

*Hombre de 58 años de edad, con infarto de pared anterior extenso antiguo,
sincope y muy baja fracción de eyección*

**Male 58 years old with old extensive anterior wall myocardial infarction,
syncope and very low left ventricle ejection fraction**

Dr Atilio Marcelo Abud M.D.

Estimado Dr Pérez Riera:

Paciente de 58 años, masculino, antecedente de infarto de miocardio anterior extenso hace 4 años derivado a nuestro servicio por síncope para implante de un Cardio-desfibrilador automático implantable(CDI).

Se encuentra en clase funcional II. El tratamiento médico es óptimo

Ecocardiograma muestra severa dilatación del ventrículo izquierdo 82.3mm diástole y /75.5 en sistole.

Aurícula izquierda de 46mm.

Grosor de la pared libre 6.8mm y del septum de 11mm.

Hipertension pulmonar 50/25.

Insuficiencia mitral leve a moderada.

Fracción de Eyección del ventrículo izquierdo de 17% confirmada por la Resonancia Magnética Nuclear que demuestra viabilidad miocárdica en territorio laterobasal.

Le enviamos el ECG con un registro de Holter

La duda es la siguiente: ¿ El paciente tiene indicación para implantar un resincronizador cardiaco junto con Cardio-desfibrilador automático implantable? o lo correcto es seria implantar solamente un CDI bicameral o unicameral y programarlo a baja frecuencia para evitar estimulación ventricular derecha?

Un gran abrazo

Dr. Abud Atilio Marcelo

Dear Dr. Pérez Riera:

A 58-year-old male, history of extensive anterior myocardial infarction 4 years ago referred to our service by syncope for ICD implant.

Actually he is in NYHA II functional class. Medical treatment is optimized.

Echocardiogram shows severe left ventricular dilatation (LV diastolic diameter 82.3mm / LV75.5 systolic diameter). Left atrium 46mm. Free wall thickness of 6.8mm and septum 11mm.

Pulmonary hypertension 50/25.

Mild to moderate mitral regurgitation.

Ejection fraction of left ventricle of 17% confirmed by magnetic resonance imaging showing myocardial viability in laterobasal territory.

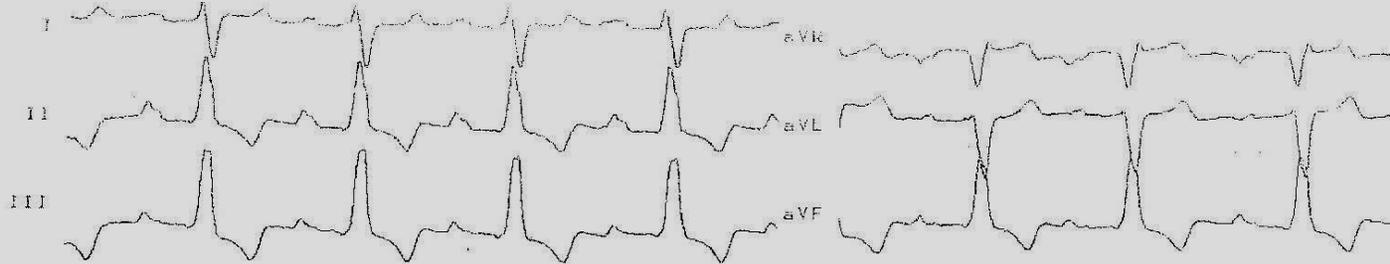
We send the ECG with Holter monitoring

The question is:

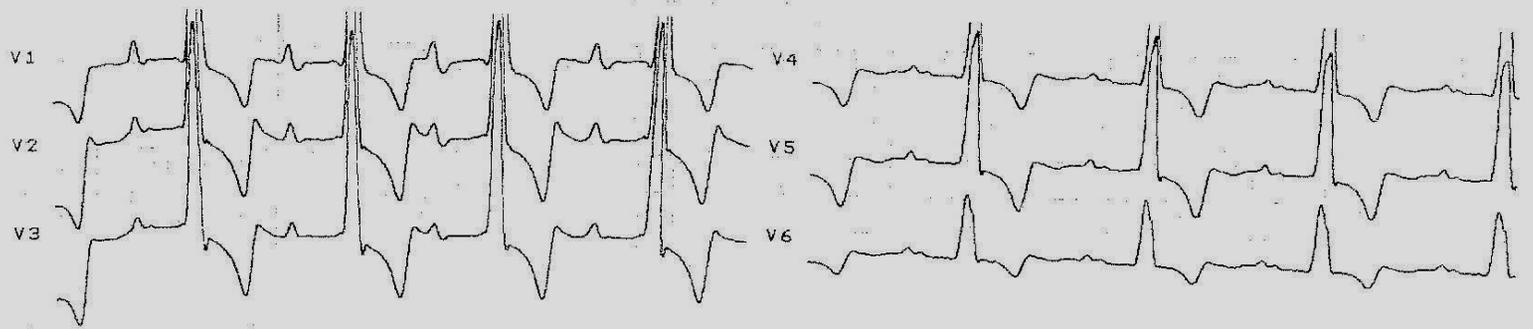
Does the patient have an indication for ICD and cardiac resynchronization therapy (CRT)? or is only an ICD implant would be bicameral or unicameral and low frequency schedule to avoid right ventricular pacing?

- A big hug

Medical Medical Inc.
Patient ID: Auto 0001
Date: 06/07/2013
Time: 11:56:38
Friday
Speed: 25mm/s
Sens: 10mm/mV
Filter: Noise + Mains
Baseline filter: On



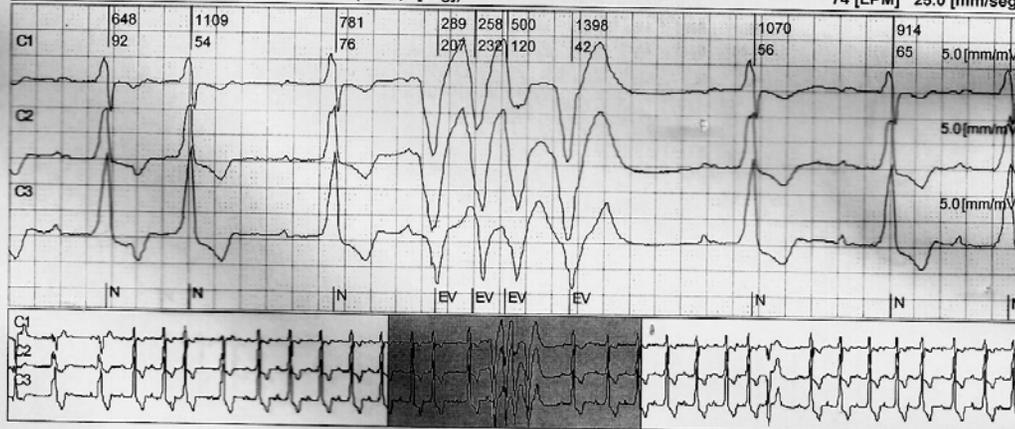
HeartMirror 3-1K0
Version 1.21B



6.7.13 11:56:38

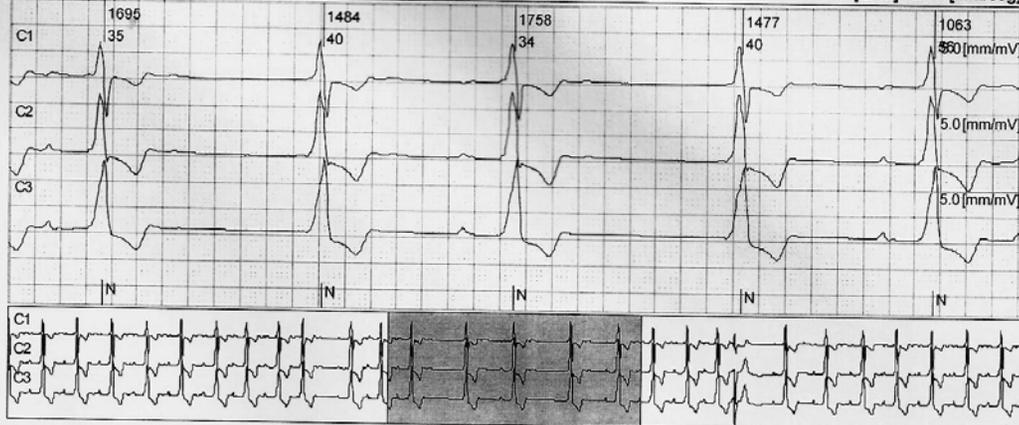
15/05/12 20:42:07 "Taquicardia V 171 LPM (4 Lat, 2[seg])"

74 [LPM] 25.0 [mm/seg]



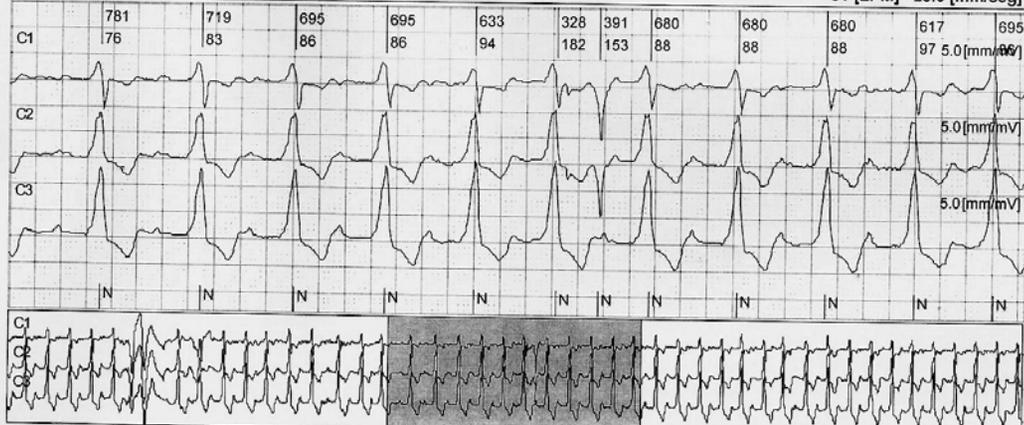
16/05/12 01:02:46 "FC Min 37 LPM"

