).

QRS loop located predominantly in the superior quadrants with **MCD** and **ECD** (the last 56 ms) in the top anterior quadrant. T-loop elongated and directed to front and downward, explaining the minus-plus T-wave in V2.

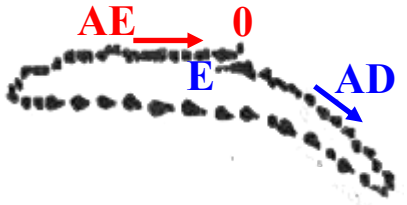
Conclusion: biatrial enlargement (BAE), anterolateral MI, LAFB.

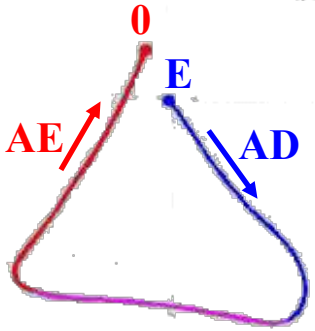




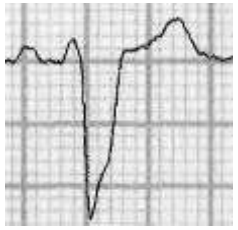
qR

Magnified P-loop





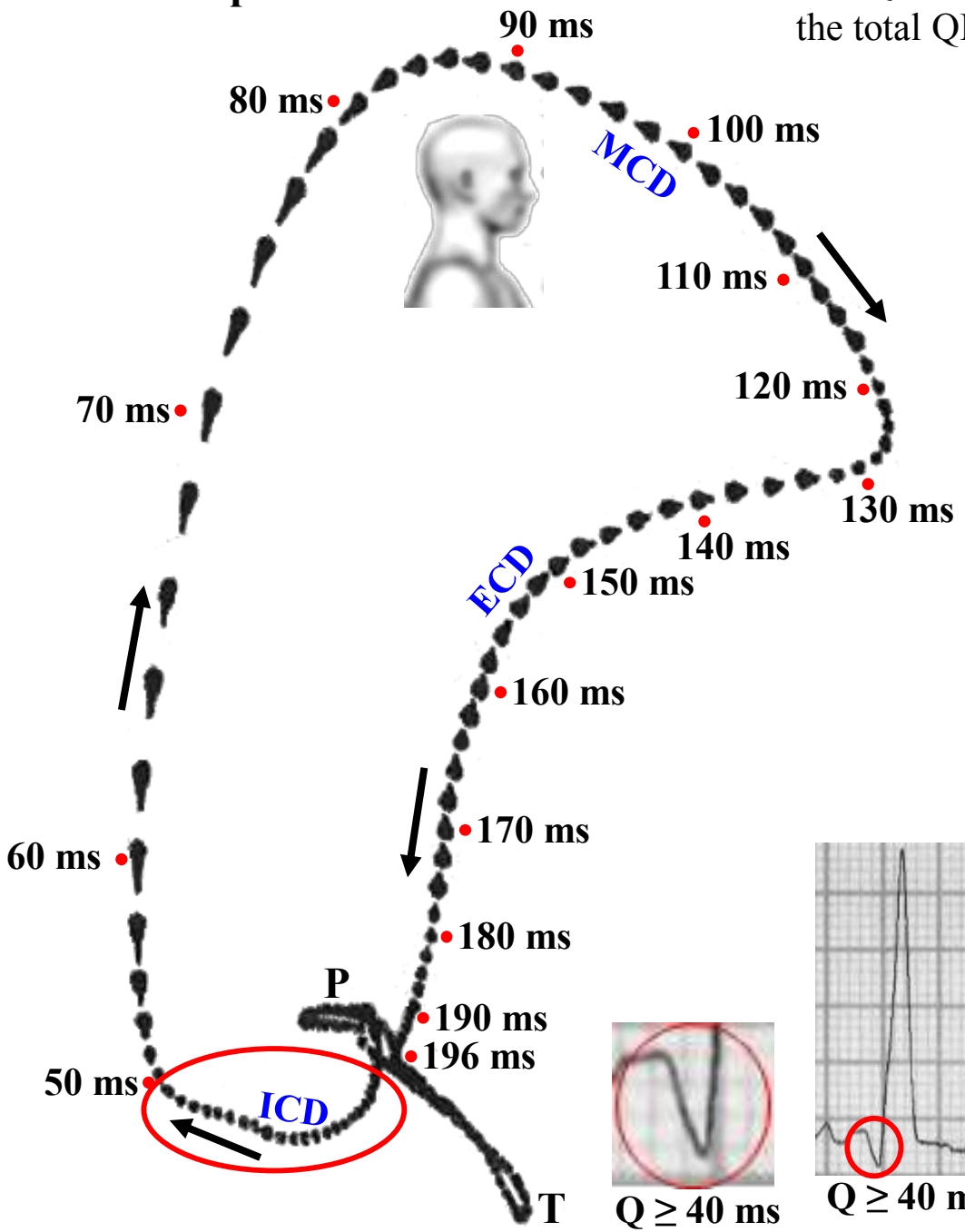
BAE



rS

ICD: Initial Conduction Delay
MCD: Middle Conduction Delay: intramural conduction delay
ECD: End Conduction Delay

The loops in the RSP



Q ≥ 40 ms

Q ≥ 40 ms

The QRS loop has a very long duration. We counted 98 comets, and as each comet lasts 2 ms, the total QRS duration is 196 ms.

Meaning according to the location of the conduction delay in the QRS loop

1. **Initial conduction delay on QRS loop** = *Preexcitation, WPW syndrome/ delta wave and initial Q-wave of MI (≥40 ms).*
