Atypical LBBB case report

Dear colleagues,

This ECG belongs to an old woman 78 yo. congestive heart failure, functional class II-III with appropriated medication and one episode of near syncope last week associated with retro-body sternum pain.

Questions

- Which is the ECG diagnosis?
- Which the following approach?

All the best for all,

Andrés.

Portugês

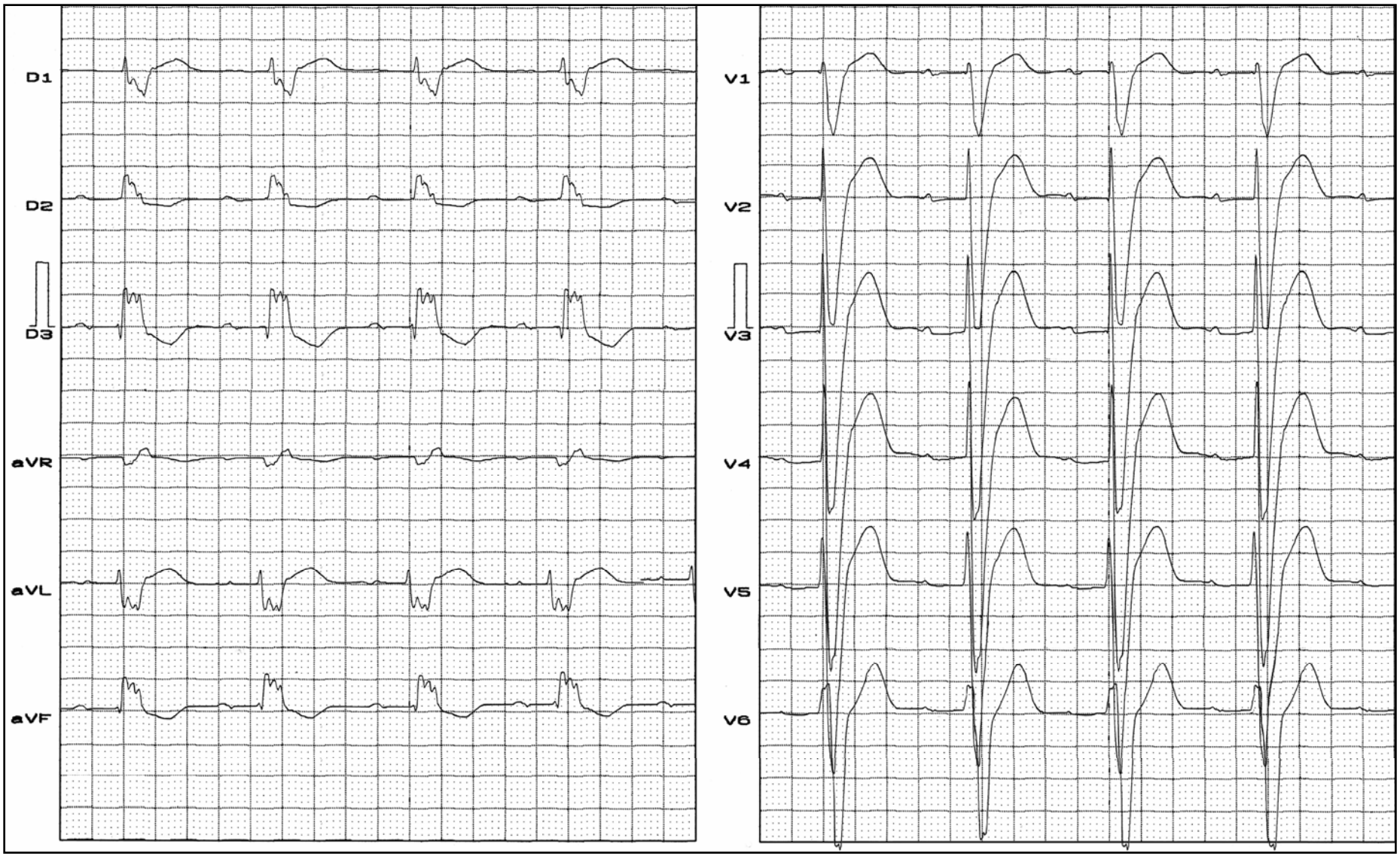
Prezados colegas,

Este ECG pertence a uma senhora idosa de 78 anos em ICC classe funcional II-III com medicação adequada e um episódio de pré-sincope a uma semana associado a dor no corpo do esterno.

- Qual é o diagnóstico ECG?
- Qual a conduta a seguir?

Saudações para todos,

Andrés.


D1
D2
D3
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

ANSWERS/RESPUESTAS/RESPOSTAS

- **Friends, I think this is bifascular block, which possibly has become complete. I would suggest hisiogram, and if beyond 55 msec, a pacemaker should be considered, and if possible consider resynchronization.**
- **Warm regards,**

- **Amigos creo se trata de un bloqueo bifascicular que posiblemente se haya hecho completo. Sugeriría hisiograma y si más allá de 55 ms valorar marcapasos y si posible considerar resincronización**

  Abrazos

- **Rolando Rogés**

- **A hisiogram? What fo', my man?.Or do you think it will be normal?**
- **And if the patient has a normal ventricular function, resynchronization, what fo', my man?. Or do you perform hisiograms to determine resynchronization?**
- **----------**
- **Y el hisiograma pa' que, chico?. O cree Ud que va a ser normal?**
- **Y si el paciente tiene funcion ventricular normal, pa' que resincronizar, chico?**
- **O Ud realiza hisiogramas para determinar resincronizacion?**
- **AB**

Prof. Adrian, and the other colleagues of the Forum, You are right, I think it is more difficult to find normal function, according to Dr. Perez Riera, ICC II-III, 78 years old.