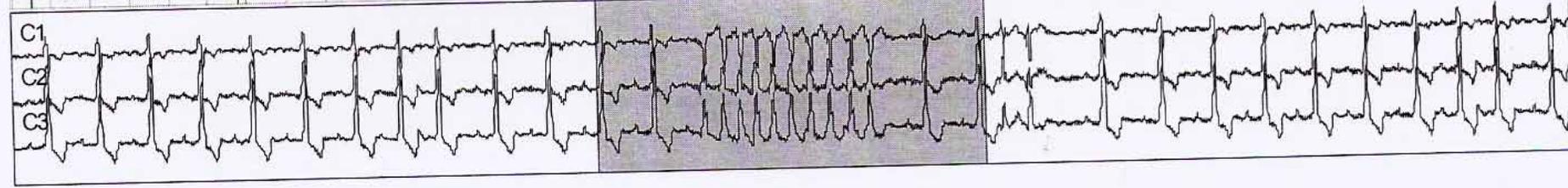
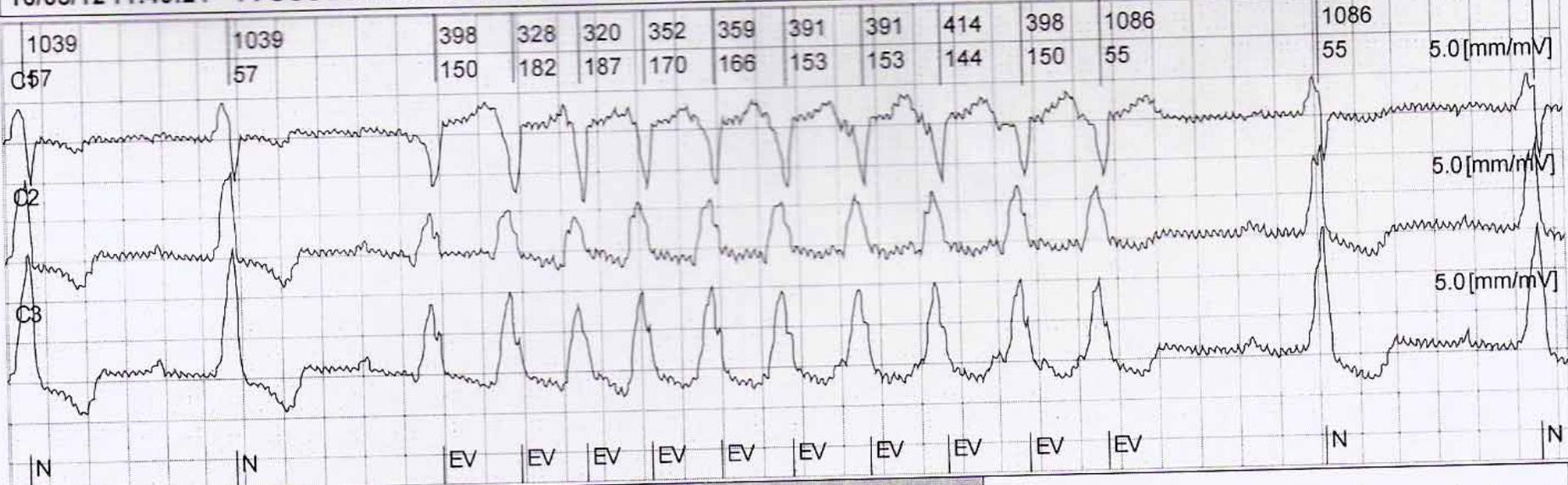
42 [LPM] 25.0 [mm/seg]



16/05/12 07:37:11 "FC Máx 117 LPM"

91 [LPM] 25.0 [mm/seg]





Colleagues opinions

There are a number of problems. First is the syncope with documented paroxysmal (non-sustained) monomorphic VT on the Holter and with the low EF, it is appropriate to be concerned with sustained VT accounting for the syncopal spell.

There is RBBB and marked right axis (Left posterior fascicular block). There is intermittent first degree AV block and also intermittent marked sinus bradycardia on one of the rhythm strips (I looked for additional P waves indicating higher grades of AV block but I did not see it in the available rhythm strip). Perhaps there is also higher grades of AV block. As such, I am not sure if the syncope was due to VT or AV block. With the low EF, I would favor a VVED (dual chamber ICD).

As to cardiac resynchronization therapy CRT, we are told that he is NYHA functional class II. Further, patients with RBBB tend not to respond to CRT. Hemodynamic may be assisted by an optimal AV delay given his intermittent first degree AV block. One could also titrate the paced or sensed AV delay to result in fusion and with his RBBB, normalize the QRS thus achieving functional CRT with a single ventricular lead. This might be something to be evaluated at the time of device implant with non-invasive echo-doppler studies or invasive studies.

This is a young man and one might also want to consider a revascularization procedure if there is viable myocardium (PCI with stent or CABG).

Paul A. Levine M.D. FHRS, FACC

E-mail: paul91321@gmail.com

25876 The Old Road, # 323, Stevenson Ranch, Ca 91381. USA.

El presente caso muestra una serie de problemas. Primero la manifestación de síncope con documentada TV monomorfa paroxística (no sostenida) en el Holter y baja fracción de eyección, es posible que se deva a TV sostenida.

Hay también un BRD con eje eléctrico del QRS marcadamente desviado a la derecha (bloqueo fascicular postero-inferior izquierdo). Asociado a bloqueo AV de primer grado intermitente y a significativa bradicardia sinusal intermitente en una de las tiras de ritmo (busqué ondas P adicionales que indican los grados más altos de bloqueo AV, pero yo no las veo en la tira de ritmo que está disponible). Tal vez haya también más altos grados de bloqueo AV. Como tal, no tengo certeza si el síncope se debe a VT o a bloqueo AV. Con la baja fracción de eyección, yo estaría a favor de implantar una VVED (DAI bicameral).

En cuanto a la Terapia de Resincronización Cardíaca (CRT), no es oportuna porque se refiere que está en grupo funcional clase II de la NYHA y además los bloqueos de rama derecha tienden a no responder a la CRT.

Hemodinámicamente puede ser asistido por un retraso AV óptimo dado su intermitente bloqueo AV de primer grado. También se podría valorar la estimulación detectando el retraso AV para dar lugar a la fusión y con su bloqueo de rama derecha, normalizar el ancho del QRS logrando una terapia de resincronización cardíaca funcional con una sola derivación ventricular. Esto es algo que debe ser evaluado con métodos no invasivos como el eco-doppler o con estudios invasivos en el momento del implante del dispositivo

Por ser un hombre joven también se podría considerar un procedimiento de revascularización si existe miocardio viable (ICP con stent o cirugía)

Paul A. Levine M.D.

Pregunto: ¿ Este paciente tiene aneurisma de punta ventricular ?

Tentar solucionar apenas el síncope y la fracción. de eyección resincronizando pienso que probablemente no será suficiente. Es muy joven, con un diámetro ventricular enorme motivo por el cual pensaria en la posibilidad de realizar una reconstrucción ventricular eventualmente asociada a revascularización.

Sergio Hauad sergiodh@arnet.com.ar

Question: Does this patient have an apical left ventricular aneurysm?

Tempt to solve just the syncope and the LV ejection fraction resynchronizing I think not enough. He is very young, with a huge LV diameter why would think the possibility of a LV reconstruction eventually associated with revascularization.

Greetings Sergio Hauad sergiodh@arnet.com.ar

Querido Sergio reciente estudio grande que incluyera 1000 pacientes(1) concluyó que la reconstrucción quirúrgica del ventriculo izquierdo asociada a la revascularización beneficia apenas aquellos pacientes con VI menos dilatados y con mejor Fracción de eyección. No ocurre lo mismo con aquellos pacientes con muy baja FE y grandes VIs como es el presente caso

Andrés.

Dear Sergio: recent large trial that included 1000 patients(1) concluded that surgical reconstruction of the LV associated with revascularization benefits only those patients with LV less expanded and with better LVEF. Not so with those patients with low EF and big LVs as is the case.

Oh JK, Velazquez EJ, Menicanti L, et al; on behalf of the STICH Investigators. Influence of baseline left ventricular function on the clinical outcome of surgical ventricular reconstruction in patients with ischaemic cardiomyopathy. Eur Heart J. 2012 May 14.

Conclusions: Subgroup analyses of the STICH trial suggest that patients with less dilated LV and better LVEF may benefit from SVR, while those with larger LV and poorer LVEF may do worse.Clinical Trial Registration #: NCT00023595.

Prezados Colegas

Conforme las últimas Guias para terapia de resincornização cardíaca de 2011 do Comitê da Sociedade Americada de insuficiencia cardiaca a resincronización recomenda-se para pacientes em ritmo sinusal com duração do QRS >150ms com severo deterioro da fração de ejeção ($\leq 35\%$) y classe funcional II o III persistente mesmo com terapia otimizada e na ausência de BCRD (evidencia A) Acho que apenas o CDI estaria indicado pelo fato de apresentar padrão eletrocardiográfico de Bloqueio de Ramo Direito.