2. **Middle and End conduction delay** = *Complete Left Bundle Branch Block.*
3. **End conduction delay on QRS loop** = *Complete or incomplete Right Bundle Branch Block.*
4. **Uniform conduction delay** = *Hypercalemia; quinidine effect; intra-infarction, intramural*
5. **Initial, middle and end conduction delay** = *the present case.* ***ICD*** *consequence of MI Q-wave,* ***MCD*** *consequence of intramural conduction delay (old peri or intra infarction conduction delay),* ***ECD*** *caused by RBBB. In these cases, between ≈ 50 and 80 ms with normal conduction.*

Each comet stretches for 2ms (0.0002 s)

The "head of the comet" points the direction of the rotation

ICD: Initial Conduction Delay
MCD: Middle Conduction Delay: intramural conduction delay
ECD: End Conduction Delay

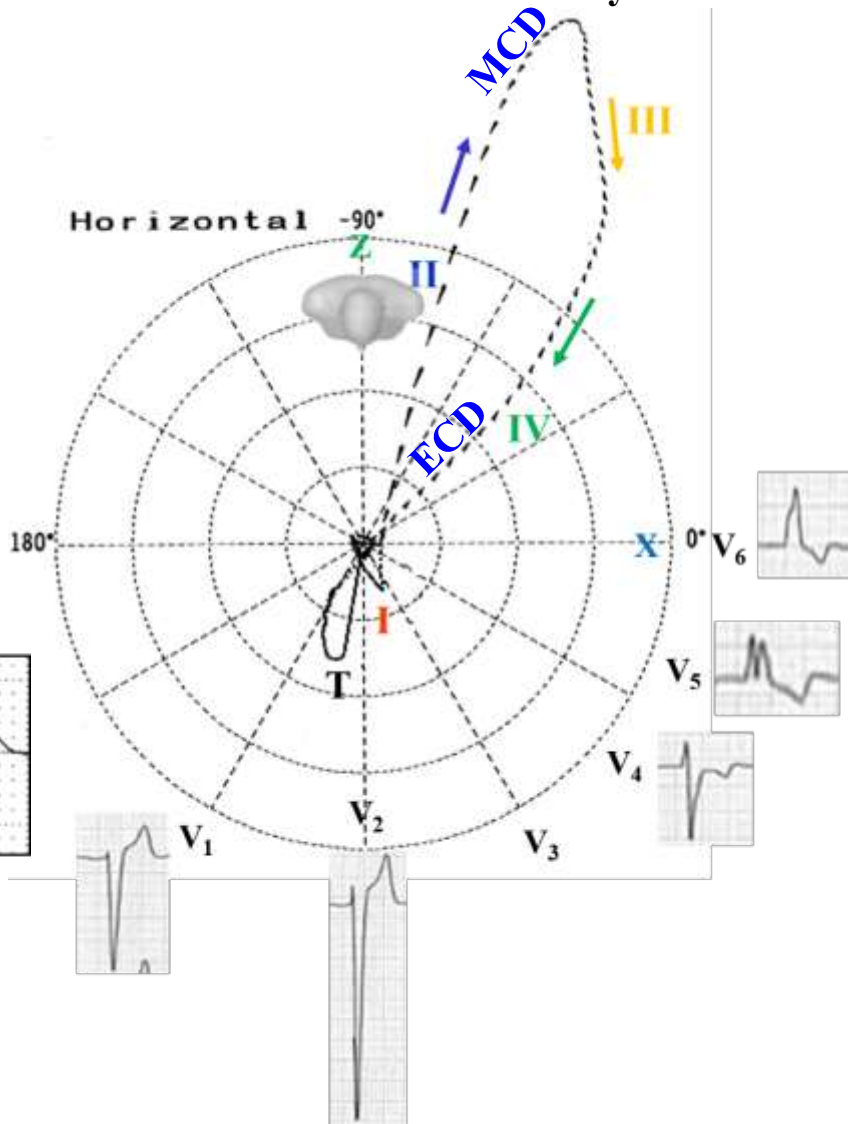
Determination of conduction velocity of stimulus