- **Warm regards from Bahia. I would be careful with digitalis.**
- **Dr. Adail - Bahia – Brazil**
- ----------
- Prof. Adrian e demais colegas do Fórum:
- Tens razão, mas parece difícil achar função normal, segundo Dr. Perez Riera CHF II-III, 78 anos.
- Caloroso abraço baiano. Eu CUIDARIA DO DIGITAL
- Dr. Adail - Bahia – Brasil
- ----------
- Prof Adrian y colegas del Foro:
- Tienes razón pero me parece difícil encontrar uma función ventricular normal cuando el Dr Pérez Riera há comentado que se encuentra em clase funcional II a III apesar de estar convenientemente medicado.
- Yo cuidaria de la digital.
- Abrazo
- Dr. Adail - Bahia – Brasil.

I see the following:
*** Complete left bundle branch block, associated to right ventricular enlargement with extreme shift of AQRS to the right (between +90º and +135º), paradoxical CLBBB or type IV of Lepeschkin.**
*** 1st degree AV block**
*** Left atrial enlargement, by slow return to the isodiphasic line, greater than 40 msec in the P wave in lead V1.**
Causes for SAQRS shift to the right in CLBBB

CLBBB associated to RV enlargement
CLBBB associated to lateral infarction
Fascicular CLBBB with degree of block in the posterior fascicle,

greater than that presented in the left anterior fascicle block.

CLBBB with accidental change of the limb leads.

There is also depression in inferior side, and r waves with increased voltage in V2-V3 and V4 are funny.
Thank you, Professor Perez Riera for everything. Thank you for recommending Surawicz' book; it's very interesting.
With the permission of Edgardo, I give you an Italian page on arrhythmias:
http://www.cardiolink.it/ecg-bachecaecg.asp (or you may search it as ecg-online of Doctor Giuseppe Oreto).
Regards for everyone,

Observo lo siguiente:
· Bloqueo Completo de Rama izquierda asociado a Sobrecarga Ventricular Derecha con extremo desvio del ÂQRS a derecha (entre +90º y +135º), o BCRI paradójico o tipo IV de Lepeschkin. BAV de 1º grado.
· Sobrecarga Auricular Izquierda, por retorno lento a la línea isodifásica mayor a 40 milisegundos en la onda P de la derivación V1. Causas de desvío del SÂQRS para la derecha en el BCRI
· BCRI asociado a SVD
· BCRI asociado a Infarto Lateral
· BCRI divisional con grado de bloqueo en la división postero-inferior mayor de lo que el bloqueo de la división antero-superior izquierda presenta.
· BCRI con cambio accidental de los electrodos de los miembros.
También presenta infradesnivel en cara inferior y me llama la atención las ondas r aumentadas de voltaje en V2-V3 y V4.
Gracias Profesor Perez Riera por todo. Gracias por recomendar el libro de Surawicz, es muy interesante.
Les dejo con el permiso de Edgardo el nombre de una pagina italiana de arritmias, http://www.cardiolink.it/ecg-bachecaecg.asp (o la pueden buscar como ECG-online del Doctor Giuseppe Oreto).
Abrazos a todos y lo mejor.
Lucas

Ken Rosen MD did a study in the late 1970- early 1980's at Cook County Hospital in Chicago.
He subjected all patients who were identified as having some form of bundle branch block to routine and serial His Bundle Studies to determine the spectrum of infra-Hisian disease. All of these patients were asymptomatic and the bundle branch block was an incidental discovery at the time of a routine ECG, a pre-op ECG or any other reason but the important point is that the ECG was obtained as a screening test in the absence of symptoms to cause a physician to look for a cardiac etiology of their symptoms. As all patients were asymptomatic, no specific therapy was initiated independent of the HV interval. Dr. Rosen is one of the investigators who identified an HV of 100 ms or longer as a good prognostic indicator for the patient's subsequent need for a pacemaker.
In this population of patients, those who subsequently returned with syncope underwent a more intensive invasive "His-bundle study" [this was before these studies were called electrophysiologic studies].
Approximately 1/3 had demonstrable significant infra-Hisian disease and underwent implantation of a permanent pacemaker. Another 1/3 had inducible VT and were started on a variety of antiarrhythmic agents being guided by repeated EP studies until a single drug or combination of drugs was effective in preventing the inducible VT or at least slowing it down so that it was able to be tolerated (this was pre-ICD). Another 1/3 had no inducible or identifiable etiology. This was prior to the appreciation of Tilt Table Testing for neurocardiogenic syncope which is likely to have unmasked the cause in some of the remaining 1/3 but I doubt this would account for all the remaining patients.
The point to be made by the above is that even with the demonstration of marked infra-Hisian disease [above 55 ms is a bit too short for my preference in recommending a pacemaker (I would prefer 70 ms and above)], also look for inducible VT.