HFSA CRT Guideline Update Indications for Cardiac Resynchronization Therapy: 2011 Update From the Heart Failure Society of American Guideline Committee, Journal of Cardiac Failure Vol. 18 No. 2 2012,

“CRT is recommended for patients in sinus rhythm with a widened QRS interval (>150 ms) that is not due to right bundle branch block who have severe LV systolic dysfunction LVEF ($\leq 35\%$) and persistent mild to moderate HF (NYHA functional class IIeIII) despite optimal medical therapy (strength of evidence A)”. Consequently, I think that CDI implant is indicated because his ECG has RBBB pattern.

Adail - Bahia - Brasil

- 1) Sin duda le implantaría un Desfibrilador Automático Implantable (DAI.)
- 2) Este CDI sería al menos bicameral porque está en ritmo sinusal pero con la conducción AV comprometida.
- 3) Solicitaría un Ecocardiograma con doppler tisular para ayudarme a decidir el número y los sitios de estimulación:
 - *Si la contracción en pared lateral es tardía y tengo posibilidad de encontrar tejido sano, le implantaría un resincronizador - ésto también ocurre en pacientes con bloqueo de rama derecha.*
 - *No siendo así, probablemente dejaría un solo electrodo en el tracto de salida del VD ó septo alto, siempre y cuando no tenga problemas con la desfibrilación.*
 - *Si del análisis de todo el Holter concluyó que va a estar estimulado la mayoría del tiempo busco los dos mejores sitios en VI y VD o eventualmente dos sitios del VD donde dejar dos electrodos.*
 - *Finalmente y como siempre la anatomía venosa mandará dónde podemos llegar y las cicatrices dónde logramos estimular y efectivamente capturar y sensor.*

Saludos: AC

Alejandro Cuesta M.D. Ph.D. República Oriental del Uruguay arritmia@yahoo.com

No doubt he would set up an Implantable Cardioverter Defibrillator (ICD.) and additionally at least bicameral because he is in sinus rhythm but with probable severe damage of intraventricular conduction system. Would ask for a tissue Doppler echocardiography to help decide the number and sites of stimulation:

If the contraction of lateral wall is late and we have found the possibility of to found healthy tissue, we would implant a resynchronization device - this also occurs in patients with right bundle branch block.

Not the case, would probably only one electrode in the right ventricular outflow tract or high septum, provided they do not have problems with defibrillation.

If the entire Holter analysis concluded that going to be stimulated most of the time I find the two best places in LV and RV or possibly two sites of the RV where leave two electrodes. Finally, and as always send the venous anatomy can we go and the scars where we encourage and effectively capture and sensing.

Estimado Dr Abud

Creo que Ud presenta un caso muy interesante y frecuente de ver en la practica diaria, sin embargo, no deja de ser un dilema.

El resumen del paciente, si lo entendi bien, es una cardiopatia dilatada isquemica con muy mala FEY, pero que NO presenta el clasic BCRI sino que presenta un trastorno de conduccion de la rama derecha, bloqueo AV de 1er grado y eje a la derecha: entonces su duda es: CDI solo (y programo FC de MP minima para evitar MP desde el VD y deteriorar mas la FVI) o CDI solamente como prevencion primaria? Su duda es muy buena, porque tanto el MADIT CRT como el RAFT canadiense, le demuestran que si Ud no tiene un verdadero BCRI, los pacientes NO responden a la terapia de resincronizacion. Pero su caso es diferente: Porque?

Porque en el Holter que nos envia hay momentos de BAVC y de bradicardia sinusal que seguramente, por el uso de BB (y si no los tiene, deberia tenerlos) Ud forzara el marcapaseo del CDI. Por lo tanto, aqui la disyuntiva es menor, ya que como el MP del VD generara un QRS mayor de 180 ms (aun desde el tracto, dado el trastorno de conduccion previo ensanchara el QRS), entonces la indicacion de CRT es mandatoria. No me caben dudas, que cualquier forma de MO que no sea resincronizada agravara la ICC de su paciente. En conclusion: CDI + CRT.

Le repito, su caso se ha vuelto una disyuntiva muy frecuente, y si no hay bloqueos tan avanzados como en su caso, si el paciente NO tiene BCRI y no predecimos que el paciente usara el MP del CDI, NO usamos CRT, ya que los pacientes NO responden satisfactoriamente.

Le mando un fuerte abrazo.

AB

The surface ECG shows a RBBB and Left posterior inferior pattern. The Holter strips show episodes of non sustained VT as well as sinus bradycardia and escape capture bigeminy (probably during sleep).He also has sinus tachycardia.

He needs an EP study to evaluate whether the syncope is related to AV block vs VT. He will definitely be a candidate for an AICD because of the very poor ejection fraction with optimal therapy.Since he has a RBBB pattern insertion of a Bi Ventricular pacing for CRT is not clearly beneficial.

He would likely benefit more with a Left Ventricular assist device and in view of his age consider heart transplant.

Melvin M Scheinman

Department of Cardiac Electrophysiology, University of California San Francisco, San Francisco, California, USA. scheinman@medicine.ucsf.edu

Professor of Medicine Address: UCSF Electrophysiology Service 500 Parnassus Avenue San Francisco, CA 94143-1354 Telephone/FAX/E-mail: Phone: (415) 476-5706 Fax: (415) 476-6260

El ECG de superficie muestra BCRD y patrón de bloqueo fascicular póstero-inferior.

La tira del Holter muestra episodios de TV no sostenida así como bradicardia sinusal y el fenómeno de bigeminismo escape-captura probablemente durante el sueño. El también tiene taquicardia sinusal.

El paciente necesita un estudio electrofisiológico para evaluar la causa del síncope si esta relacionada al bloqueo AV o a la TV. El es un candidato definitivamente a AICD porque tiene muy baja fracción de eyección con la terapia farmacológica optimizada.

Por tener un patrón de BRD el implante de un marcapaso biventricular para resincronización no me parece que sea claramente benéfico.

El podría beneficiarse más con un aparato provisorio de asistencia ventricular teniendo como objetivo por su juventud un trasplante cardíaco.

El ECG es de muy mala calidad el ritmo sinusal con agrandamiento biauricular y trastornos intra e interauriculares. PR muy prolongado, pero el ritmo ventricular parece conducido.

El primer vector se orienta hacia arriba atrás y a la izquierda con rotación antihoraria en el plano frontal y creo que también en el horizontal. para justificar las melladuras iniciales que se ven en I y aVL y en el resto del ECG también Después no hay dudas que es horaria en los dos planos. Que es lo que se activa? Dos posibilidades:

- Pared lateral del VI
- O septum de derecha a izquierda, para esta opción no me gusta la rotación, sólo que el septum este hipertrófico y no lo esta porque esta necrotico. La activación de esta zona está demorada y lo hace a traves del fasciculo anterior .El retraso en la llegada del impulso al VI puede ser debida a la gran necrosis, lo único que se ve es esa zona lateral. Tiene un BRD verdadero, más un gran agrandamiento del ventriculo derecho e hipertensión pulmonar. Eso explica que gran parte del bucle este en el cuadrante anterior

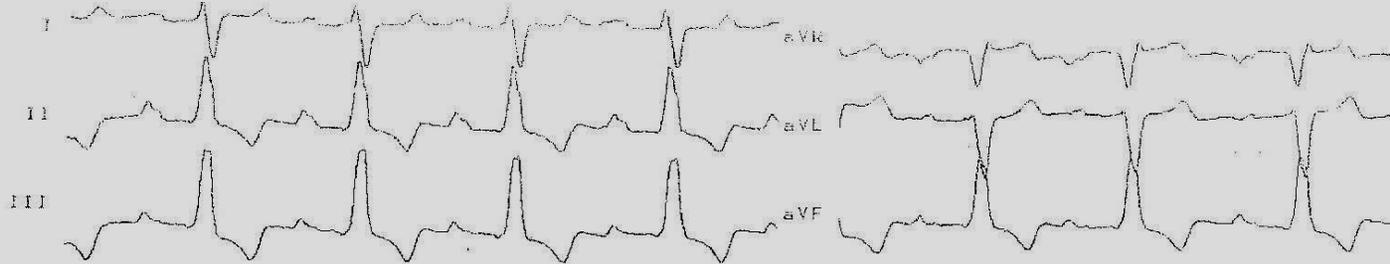
Las fuerzas en el plano horizontal tienen que ser concordantes con las del frontal; **DI MARCA UNA GRAN NEGATIVIDAD** y la mayor parte del bucle se va a la derecha ya que la máxima positividad la vemos en DIII. El ECG en las precordiales izquierdas es por lo tanto posicional y no real. Para poder realizar el diagnóstico de bloqueo septal tengo que ver las fuerzas demoradas del septum y como esta todo necrosado si lo tiene no puedo realizar el diagnóstico. De igual manera no veo que sea un HBP. Conclusión: miocardio destruido secundario a una gran necrosis con severos trastornos de conducción, .Como ves tenemos tenemos algunos coincidencias pero no veo agrandamiento de VI? sino una gran necrosis y casi todas las fuerzas vectoriales son del VD-Cariños

Rafael Acunzo M.D. Argnetina

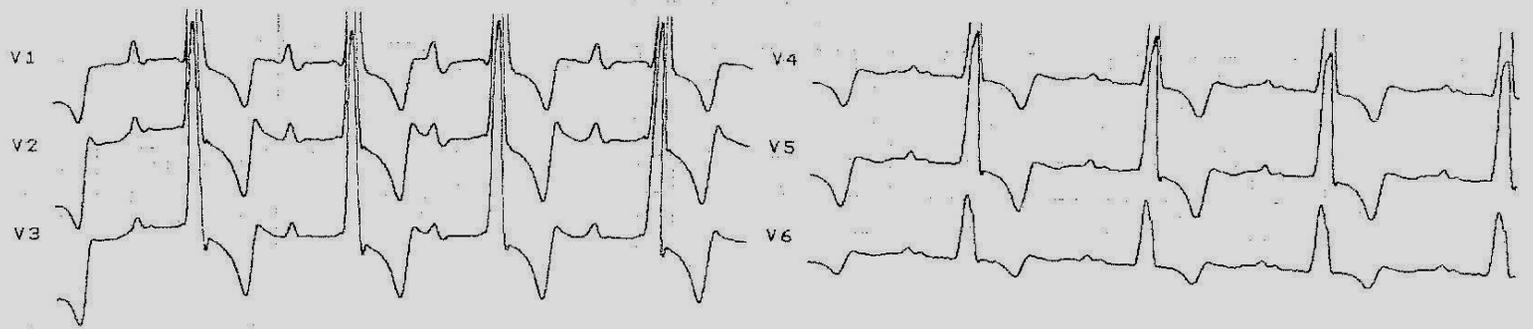
Final Comments

By Andrés Ricardo Pérez-Riera M.D. Ph.D.