The greater or the lesser distance between comets indicates the greater or the lesser conduction velocity in the area. Thus, when they are very close to each other, it indicates the presence of conduction delay. To consider the phenomenon as true, it is necessary for it to be evident in at least 2 planes.

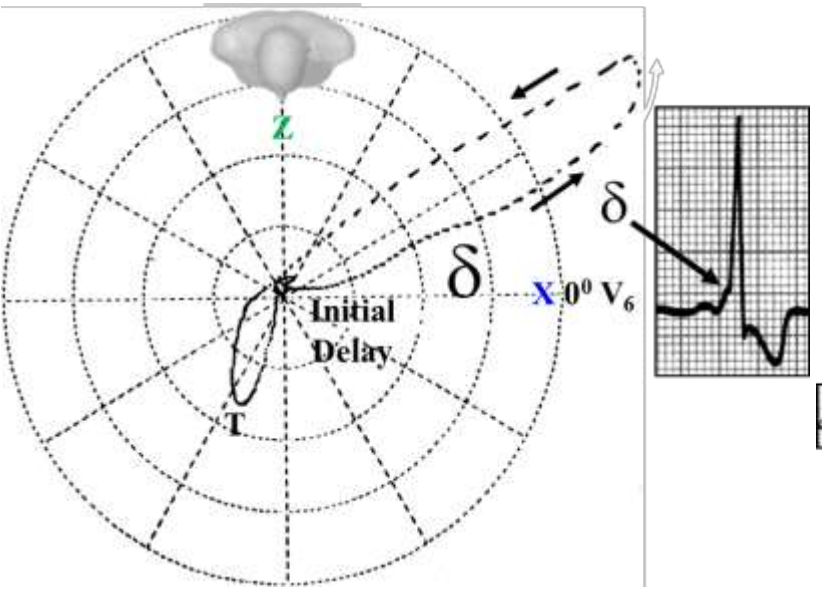
Separate comets = faster

Very close comets = slower

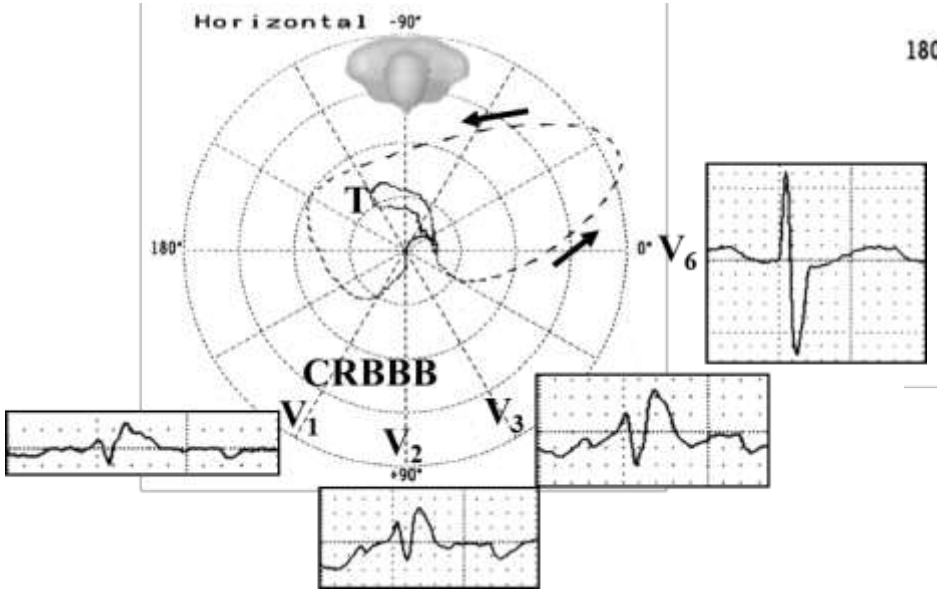
Middle + End conduction delay: LBBB



Initial conduction delay (ICD): WPW



End conduction delay (ECD): RBBB



ECG/VCG diagnosis:

1. LAE: prolonged P duration (120 ms), bimodal P-wave in I and II, Maximal Posterior Forces ≥ 0,05mV, Maximal Left Forces ≥ 0,01mV in the P-loop of HP.
2. RAE: because the P-loop in the RSP has increased anterior and posterior forced.
3. Biatrial enlargement (BAE) or combined atrial enlargement: consequence of item number 1 and 2.
4. Complete RBBB: broad QRS in sinus rhythm, final wide R-wave in aVR (qR complex), broad S-wave in I and qR in the right precordial leads, prolonged R-wave peak time in V1.
5. Atypical LAFB: extreme QRS deviation to the upper quadrants, SIII>SII.
6. Anterolateral transmural infarction: QS from V3 to V6.
7. Probable anterolateral left ventricular aneurysm (LVA): persistent ST segment elevation from V3 to V6 after long time AMI. some degree of ST elevation remains in 60% of patients with anterior STEMI and 5% of patients with inferior STEMI. The mechanism is thought to be related to incomplete reperfusion and transmural scar formation following an acute MI. This ECG pattern is associated with paradoxical movement of the ventricular wall on echocardiography (ventricular aneurysm). The definition of a LVA remains controversial. A true LVA is defined as a well delineated, thin, scarred, or fibrotic wall, devoid of muscle or containing necrotic muscle, that is a result of a healed transmural MI. The involved wall segment is either akinetic (without movement) or dyskinetic (with paradoxical ballooning) during systole, and collapses inward when the ventricle is fully vented during surgery. Aneurysms of the apex and anterior wall are more than four times as common as those of the inferior