While the ICDs in the mid to late 1980's were simple shock boxes and could not provide pacing support, our current generation of ICDs can also provide pacing support. It would be a shame to implant a pacemaker for what might be an incidental finding with a mildly prolonged HV interval only to have the patient have recurrent syncopal episodes after the implant and even sudden death that was preventable by an ICD.

Virtually by definition, all individuals who have some conduction system disease (or a disease of the electrical system of the heart) and as such, have heart disease. Mechanically the heart disease might be mild but just like the channelopathies (e.g. Long QTSyndrome) were the mechanical function of the heart is usually normal, the electrical problems can still kill the patient.

With respect to the comment that with pacing, one should intentionally chose to implant a CRT system is very interesting. There is not a lot of support for this but the recent MADIT-CRT trial sponsored by Boston Scientific, the PAVE trial sponsored by St. Jude Medical and some smaller studies from Europe suggest that in those patients who will need to be paced in the ventricle, CRT may have some long term benefits and at least delay the development of overt heart failure. More studies involving larger numbers of patients followed for longer periods of time need to be performed. Increasing numbers of physicians are tending towards implantation of a CRT system in patients who need ventricular pacing, particularly with an underlying bundle branch block but who do not yet have overt pharmacologically refractory heart failure.

•Medtronic has popularized the idea of saying no to all ventricular pacing but that presumes intact AV nodal conduction at a normal PR interval in patients who need pacing for sinus node dysfunction.

There are no studies that I am aware of in a population of patients who have a bundle branch block and might need a pacemaker as to how they should be managed even if their indication for pacing is sinus node dysfunction. I disagree with the mantra of "say no to ventricular pacing" - rather it should be "say YES to an appropriate AV delay" but this is a topic for another discussion.

El Dr. Ken Rosen realizó un estudio a fines de los 1970, principios de los 1980, en el Hospital Cook County de Chicago. Sometió a todos los pacientes que se identificaron con alguna forma de bloqueo de rama, estudios del haz de His de rutina y seriales, para determinar el espectro de la enfermedad infra-Hisiana. Todos estos pacientes eran asintomáticos, y el bloqueo de rama era un descubrimiento indicental a raíz de un ECG de rutina, ECG pre-operatorio o cualquier otra razón; pero lo importante es que el ECG se obtenía como "screening" en ausencia de síntomas que hicieran que un médico buscara una etiología cardíaca para sus síntomas. Como todos los pacientes eran asintomáticos, no se inició terapia específica independiente del intervalo HV. El Dr. Rosen es uno de los investigadores que identificó un HV de 100 ms o más, como un buen indicador pronóstico para la necesidad posterior del paciente de marcapasos.

En esta población de pacientes, los que regresaron luego con síncope, se sometieron a un estudio de haz de His más intensivo e invasivo [esto fue antes de que estos estudios se denominaran estudios electrofisiológicos].

Aproximadamente 1/3 tuvo enfermedad infra-Hisiana significativa demostrable, y se sometieron a implante de marcapasos permanente. Otro 1/3 tuvo TV inducible, y comenzaron con una variedad de agentes antiarrítmicos, siendo guiados por estudios EF repetidos, hasta que una droga única o combinación de drogas resultara efectiva en la prevención de TV inducible, o al menos retardándola de manera que fuera tolerable (esto fue antes de los CDI). Otro1/3 tuvo etiología no inducible o identificable. Esto fue antes de la apreciación de la prueba de mesa basculante para el síncope neurocardiogénico, que probablemente habría desenmascarado la causa en algunos del 1/3 restante, pero dudo que esta fuera la causa en los pacientes restantes.

El punto a destacar de lo anterior, es que incluso con la demostración de enfermedad infra-Hisiana marcada [más de 55 ms es un poquito corto para mi preferencia para recomendar un marcapasos (yo preferiría 70 ms y más)], también hay que buscar TV inducible.

Mientras los CDI a mediados o fines de los 1980, eran simples cajas de descargas y no podían ofrecer soporte de estimulación, nuestra generación actual de CDI también puede ofrecer soporte de estimulación. Sería una vergüenza implantar un marcapasos por lo que podría ser un hallazgo incidental con un intervalo HV ligeramente prolongado, solamente para que el paciente sufriera episodios sincopales recurrentes luego del implante, e incluso muerte súbita que fuera evitable con un CDI.

Prácticamente por definición, todos los individuos que tienen alguna enfermedad del sistema de conducción (o enfermedad del sistema eléctrico del
corazón) y como tales, tienen una cardiopatía. Mecánicamente la cardiopatía puede ser leve, pero al igual que con las canalopatías (por ej., síndrome de QT prolongado), donde la función mecánica del corazón generalmente es normal, los problemas eléctricos aun así pueden matar al paciente.