Medical Medical Inc.
Patient ID: Auto 0001
Date: 06/07/2013
Time: 11:56:38
Friday
Speed: 25mm/s
Sens: 10mm/mV
Filter: Noise + Mains
Baseline filter: On

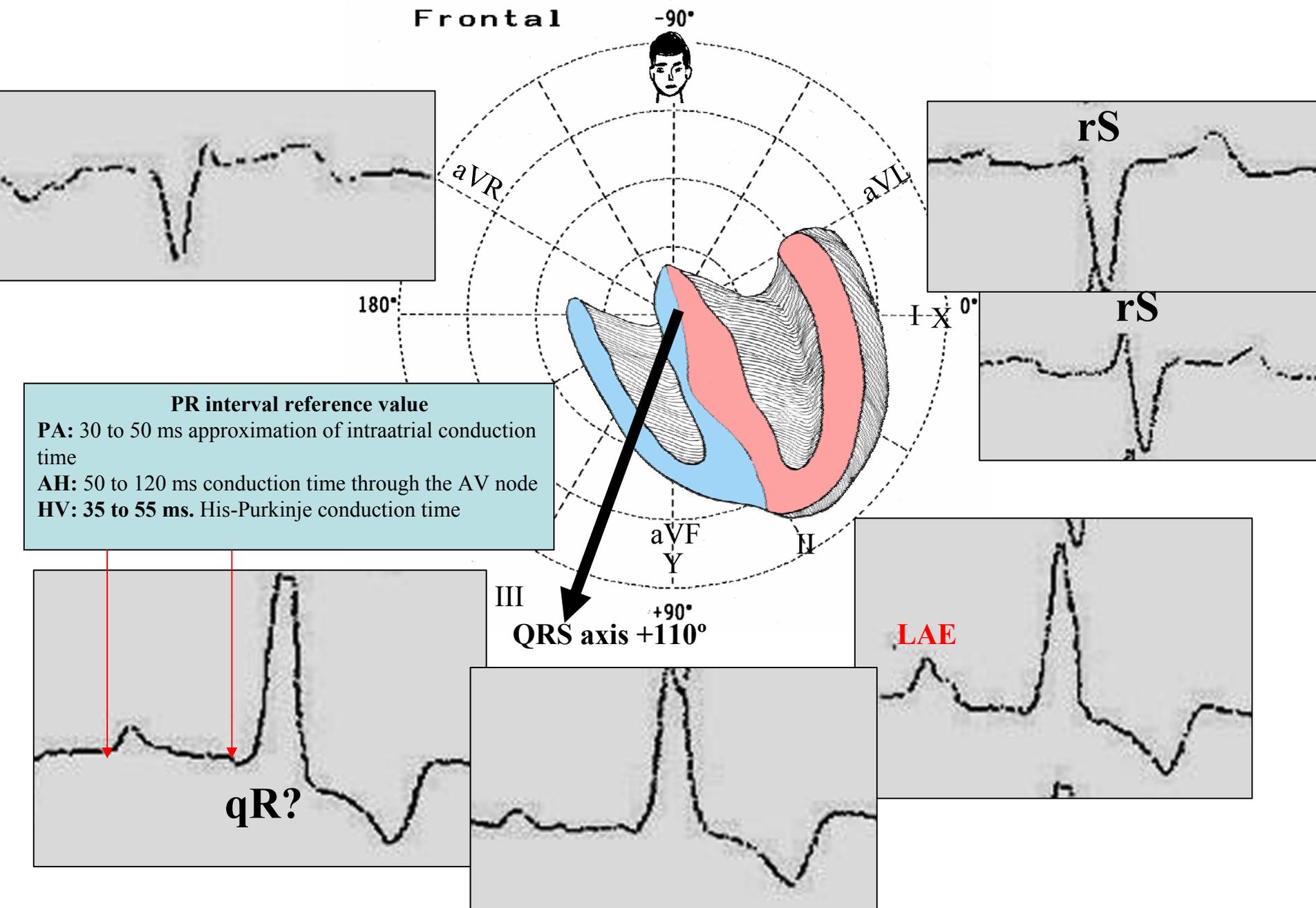


HeartMirror 3-IK0
Version 1.21B

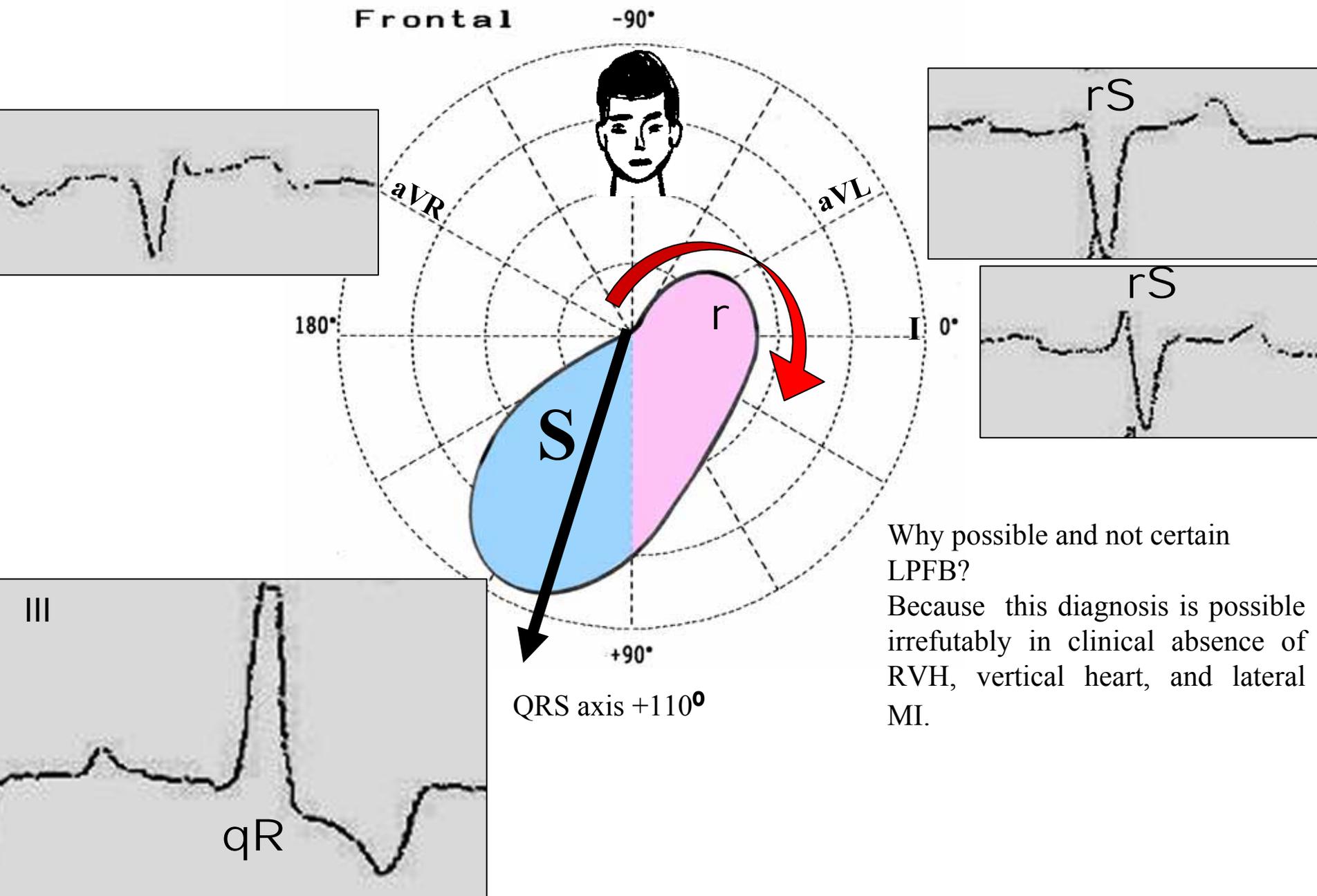


6.7.13.11.56.38

Is this frontal frontal plane QRS pattern has a Left Posterior Fascicular Block pattern associated with RBBB and First-degree AV block Possible trifascicular block ?