or inferolateral walls. It was previously estimated that LVA develops in up to 30 to 35 percent of patients with Q wave MI (**Mills 1993; Kirklin1993**). However, the incidence of this complication is decreasing, and currently is about 8 to 15 percent in such patients (**Glower1997**). This change is related to the introduction of major improvements in the management of patients with acute MI (**Tikiz 2001**). Although the collateral perfusion existing at the onset of acute myocardial infarction may not improve ventricular function, it exerts a beneficial effect on the prevention of left ventricular aneurysm formation (**Hirai 1989**).
8. On QRS loop we observe: Initial Conduction Delay**(ICD)** represent the necrotic Q wave, Middle Conduction Delay**(MCD)** consequence of focal, intramural conduction delay or peri-intrainfarction block (the ECG abnormality is associated with and old myocardial infarct and is caused by delayed activation of the myocardium in the region of the infarct) and end conduction delay**(ECD)** caused by the right bundle branch block

Management

ACE inhibitors or ARBs, plus β-blockers

These drugs reduce stress hormones that can cause further weakening of the heart muscle and disease progression. They also decrease the amount of work required for the heart to pump effectively. Everyone with low LVEF should be on both medications, if they can tolerate them. Starting these drugs early will prolong the phase of their disease in which they feel good and have good quality of life. However, many patients do not tolerate these drugs in the high-dose combinations used in clinical trials. For example, ACE inhibitors and ARBs can cause a dry cough, while β-blockers can make some people feel drowsy.

Diuretics

Diuretics such as furosemide and torsemide help the body eliminate excess fluid that can cause swelling or shortness of breath. Although the reasons why heart disease impairs the body's ability to maintain fluid balance are complex, most people who require a diuretic need it indefinitely. This is one of the most important drugs to individualize, since the optimal dose can vary from 10mg to 400 mg a day, depending on how well the kidneys are working.