Con respecto al comentario de que con estimulación, uno debe intencionalmente elegir implantar un sistema de TRC, es muy interesante. No hay mucho respaldo para esto, pero el reciente ensayo MADIT-CRT, patrocinado por Boston Scientific, el ensayo PAVE patrocinado por St. Jude Medical, y algunos estudios menores de Europa, sugieren que en aquellos pacientes que precisarán estimulación en el ventrículo, la TRC puede tener beneficios a largo plazo y al menos un retardo en el desarrollo de insuficiencia cardíaca expresa. Deben realizarse más estudios que involucren una mayor cantidad de pacientes, seguidos por períodos más prolongados de tiempo. Una cantidad creciente de médicos tienden hacia el implante de un sistema de TRC en pacientes que precisan estimulación ventricular, especialmente con bloqueo de rama subyacente, pero que no tienen aun insuficiencia cardíaca farmacológicamente refractaria expresa.

•Medtronic ha popularizado la idea de decir que no a toda la estimulación ventricular, pero eso supone una conducción intacta del nodo AV a un intervalo PR normal en pacientes que precisan estimulación para la disfunción del nodo sinusal. No hay estudios que yo sepa, en una población de pacientes con bloqueo de rama y que pudieran necesitar marcapasos, en relación a cómo deben tratarse incluso si su indicación para la estimulación es la disfunción del nodo sinusal. No estoy de acuerdo con el mantra de "decirle no a la estimulación ventricular" – por el contrario debería ser "decirle SÍ a un retardo AV adecuado", pero ése se un tema para otra discusión.

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Dear friend Roges,

With all due respect, and without going deep into the ECG or the case's analysis, allow me to disagree with your point of view.
First moment impressions, as you call them, could be fatal, if not supplemented with an appropriate clinical judgement, experience, and evidence. Allow me to suggest, as a friend of the forum, to ALWAYS mistrust your first impression, mistrust what you've been taught, what you read, what you are told. Before making a judgement (conducting an electrophysiologic study or not, for instance) bring all those elements together, make an analysis, and then make a decision.
Do not stay with one point of view: I AM AGGRESSIVE. Sometimes, being aggressive is good, sometimes is bad. Sometimes you have to be invasive, and sometimes you have to hold your guns, and save them for another battle. Do not confine to one position.
The risk of confining to one position, is to get stuck with a single point of view, fixed and immovable.
The key to what we do is variety, in sometimes indicating a device, and sometimes not; in seeing a patient 4 times a month, or seeing him/her once every 2 years; in saying yes or saying no about the same case.
And finally, and I am not reinventing the wheel with this, follow the evidence (but with the ability to surround it) and you will find the road of science (and magic).
Warm regards from Canada,

Estimado Amigo Roges

Con total respeto, y sin adentrarme en el analisis del ECG, ni del caso; permitame discrepar con su posicion.
Las impresiones de primer momento, como las llama Ud, pueden ser fatales, si no se las complementa con el adecuado juicio clinico, la experiencia y la evidencia. Permitame sugerirle, como amigo del foro, que SIEMPRE desconfie de su primera impresion, desconfie de lo que le enseniaron, de lo que lea, de lo que le cuenten. Antes de hacer el juicio (hacer o no hacer un estudio electrofisiologico, por ejemplo) traiga todos estos elementos juntos, analice, y luego decida. No se case con una postura: SOY AGRESIVO. A veces, ser agresivo es bueno, a veces es malo. A veces hay que invadir, y a veces dejar las armas quietas, y usarlas en otra batalla. No se autodefina.
El riesgo de autodefinirse es quedar pegado a una postura unica, fija e innamovible.
La gracia de lo que hacemos esta en la variedad, en a veces, indicar un dispositivo, y a veces no, en ver al paciente 4 veces por mes, o verlo 1 vez cada 2 anos, en decir si o en decir no sobre el mismo caso.
Y por ultimo, y no voy a descubrir la polvora con esto, ajustrase a la evidencia (pero con la cintura suficiente para circunvalarla) lo llevara por el camino de la ciencia (y de la magia).
Lo saludo fraternalmente desde Canada
AB

- **Wise words, it is the only thing I can say. Thank you for your opinion. I am flexible and I analyze as a Libra, but it is true that within the aggressive area...I have to learn not to feel compelled by the chronometer. So, I appreciate your words, and honestly, I think they are useful.**
- **Roges**

- **Sabias palabras es lo unico que puedo decir gracias por su opinion. Soy flexible y analizo como librano pero es cierto que dentro de la esfera agresiva..... debo aprender a no sentirme acosado por el cronómetro por lo que aprecio sus palabras y sinceramente las considero útiles**

- **Roges**

With respect to this most recent comment suggesting that one should always consider a differential diagnosis, I agree with this recommendation.

I would like to share a personal approach that I have now used for more than 30 years. It came about in a very interesting manner. I had been invited to give Medical Grand Rounds at a hospital in Connecticut. The topic was "indications for pacing". On the topic of Mobitz II second degree AV Block, one of the participants at the end of my presentation asked if I would still "automatically" recommend a permanent pacemaker if the intermittent low grade (only rare blocked QRS) Mobitz II second degree AV Block was discovered incidentally in an asymptomatic patient? My answer was "yes". In follow-up to the first question, he asked would I still make this recommendation if the patient was my wife or one of my parents? Feeling the need to be consistent, I said "yes" however that was an outstanding question. I wish I knew who the physician was who asked the question to now thank him.