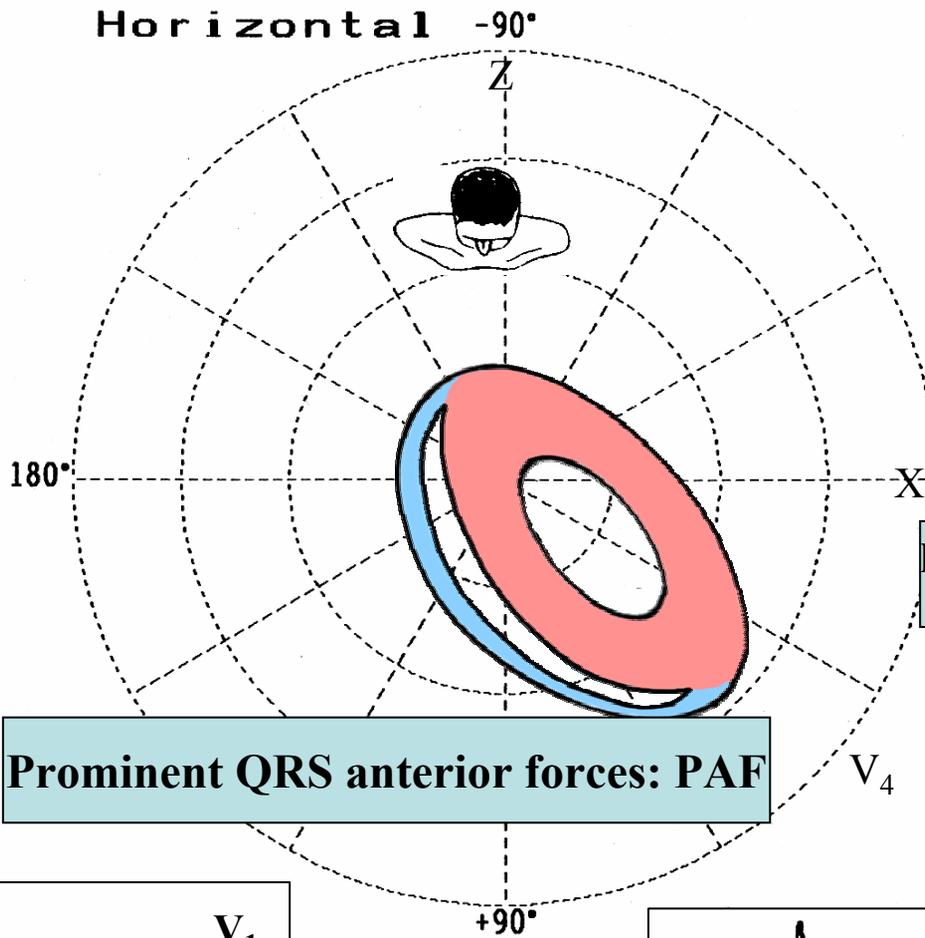
Possible Left Posterior Fascicular Block



Why possible and not certain LPFB?

Because this diagnosis is possible irrefutably in clinical absence of RVH, vertical heart, and lateral MI.

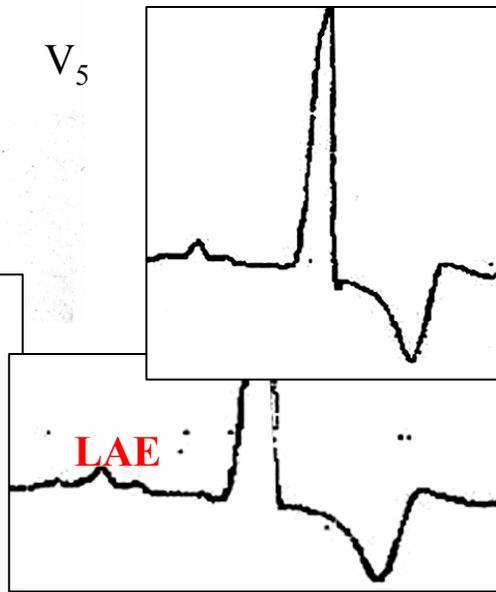
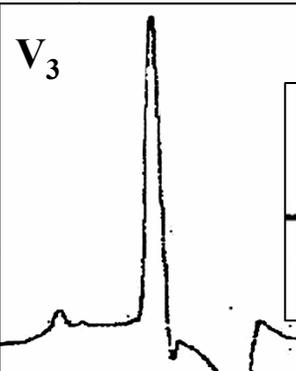
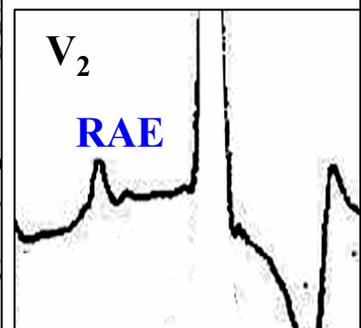
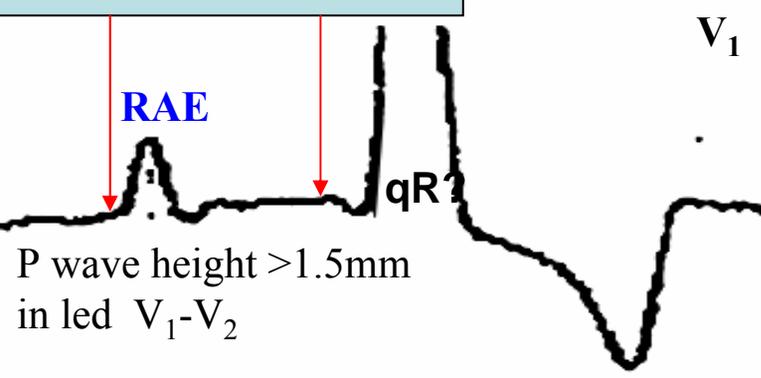
Is this precordial QRS pattern a precordial Masquerading Bundle Branch Block?



Prominent QRS anterior forces: PAF

Left Bundle Branch Block
In the left-side leads

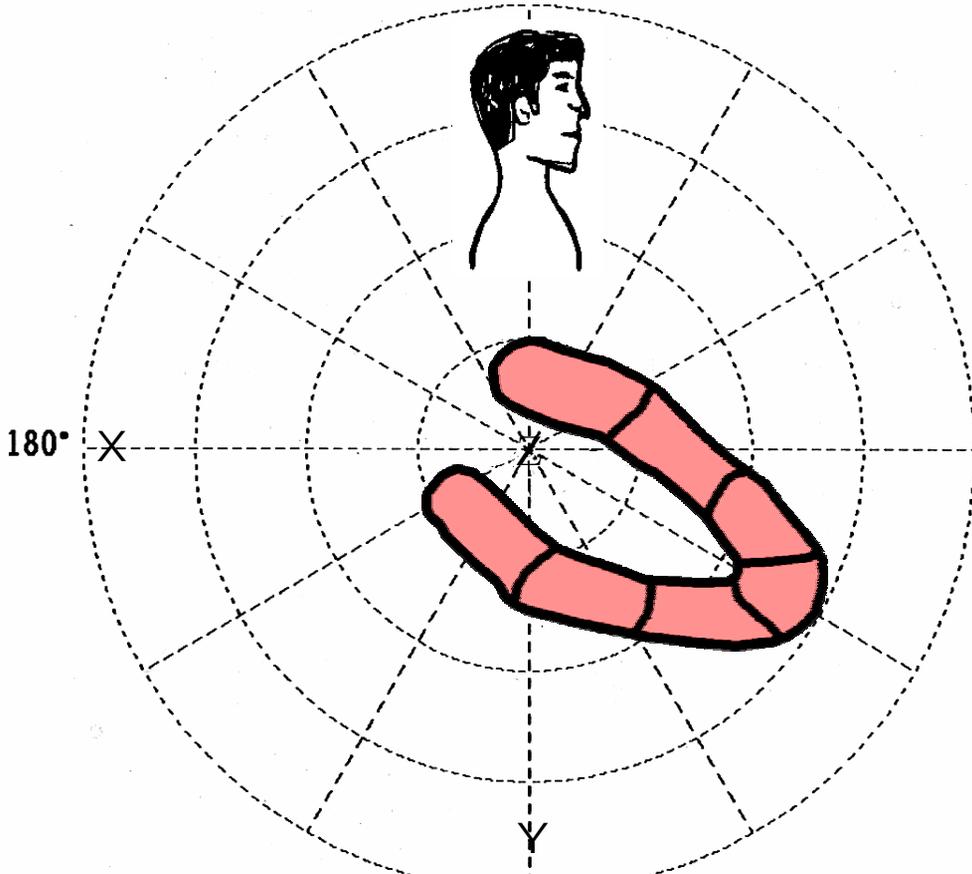
First-degree AV block



Right Bundle Branch pattern in the right precordial leads?