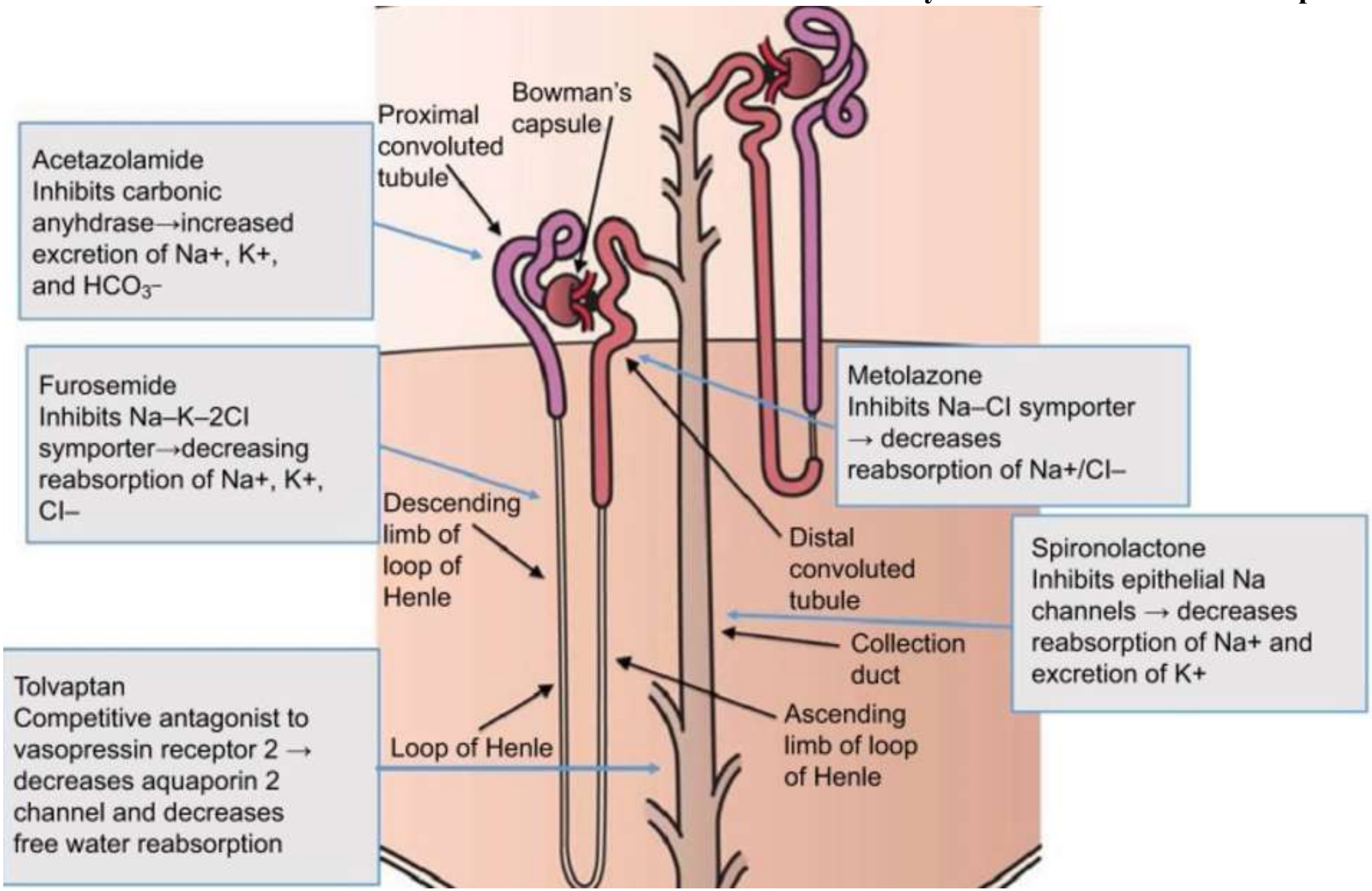
Hydralazine-nitrate

This is an additional drugs to consider. This combination of blood pressure–lowering medications can be added when someone taking an ACE inhibitor or ARB plus a β-blockers continues to have severe symptoms. The combination was studied extensively in African Americans with HF(The A-HeFT trial), many of whom experienced increased survival and quality of life and a decrease in the number of hospitalizations. Since that time, it has proved useful in people of other races. It has been suggested that hydralazine might enhance nitric oxide (NO)-mediated effects of organic nitrates by decreasing superoxide (O (2) (-)) formation, one of the factors inducing NO resistance. On the other hand, Chirkov et al. do not support the assumption that hydralazine could be viewed as a "NO enhancer“. These authors observed that there is no evidence of attenuation of NO resistance by hydralazine treatment.(**Chirkov 2010**)