Since that time, whenever I was presented with recommending an invasive procedure, whether it was doing an EP or cath study, implanting a pacemaker or ICD, referring the patient for open heart surgery, ... I started asking myself (before I made the recommendation) if I would make this same recommendation at this time in the patient's evaluation if the patient was my wife, one of my parents or my sibling? Sometimes the answer was no, other things needed to be done first. When the answer was yes and a complication was subsequently encountered,

I was upset that the complication occurred but I could comfortably sit down with the patient(hopefully) and family and review everything that happened, the basis for making that recommendation and, based on the knowledge of the day, what I anticipated as the outcome if the intervention had not been done. For example if we didn't implant a pacemaker in a patient with recurrent syncope and complete heart block.

I am only sorry that no one has yet invented the crystal ball to see into the future. Then decision making would be easy. Clearly if I knew in advance that a major complication would occur (for example, in the setting described above, what if a myocardial perforation occurred that could not be managed with observation and tamponade not only occurred but then recurred after emergency pericardiocentesis such that referral to a CT Surgeon was required for an emergency open chest procedure?), I might elect a different approach for that patient. But we do not have the magic crystal ball. If we did, all of our decisions would be easy.

I agree with the recommendation to not only consider all potential options, but also but also do not rush in to invasive procedures without considering whether or not all options have been covered. In the course of my career, I have had 4 myocardial perforations associated with permanent pacemaker implant procedures (that I know about). Two of these required the assistance of my thoracic surgical colleagues. One of these involved an elderly (over 80 years) who presented with funny ill-defined "spells". We identified Wenckebach 2nd degree AV Block on the admission ECG. An "acute" MI was ruled out by negative enzymes (CPK-MB, SGOT and LDH, this was pre- Troponin), we did not see an acute MI on her ECG and the next morning took her to the lab to implant a pacemaker. The ventricular lead perforated the RV wall like a hot knife through butter. When her chest was opened, we found the lead had gone through an area of liquifaction necrosis involving the anterior RV wall. A small biopsy subsequently demonstrated evidence of a 1-2 week old acute MI subsequently confirmed with right anterior wall ECG leads. Her symptoms were probably due to the MI and the low grade second degree AV block is very likely to have resolved on its own with a little time. This is another patient in whom access to a crystal ball would be nice. This is also patient who taught me a very valuable lesson.

If the problem that you see is not immediately life-threatening, take some time before rushing in.

Con respecto al comentario más reciente, sugiriendo que uno siempre debería considerar un diagnóstico diferencial, estoy de acuerdo con esta recomendación.

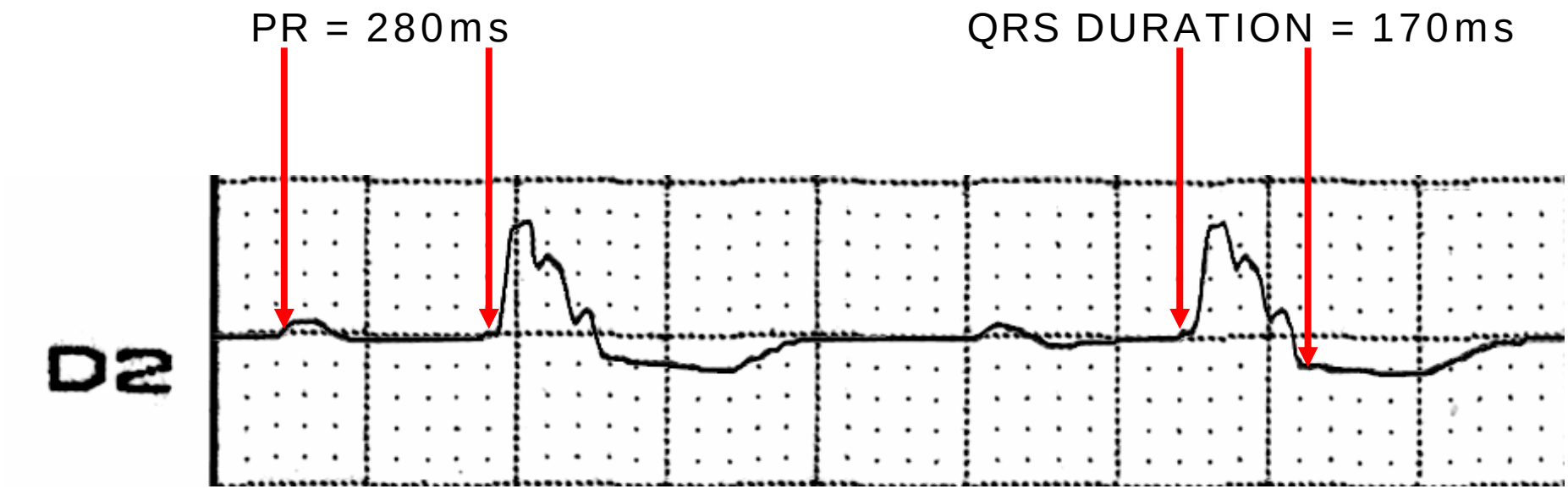
Me gustaría compartir un enfoque personal que he usado por más de 30 años. Surgió de manera muy interesante. Me habían invitado a dar sesiones principales médicas en un hospital de Connecticut. El tema era "indicaciones de estimulación". Sobre el tema de bloqueo AV de segundo grado tipo Mobitz II, uno de los participantes al final de mi presentación preguntó si yo seguiría recomendando "automáticamente" un marcapasos definitivo si se descubriera incidentalmente bloqueo AV de segundo grado tipo Mobitz II, de grado inferior intermitente (sólo el poco frecuente QRS bloqueado) en un paciente asintomático. Mi respuesta fue "sí". Como consecuencia de la primera pregunta, me preguntó si aun haría esta recomendación si el paciente fuera mi esposa o uno de mis parientes. Sentí la necesidad de ser consistente, y dije que "sí", sin embargo la pregunta era extraordinaria. Desearía saber qué médico hizo la pregunta, para agradecérsela hoy.