Sagittal

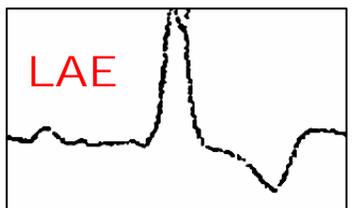
-90°



0° V₂

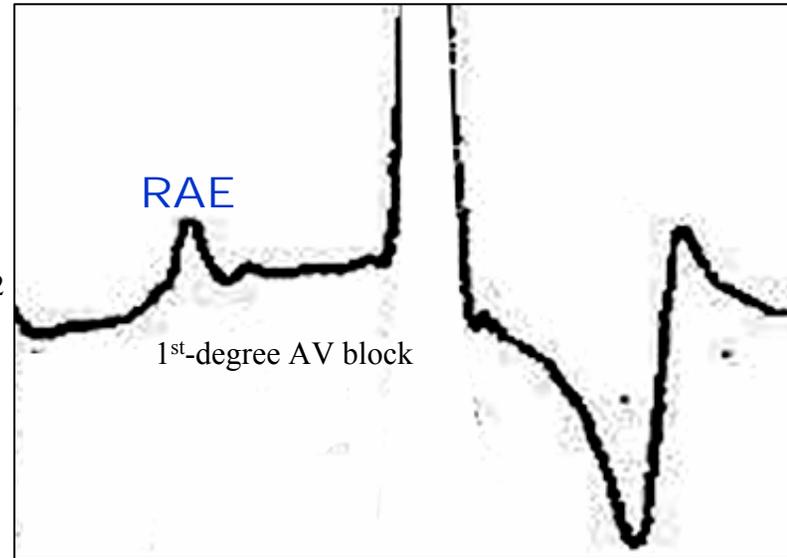
+90°

aVF



Prominent Anterior Forces

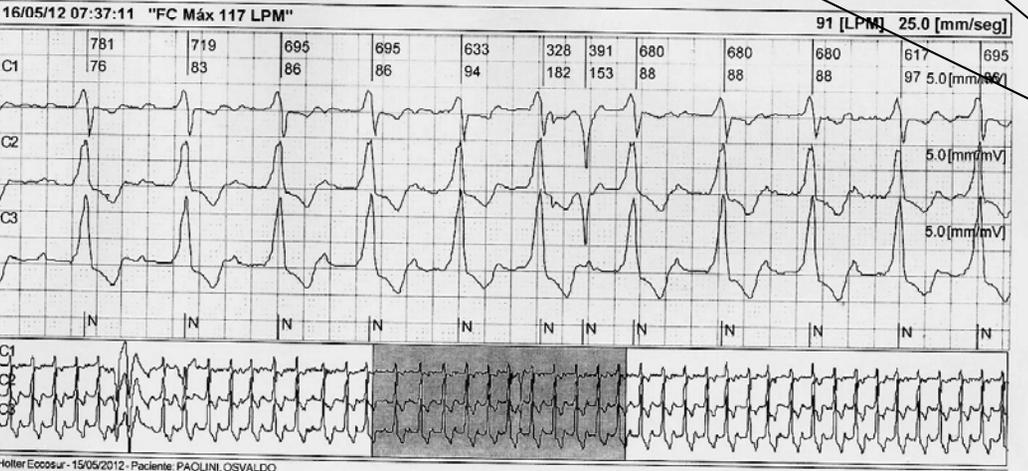
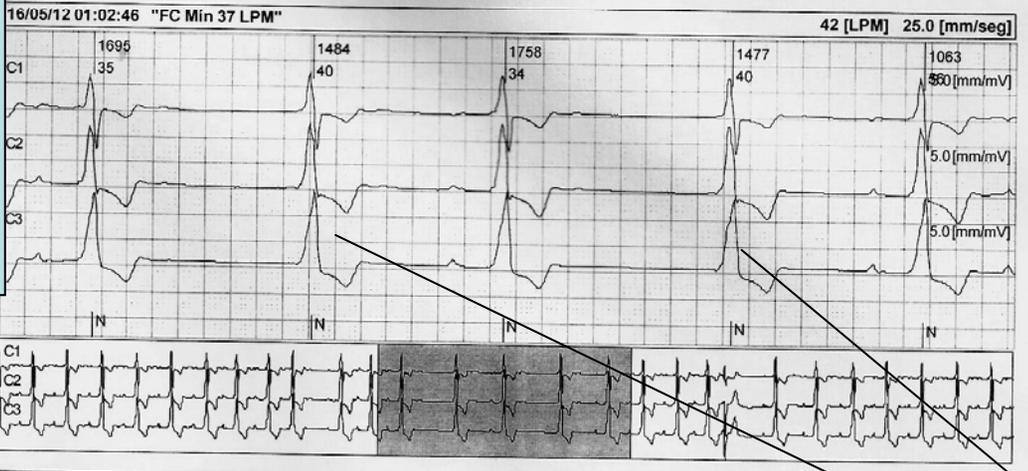
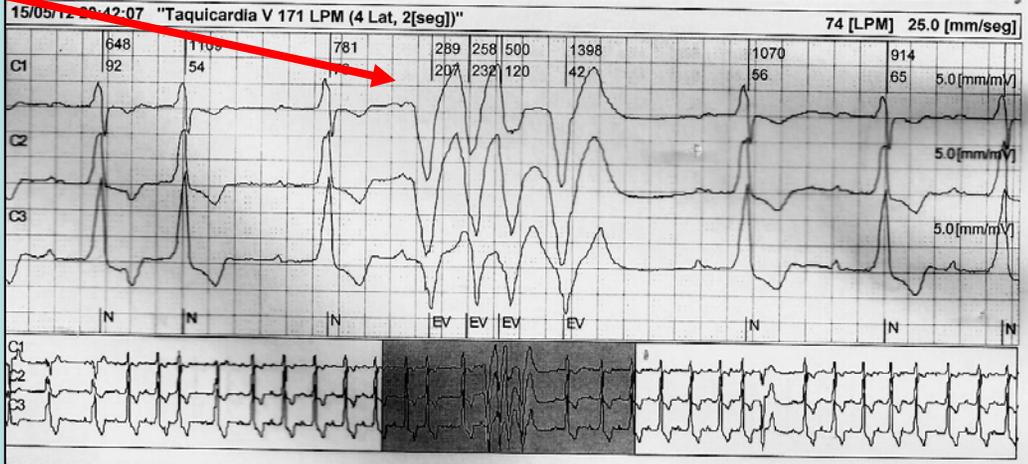
PAF



PAF possible causes

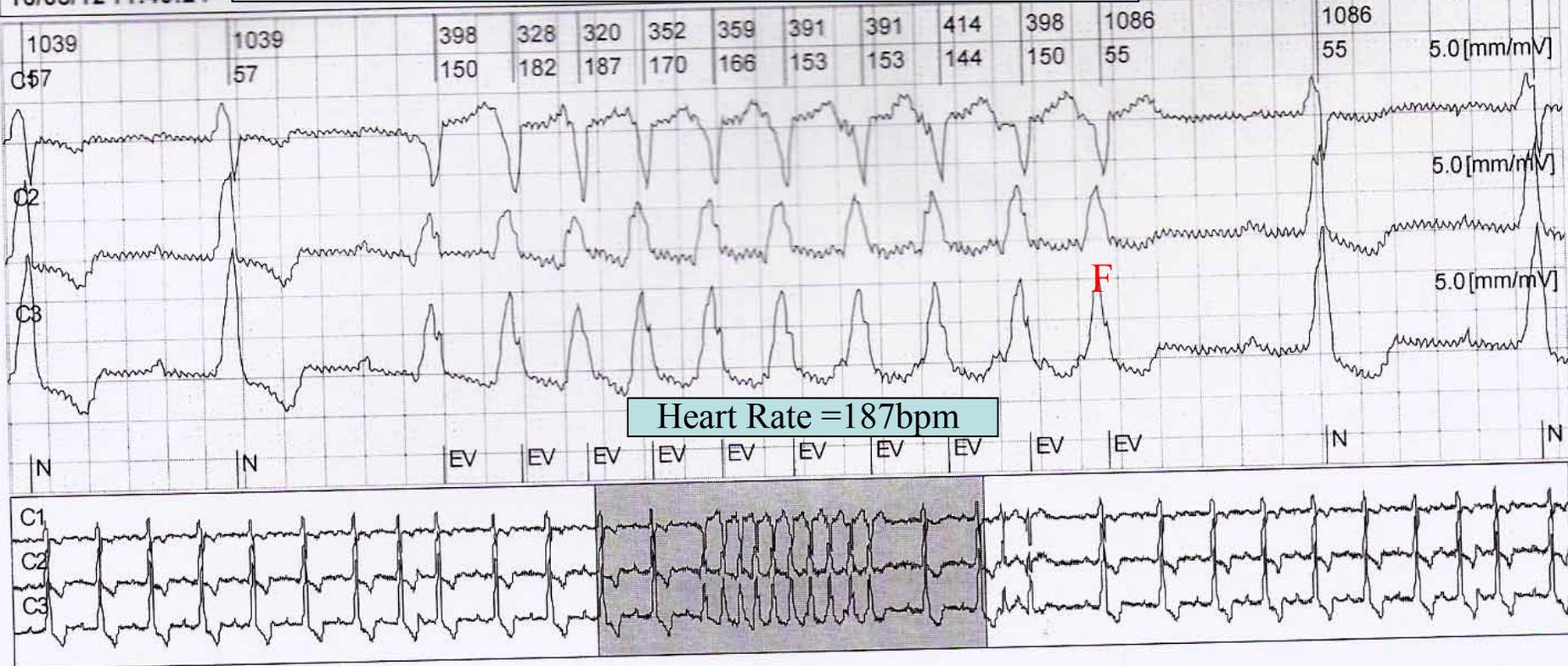
- Right Bundle Branch Block?
- RBBB+ Right Ventricular Hypertrophy?
- Left Septal Fascicular Block (LSFB)?
- RBBB+LSFB?
- RVH?

No sustained VT, NS-VT, VT runs, or transitory: more than three consecutive ventricular depolarizations with a HR above 100 bpm and with a duration lower than 30 seconds. VT terminates spontaneously within 30s. NS-VT is defined as a run of VT of less than 30 seconds duration



Severe Sinus bradycardia with "escape-capture bigeminy" (during sleep). The junctional rate is faster than the sinus rate.