Spironolactone or eplerenone

These agents primarily decrease the formation of abnormal fibers that make the heart stiffer and less elastic, although they also help eliminate excess fluid. People who take these drugs are at higher risk for developing very high K^+ levels, which can be fatal. Kidney function must be carefully reviewed before either agent is prescribed, and both kidney function and K^+ levels must be monitored during therapy.

Schematic of sites of action and mechanism of ion channel blockade by selected diuretics in the nephron



New drugs approach:

LCZ-696 Sacubitril + Valsartana (Entresto®) is a new drug for HF. In HF with reduced LVEF. This is the first approved angiotensin receptor-neprilysin inhibitor, that is superior to an ACE inhibitor. LCZ-696, sacubitril/valsartan, is a dual-acting molecule consisting of the angiotensin II (Ang II) receptor blocker valsartan and the neprilysin (neutral endopeptidase) inhibitor AHU-377 with significant beneficial effects in patients with hypertension and HF. Recent studies have demonstrated a higher effectiveness of LCZ-696 compared to valsartan in the treatment of hypertension and HF. The rationale for the development and the FDA approval of LCZ-696 was based on the concept of an additive effect of the Ang II receptor blocker valsartan and the neutral endopeptidase (neprilysin) inhibitor AHU-377 for the treatment of hypertension and HF. The synergism from these drugs arises from the vasodilating effects of valsartan through its blockade of Ang II type 1 receptor and the action of natriuretic peptides atrial natriuretic peptide and B-type natriuretic peptide (BNP) by preventing their catabolism with neprilysin resulting in increase of cyclic guanosine monophosphate. This action of neprilysin is associated with increased natriuresis, diuresis, and systemic vasodilation,. since these peptides have been shown to have potent diuretic, natriuretic, and vasodilating effects. In addition, it reduces the levels of N terminal pro-BNP. Therefore, administration of LCZ-696 results in significant reduction of wall stress from pressure and volume overload of the LV as demonstrated by the reduction of Nterminal pro-BNP, both significant constituents of hypertension and HF, and it is safe, well tolerated and is almost free of cough and angioedema.(**Li 2017**).

2) Implantable Cardioverter-Defibrillator

Recently, the Canadian Cardiovascular Society/Canadian Heart Rhythm Society published new Implantable Cardioverter-Defibrillator Guidelines. *Main conclusions of their experts*: Canadian expert recommend that patients with persistent LV dysfunction due to either ischemic or non-ischemic cardiomyopathy and LVEF ≤30 % receive an ICD, where persistent refers to at least 3 months of optimal medical therapy in all patients and, in patients with ischemic heart disease, at least 3 months post revascularization and at least 40 days following a MI (**Strong recommendation; High quality evidence**). Additionally, they suggest that an ICD may be considered for patients with persistent LV dysfunction due to either ischemic or non-ischemic cardiomyopathy and LVEF 31-35 % where persistent refers to at least 3 months of optimal medical therapy in all patients and, in patients with ischemic heart disease, at least 3 months post revascularization and at least 40 days following a MI (**Weak recommendation; Moderate quality evidence**) (**Bennett 2017**). The authors also recommend that patients likely to have LV dysfunction 3 months after revascularization for MI or 40 days post MI without revascularization undergo an assessment of LVEF at those time points (**Strong recommendation; Low quality).**

Practical tip: As the LVEF may worsen in the first 3 months following a MI, the authors recommend repeat assessment of LVEF (40 days or 3

months following MI depending on the clinical circumstances) for patients with an LVEF ≤ 45% at the time of or immediately following an MI. The method of EF assessment depends on local expertise and the reliability and accuracy of available assessment methods.