Desde ese momento, siempre que se me planteaba recomendar un procedimiento invasivo, ya sea un estudio EF o un estudio de cateterismo, implantar un marcapasos o CDI, derivar el paciente a cirugía a corazón abierto… comencé a preguntarme (antes de hacer la recomendación) si haría la misma recomendación en este momento en la evaluación del paciente si el paciente fuera mi esposa, uno de mis parientes o mi hermano. A veces la respuesta es no, porque es necesario hacer otras cosas primero. Cuando la respuesta era sí y se hallaba un complicación más tarde, me perturbaba que la complicación ocurriera, pero podía sentarme cómodamente con el paciente (com optimismo) y su familia, y revisar todo lo que había ocurrido, la base para hacer tal recomendación y, en base al conocimiento del día, lo que anticipaba como resultado si la intervención no se había realizado. Por ejemplo, si no implantaba un marcapasos en un paciente con síncope recurrente y bloqueo cardíaco completo.

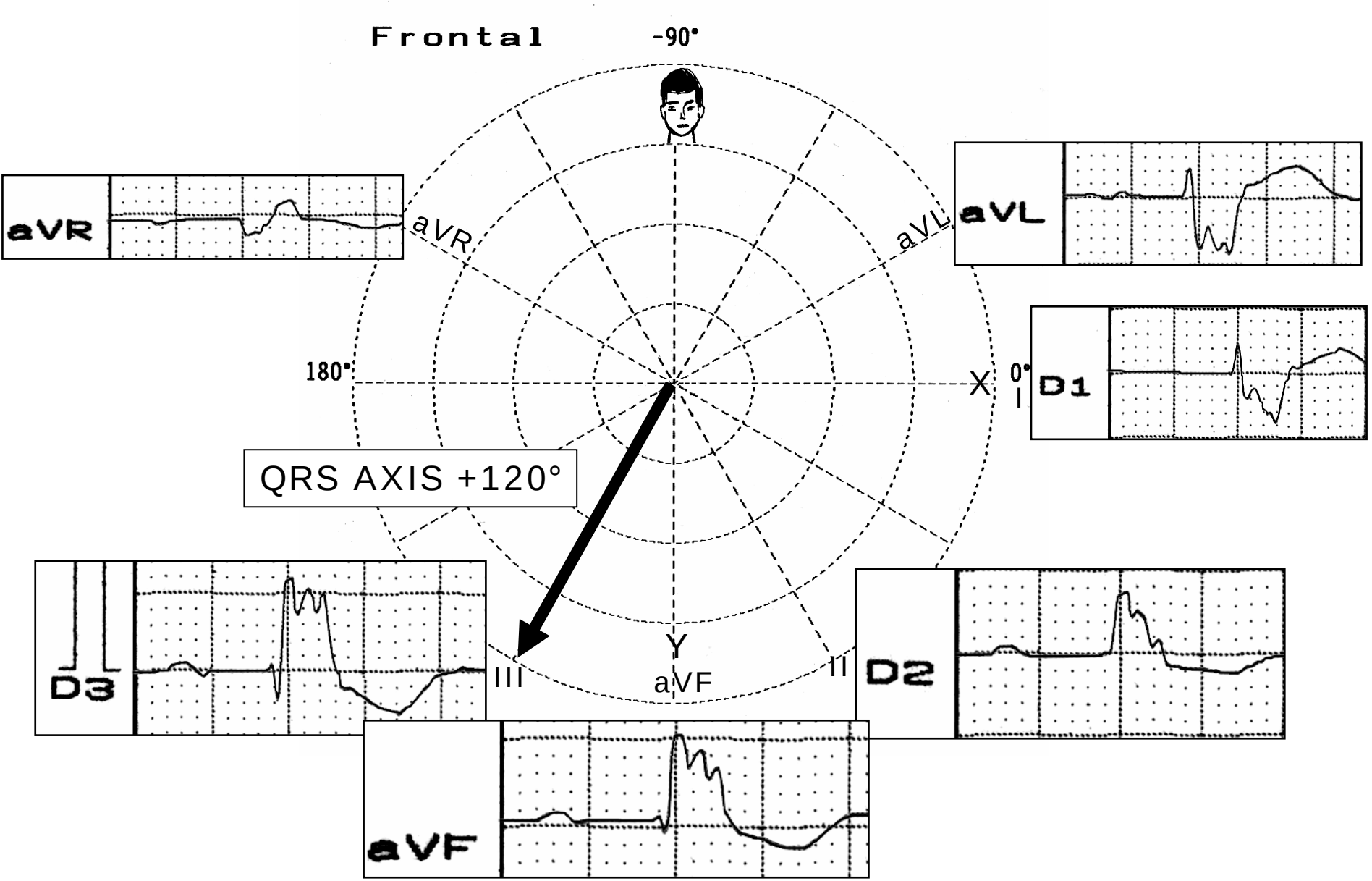
Solamente lamento que nadie haya inventado aun la bola de cristal para ver el futuro. Entonces la toma de decisiones sería fácil.