Junctional escape beats



Sustained VT (S-VT): is defined as continuous VT lasting for >30s or that requires and intervention for termination (such cardioversion) 70% present prior infarction: or with hemodynamic involvement. Duration greater than 30 seconds or when they cause symptoms that made their interruption mandatory. While nonsustained ventricular tachycardia is a frequently observed dysrhythmia, sustained, monomorphic ventricular tachycardia is uncommon in the emergency department (ED) setting due to aggressive treatment of myocardial ischemia. When sustained VT causes signs or symptoms of diminished perfusion, emergent treatment is necessary.

F: Ventricular Fusion beat: A fusion beat that occurs when the ventricles are activated partly by the descending sinus or atrioventricular nodal impulse and partly by an ectopic ventricular impulse

I) Standard masquerading type of bundle branch block

The so-called “standard masquerading” type of bundle branch block is caused by the simultaneous presence of a high degree left anterior fascicular block (LAFB) often accompanied with severe left ventricular enlargement/hypertrophy and/or fibrotic block in the anterolateral wall of the left ventricle. The sine qua non for this condition is a high degree of LAFB. These conditions tend to reorient the terminal electrical forces of the QRS complex toward the left and upwards in such a way that the characteristic slurred S wave in lead I becomes smaller or even disappears. In many cases of standard masquerading right bundle branch block, a small Q wave in lead I is present due to the initial forces of the left anterior fascicular block, which are oriented rightwards and inferiorly. However, in some cases, the Q wave in lead I also vanishes and the mimicking of a left bundle branch block becomes perfect. This is commonly associated with an inferior myocardial infarction or severe inferior fibrosis consequence of cardiomyopathies.

The standard masquerading bundle branch block the QRS pattern in limb leads resembles that of LBBB although the precordial ECG remain diagnosis of RBBB.

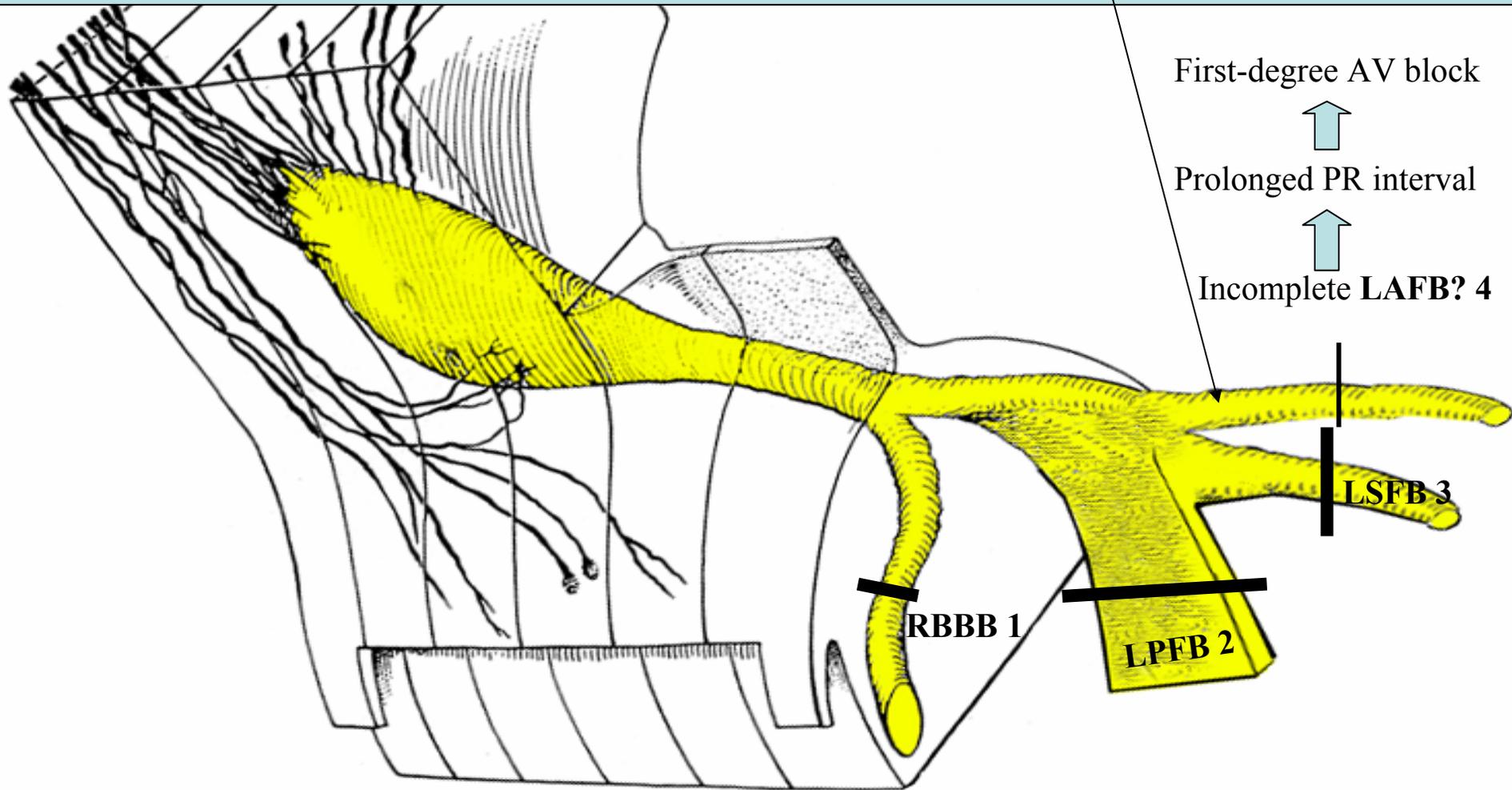
II) Precordial Masquerading Bundle Branch Block

Some ventricular conduction defects show **the pattern of RBBB in the right precordial leads and LBBB in the left side leads** such as the present case. This pattern has been termed **Precordial Masquerading Bundle Branch Block**.

Precordial Masquerading Bundle Branch Block results from RBBB associated with severe left ventricular enlargement, a localized block in the anterolateral wall of the left ventricle (often due to myocardial infarction) and usually left anterior fascicular block. In the present case we have left posterior fascicular block(I think that there are not another case described in the literature??)

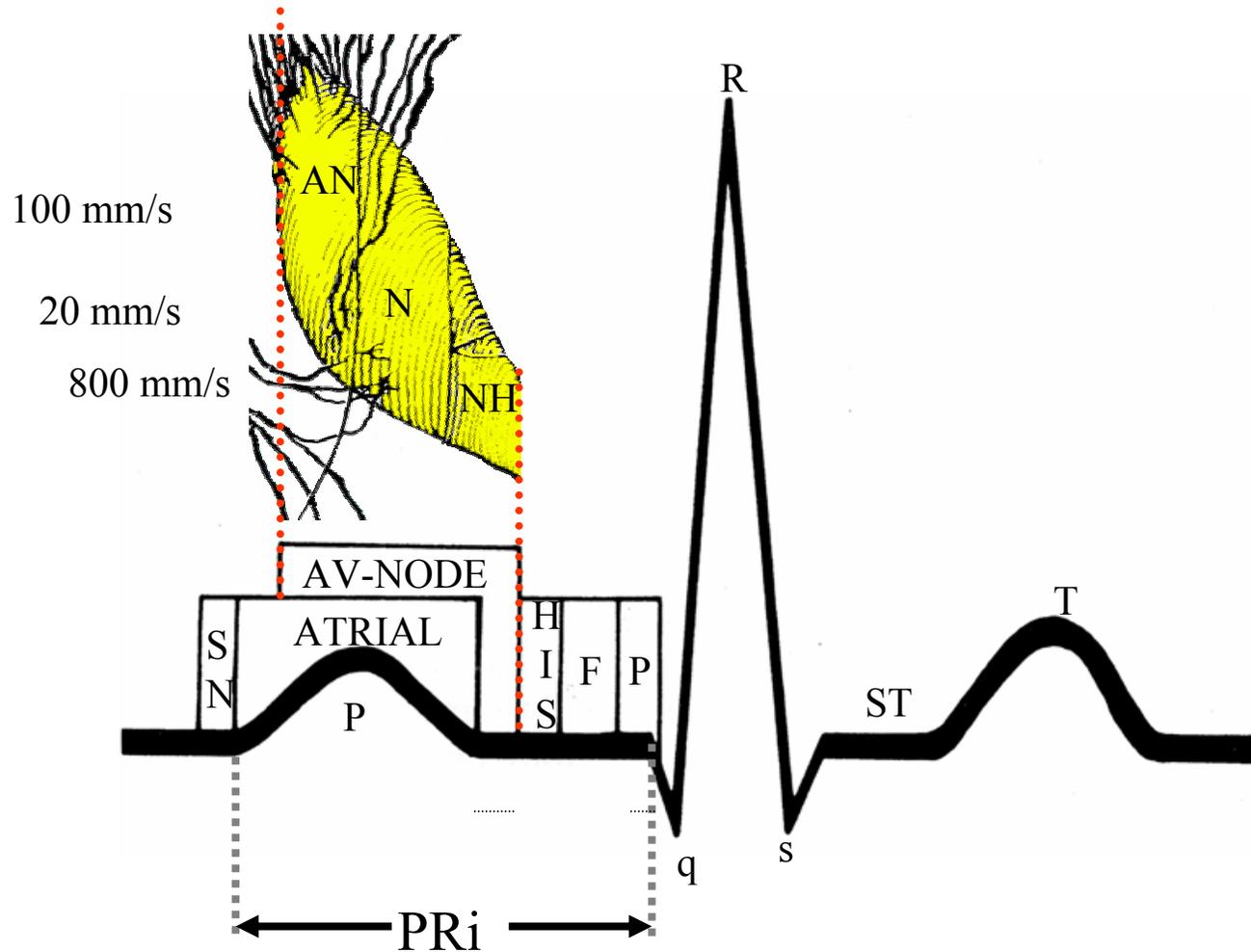
First-degree
AV block

1. **Supra-hisian or pre-Hisian.**: may extend PA and/or AH intervals: conduction slowing in the atria (PA) and/or AV node (AH).
2. **Hisian and infra-Hisian.**
Hisian: His bundle block.
Infra-hisian, fascicular block: branches and divisions.
3. **Mixed:** they affect the PA, AH and HV intervals.