They suggest that an ICD be considered in patients with an acute indication for cardiac pacing within 3 months post revascularization or 40 days of MI and prior to achieving optimal medical therapy when there is a high probability that the LVEF will remain below 35% (**Weak recommendation; Low quality evidence**) (Optimal medical therapy refers to evidenced based HF therapies at their maximally tolerated doses).

Practical tip: There is a high probability that the LVEF will remain below 35% if there is a high burden of scar on MRI, minimal myocardial viability, the LVEF was significantly low despite a minor biomarker rise at the time of a MI or when revascularization addressed only a small amount of myocardium. They recommend an ICD not be offered to patients where comorbidities make it unlikely that an ICD will substantially increase a patient's life expectancy (**Strong recommendation**; **Moderate quality evidence**). They also recommend that an ICD be replaced by a pacing system, removed or abandoned at the time of ICD generator end of life/service in patients with life threatening comorbidities, at the request of the patient or when an ICD is unlikely to increase life expectancy (**Strong recommendation; Low quality evidence**).

Practical tip: Validated risk calculators/risk assessment tools such as the Charlson Comorbidity index, the Seattle HF Model or the SHOCKED or MADIT risk scores can be of aid in the estimation of each patient's benefit/risk of an ICD.

The authors recommend an ICD for patients following cardiac arrest due to ventricular arrhythmias in the absence of a reversible cause (**Strong recommendation; High quality evidence**). They also suggest an ICD be considered post MI in the presence of sustained VT or VF >48 hours post MI or >48 hours after revascularization for MI in the absence of reversible causes (**Weak recommendation; Low quality evidence**) (Sustained VT refers to hemodynamically significant VT or VT greater than 30 seconds in duration).

Finally, recommend an ICD for patients with sustained VT in the presence of significant structural heart disease in the absence of reversible causes (**Strong recommendation; Moderate quality evidence**).

The authors suggest an ICD be considered in patients with significant structural heart disease and syncope of unknown origin where the cause of syncope is most likely VT/VF and where the risk of recurrent VT/VF is high (**Weak recommendation; Moderate quality evidence**). They recommend an ICD for patients with syncope of unknown origin with inducible VT at EPS and structural heart disease (**Strong recommendation; Moderate quality evidence**).

Practical Tip: All patients with a history of VT or VF should undergo evaluation for reversible causes of ventricular arrhythmias. Depending on the clinical circumstances this will likely include, but not be limited to, evaluation for coronary artery anomalies, ischemia or acute MI immediately preceding the arrhythmic arrest (angiogram or CT coronary angiogram), structural heart disease (Echo, MRI or PET) or risk of recurrent arrhythmias (electrophysiology testing).

Cardiac Ressincronizaton Therapy is not indicated in this case. Why?

Explanation: The clinical application of CRT began in 1994 when the first cases of atrio-biventricular pacemaker implantations in patients with severe CHF were described (**Cazeau 1994; Bakker 2000**). The surface ECG of these patients often showed a prolonged PR interval and a widened QRS complex due to ventricular conduction disturbances.

The first randomized crossover trial investigating the clinical efficacy of CRT was the Multisite Stimulation in Cardiomyopathy (MUSTIC) study (**Linde 2002)**. This trial in patients with chronic severe HF NYHA III, reduced LVEF <35 % and a broad QRS complex (>150 ms), showed that biventricular (BiV) pacing improved the 6-min walking distance, peak oxygen uptake, quality of life score, and NYHA class. The multi-center insync randomized clinical evaluation (MIRACLE) study confirmed these results in patients with a QRS duration ≥130 ms (**Abraham 2002; Sutton 2006**). This study also showed a clear reduction in LV volumes, reduced HF hospitalization, and better survival. Similar results were shown by the COMPANION (**Bristow 2004**) and the cardiac resynchronization (CARE)-HF (**Cleland 2005**) trials, which included patients with QRSd ≥120 ms and NYHA class III–IV. These favorable and consistent results led to the recommendation of CRT in patients in NYHA class III–IV despite optimal medical treatment, with a reduced LVEF (<35 %), in sinus rhythm, and a wide QRS complex (≥120 ms) (**Vardas 2007**).