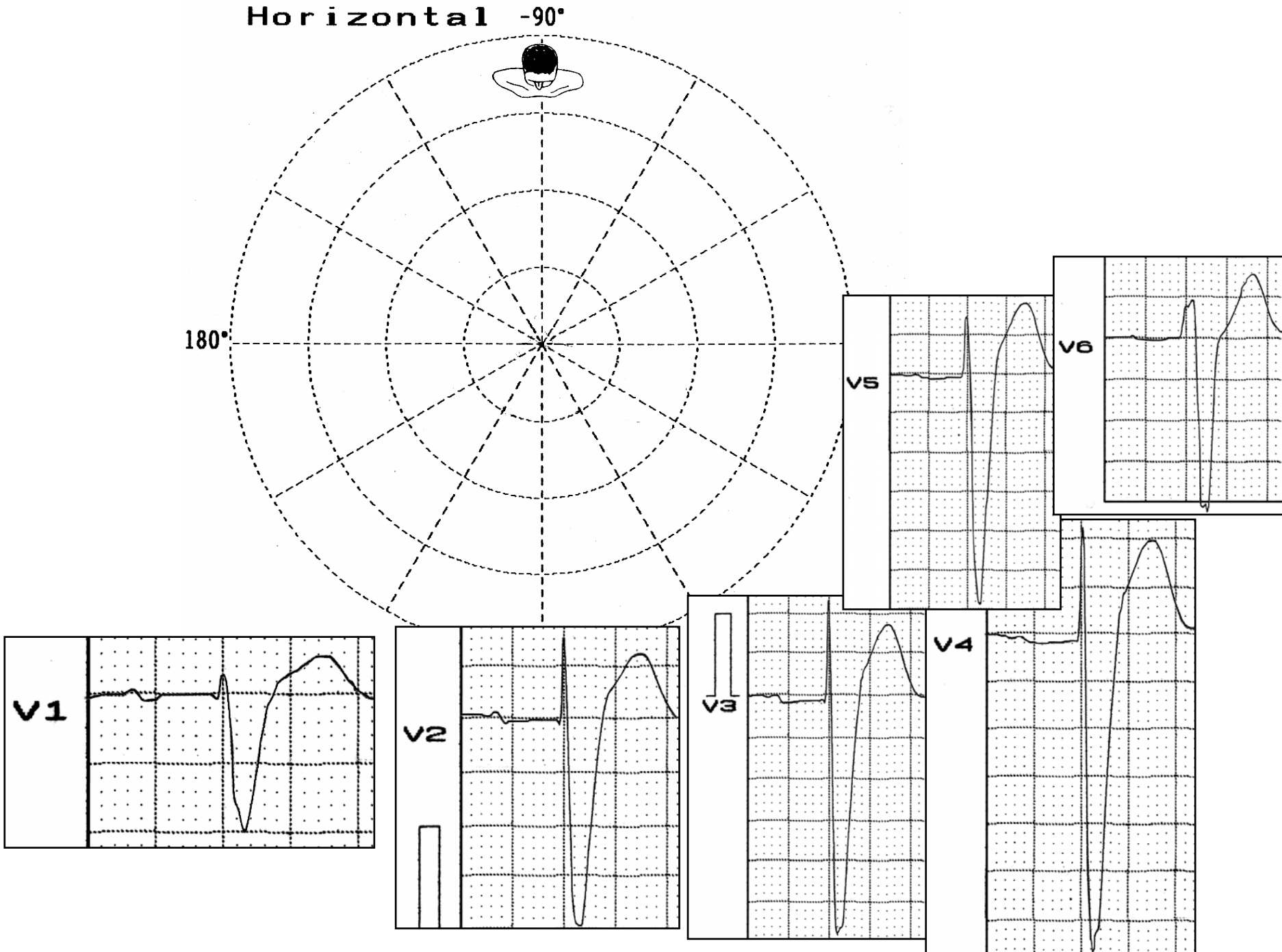
Claramente si supiera por adelantado que va a ocurrir una complicación mayor (por ejemplo, en el contexto descrito antes, ¿y si ocurriera perforación miocárdica que no pudiera tratarse con observación y no sólo ocurriera taponamiento sino que luego recurriera después de pericardiocentesis de emergencia, de manera tal que la derivación a un cirujano cardiotorácico fuera necesaria para un procedimiento de emergencia a corazón abierto?), podría elegir un enfoque diferente para ese paciente. Pero no tenemos una bola de cristal. Si la tuviéramos, todas nuestras decisiones serían fáciles. Estoy de acuerdo con la recomendación de no sólo considerar todas las opciones potenciales, sino también en no apresurarse a realizar procedimientos invasivos sin considerar si se han cubierto todas las opciones o no. En el curso de mi carrera he tenido 4 perforaciones miocárdicas asociadas con procedimientos de implante de marcapasos definitivo (que yo sepa). Dos de los mismos precisaron la asistencia de mis colegas cirujanos torácicos. Uno de ellos implicó a una paciente anciana (de más de 80 años) que se presentó con "malestares" raros y poco definidos. Identificamos bloqueo AV de segundo grado de Wenckebach en el ECG de ingreso. Se descartó IAM por enzimas negativas (CPK-MB, SGOT y LDH, esto fue en la época pre-troponina). No vimos IAM en su ECG y a la mañana siguiente la llevamos al laboratorio para implantarle un marcapasos. El electrodo ventricular perforó la pared del VD como un cuchillo caliente a través de manteca. Cuando abrimos su tórax, hallamos que la derivación había atravesado un área de necrosis por licuefacción que comprometía la pared anterior del VD. Una pequeña biopsia demostró posteriormente evidencias de IAM de 1-2 semanas, confirmado luego por las derivaciones ECG de la pared anterior derecha. Sus síntomas probablemente se debieran a IM; y el bloqueo AV de segundo grado de grado bajo muy probablemente se hubiera resuelto por sí mismo en poco tiempo. Éste es otro paciente en quien el acceso a una bola de cristal sería agradable. Éste también es un paciente que me enseñó una lección muy valiosa. Si el problema que se ve no es una amenaza inmediata para la vida, hay que tomarse un tiempo y no apurarse.