1 + 2 + 3 + 4 Possible tetrafascicular intraventricular Block

THE PR INTERVAL COMPONENTS



Representation of the PR interval of the onset of P wave at the onset of the QRS complex. During the PR interval, the stimulus runs through the SA node (SN), the atria, the AV node, the His bundle, Fascicles(F), and Purkinje arborizations (P). In the superior part of the figure the three areas of the AV node are represented: AN region (conduction velocity: 100 mm/s), N or central region (conduction velocity: 20 mm/s) and the NH region (conduction velocity: 800 mm/s).

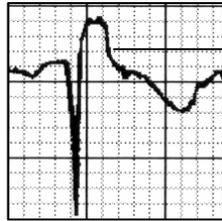
Final ECG diagnosis

1. **Left Atrial Enlargement (LAE):** prolonged P wave (P-duration >110ms), notches and bimodal in P wave with distance between the apexes of ≥ 40 ms
2. **Right Atrial Enlargement (RAE):** P wave height >1.5mm in led V_1 - V_2 . The criteria has 100% specificity preserved Presence of Sodi Pallares sign: qR, V_1 and V_2
3. **Biatrial enlargement: LAE+ RAE**
4. **First degree AV block:** Prolonged PR interval possible manifestation of minor degree of LAFB. It is necessary electrophysiological study for confirmation dromotropic disturbance level. Possible Infra-hisian, fascicular or divisional: 20%. associated to wide QRS complex.

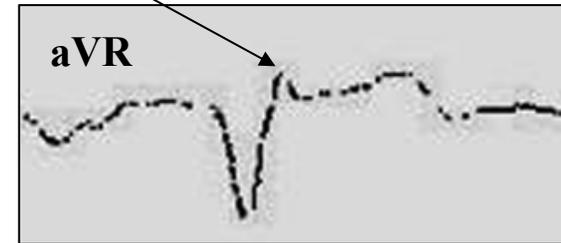
	SUPRA-HISIAN OR PRE-HISIAN	HISIAN AND INFRA-HISIAN
LOCATION: PERCENTAGE:	AV node 75%	His bundle or divisions. 5% and 20%.
QRS DURATION:	Up to 100 ms.	≥ 120 ms : bundle branch block pattern
HISIAN ELECTROGRAM	Prolongation of AH or PA intervals	Infra-hisian: splitting of H deflection with progressive distancing: H1-H2
AUTONOMIC INFLUENCE PROLONGED WENCKEBACH CYCLES:	Important. Frequent.	Less. Rare.
PROGNOSIS:	Better.	Worse: it may evolve abruptly into advanced block. Risk of SCD.

5. **Possible Complete LPFB pattern:** rS pattern in I and aVL, qR en III, QRS axis +110°. This diagnosis is not of certain because it is necessary absence of RVH/RVE to be clinical-electrocardiographic.
6. **Possible Left Septal Fascicular Block:** Prominent QRS Anterior Forces, (PAF), absence of initial q wave in left leads (V5-V6) consequence of absence of first I_{AM} septal vector,.
7. **Possible ATYPICAL Complete RBBB?** Why atypical? Because there are not final S waves in left precordial leads (*Precordial Masquerading Bundle Branch Block*), and final r wave in aVR is not broad.

Typical aVR
In CRBBB



→ Broad final R wave



8. **Possible tetrafascicular intraventricular Block:** This is a new proposal nomenclature: LPFB, minor degree of LAFB manifested by first degree AV block, LSFB and CRBBB.
9. **Left Ventricular Enlargement/Hypertrophy (LVE/LVH)** dilated LV
10. **Right Ventricular Enlargement/Hypertrophy (LVE/LVH)** dilated RV
11. **Biventricular Enlargement?**
12. **Prominent QRS Anterior Forces (PAF):** Cause? RBBB? LSFB? Both dromotropic disorders? RVH?; RVH + CRBBB?
13. **New POSSIBLE variant of Precordial Masquerading Bundle Branch Block: RBBB pattern in the right precordial leads and LBBB in the left side leads in absence of LAFB and presence of LPFB.**
14. **NS-VT and S-VT**
15. **Severe bradychardia and tachycardia in the same Holter recorder**
16. **Possible Sick Sinus Syndrome?** Why? because bradycardia-tachycardia syndrome is a variant of sick sinus syndrome in which slow arrhythmias and fast arrhythmias alternate. Coronary artery disease, high blood pressure, and aortic and mitral valve diseases may be associated with sick sinus syndrome, although this association may only be incidental. The mechanism is related to delayed escape
17. **Severe sinus bradycardia with “escape-capture bigeminy”(during sleep).**
18. **Junctional escape with rate faster than the sinus rate.**

Management

Cardiac resynchronization therapy (CRT) through biventricular pacing is an effective treatment for NYHA class III or IV congestive heart failure (CHF) improved quality of life and functional mitral regurgitation, functional status, and exercise capacity in patients with moderate to severe HF, a wide QRS interval (≥ 130 ms) with left bundle branch block (LBBB), left ventricular ejection fraction (LVEF) $\leq 35\%$ and life-threatening arrhythmias. These improvements occurred in the context of underlying appropriate medical management without proarrhythmia or compromised ICD function.(1) A critical analysis of the data would suggest the true non-responder rate can be estimated as perhaps 40-50%. The data indicate that on a population basis non-response is multi-factorial and the extent of mechanical dyssynchrony, left ventricular pacing site and cause of CHF are likely to be important. Ongoing research is exploring the utility of various techniques for quantifying mechanical dyssynchrony and the potential benefits of targeted LV lead placement and post-implant optimization. Patients with an improvement of ≥ 1 NYHA class from baseline to the 6-month follow-up are considered responders and those who had no change or worse NYHA class or died are classified as nonresponders. Responders are subdivided into early (within 1-3 months) and late (6 months). Based on improvement of ≥ 1 NYHA class, less than 65% of patients enrolled in the, REVERSE, MADIT-CRT, and RAFT, MUSTIC, CONAK-CD, MIRACLE and MIRACLE-ICD trials(2) responded to CRT, with just more than half responding within the first month. Several factors predicted CRT response and timing, but given their modest predictive accuracy, comparable for the several studies, additional methods for selecting candidates most likely to benefit from CRT are needed.

1. **Young JB, Abraham WT, Smith AL, et al; Multicenter InSync ICD Randomized Clinical Evaluation (MIRACLE ICD) Trial Investigators. Combined cardiac resynchronization and implantable cardioversion defibrillation in advanced chronic heart failure: the MIRACLE ICD Trial. JAMA. 2003 May 28; 289:2685-2694.**
2. **Linde C, Ellenbogen K, McAlister FA. Cardiac resynchronization therapy (CRT): Clinical trials, guidelines, and target populations. Heart Rhythm. 2012 Apr 20. [Epub ahead of print]**

In the present case we have not ideal conditions:

- 1) Since he has a RBBB pattern implantation of a biventricular pacing for CRT is not clearly beneficial. Patients "nonresponders" with deterioration are more likely to be men, have a non-left bundle branch block morphology(1)
- 2) The patient has a giant LV distolic diameter (LV diastolic diameter 82.3mm / LV75.5 systolic diameter). Additional, free wall and septum are fine: 6.8mm 11mm. (By Echo and confirmed by MRI). Finally, LVEF is very low 17%. Candidates for cardiac transplantation demonstrates LVEF of less than 20 to 25%. Attempts are made to stabilize the cardiac condition while the evaluation process is undertaken. He would likely benefit more with a Left ventricular assistance device bridging, (LVAD), left ventricular mechanical assistance or mechanical circulatory support. These devices are good therapeutic option that can be used as a bridge to transplantation or recovery. and in view of his relatively young age consider heart transplant
- 3) Although both heart transplantation and LVAD therapy have enjoyed clinical success in the treatment of patients with end-stage heart disease, newer LVADs currently undergoing testing are likely to have a tremendous impact on the management of these patients. Smaller, more durable devices with improved safety profiles will allow for longer duration of therapy and make biventricular support more feasible, obviating the need for the total artificial heart.(1) The general indications for cardiac transplantation include deteriorating cardiac function and a prognosis of less than 1 year to live. Specific indications include the following: Dilated cardiomyopathy, ischemic cardiomyopathy, congenital heart disease for which no conventional therapy exists or for which conventional therapy has failed, LVEF less than 20%, intractable angina or malignant cardiac arrhythmias for which conventional therapy has been exhausted, pulmonary vascular resistance of less than 2 Wood units, age younger than 65 years. ability to comply with medical follow-up care.(2)

1. Rickard J, Jackson G, Spragg DD, et al. QRS prolongation induced by cardiac resynchronization therapy correlates with deterioration in left ventricular function. *Heart Rhythm*. 2012 May 11. [Epub ahead of print]
2. Milla F, Pinney SP, Anyanwu AC. Indications for heart transplantation in current era of left ventricular assist devices. *Mt Sinai J Med*. 2012 May;79:305-316.