Subsequent trials investigated the effect of CRT in less symptomatic patients (the resynchronization reverses remodeling in systolic left ventricular dysfunction (REVERSE) (**Linde 2008**), multicenter automatic defibrillator implantation trial (MADIT)-CRT (**Moss 2009**), and resynchronization/defibrillation for ambulatory heart failure trial (RAFT) trials (**Tang 2010**). Again, LV function improved, and both all-cause mortality and non-fatal HF events improved. However, subgroup analyses of these three trials demonstrated that these effects were predominantly confined to patients with a QRS duration ≥150 ms (**Bryant 2013**). This evidence resulted in the addition of a class I indication to CRT for patients presenting with NYHA class II, a reduced LVEF, and a QRS duration >150 ms, in the 2010 guidelines (**Dickstein 2010**). Interesting and important, however, is that the definition of complete LBBB from the 12-lead ECG varies between European and American guidelines and between large clinical trials (**Zareba 2011; Gold 2012**) or studies (**Strauss 2011**) that investigated LBBB as a predictor of CRT effectiveness. The refinement of LBBB morphology with the presence of notching or slurring in at least 2 contiguous leads appears to significantly improve the prediction of CRT response and clinical outcome, at least in small single-center studies (**Tian 2013; van Deursen 2015**). While QRS morphology is now one of the primary indicators for CRT, a recent meta-analysis, combining data from CARE-HF, MIRACLE, MIRACLE ICD, REVERSE, and RAFT showed that QRS duration is a more powerful predictor of CRT outcomes (mortality and morbidity) than QRS morphology (**Cleland 2013**).

This conclusion is in contrast to several reports derived from some of the individual trials and to a meta-analysis of the MADIT-CRT, RAFT, and REVERSE study (**Zusterzeel 2014**). One possible explanation for this discrepancy is the use of “liberal” LBBB criteria. In that case, it is likely that QRS duration provides additional information. Indeed, when using “liberal” LBBB criteria the non-LBBB patients tended to have a lower

QRS duration than the LBBB patients (**Zareba 2011**), but this difference could not be observed when stricter LBBB criteria were used (**Tian 2013**). Furthermore, in the studies where strict LBBB criteria as defined by Strauss et al. (**Strauss 2011**) were used, QRS duration was not a predictor of response while LBBB was (**Tian 2013; Mascioli 2012**).

In conclusion, currently it is not clear whether QRS duration or morphology should be preferred as primary marker for selection of CRT patients. QRSd may not be specific, but LBBB criteria may be too complex and/or dependent. In order to come to a possible solution, it may be worthwhile to go back to the basic physiology of dyssynchronous HF and the mechanisms of CRT.

Based on the evidence obtained from electro-anatomic mapping that QRS_{AREA} reflects LV activation delay, the primary electrical substrate for CRT, and on the better prediction of CRT response by QRS_{AREA}as compared to QRSd, Engels et al propose to include QRS_{AREA} in the guidelines as a selection criterion for CRT implantation. The possibly even better prediction of CRT response by using the T-wave rather than the QRS complex requires further investigation (**Engels 2016**).

Electrocardiographic parameters associated with low LVEF in the presence of LBBB

1. P wave duration ≥ 120ms
2. Final negative component of the P wave in V1 with positive Morris criterion (deep and slow)
3. QRS electric axis in the frontal plane with extreme deviation to the left (> 30º) or to the right (> +90º Norwest no man no land)
4. QRS duration ≥ 160ms
5. Deep S waves in V3-V4
6. Notches in the lower leads.

Markers of poor response to cardiac resynchronization therapy

1. Masculine sex: The present case
2. RBBB pattern: The present case
3. Narrow QRS complex
4. Co-morbidities
5. Chronic Renal Failure: creatinine clearance <30ml / min / m2
6. Chronic Obstructive Pulmonary Disease (COPD)
7. Anemia
8. Pulmonary hypertension
9. Coronary artery disease: The present case
10. Significant cardiomegaly: The present case
11. Previous LVEF ≤ 23% The present case
12. Previous functional class IV The present case
13. Ischemic patients in acute myocardial infarction (**Van Bommel 2009**)
14. LV end diastolic diameter> 75mm: severely dilated and remodeled ventricles. The present case
15. Severe mitral regurgitation
16. Scars in the area of the electrode implant (high thresholds) or large scars that affect> 50% of the VE The present case
17. Unadjusted programming of AV and VV intervals With mitral Doppler separation of E / A waves with AV interval optimization

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