Paul A. Levine MD Vice President, Medical Services St. Jude Medical CRMD

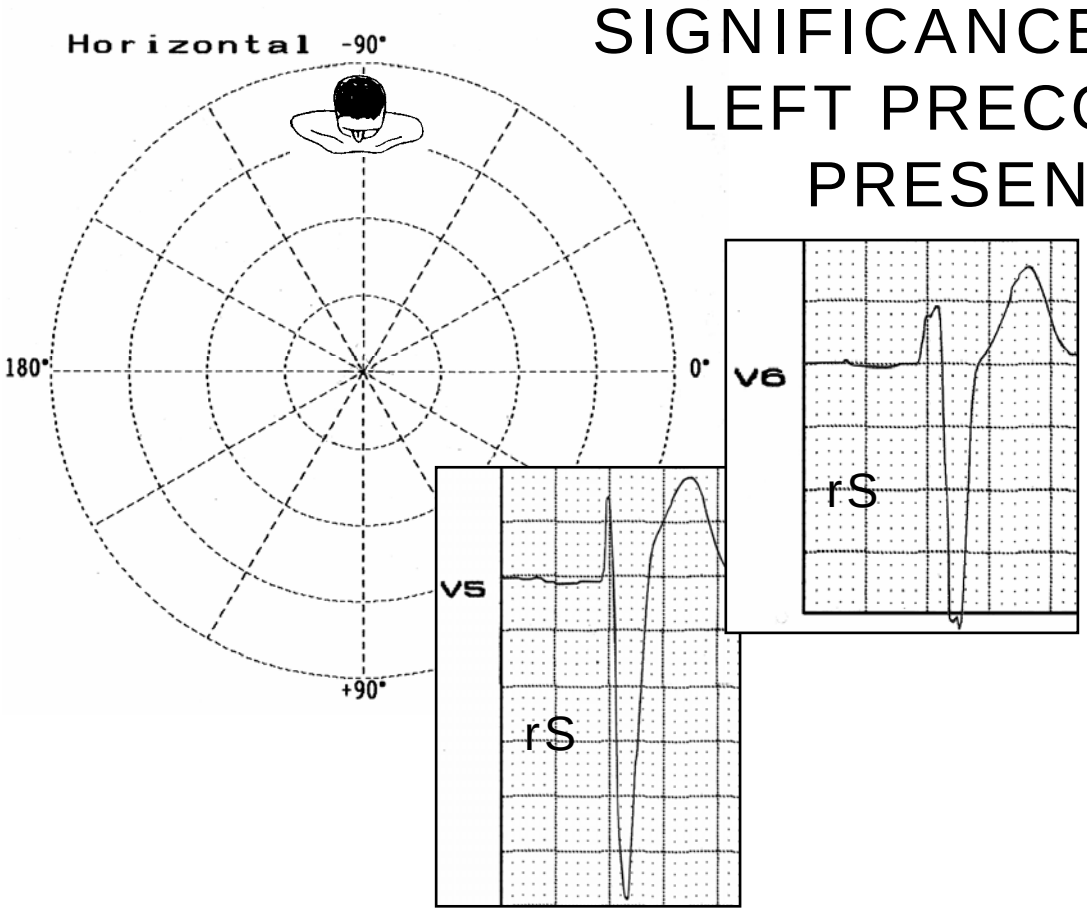

PR = 280ms
QRS DURATION = 170ms
D2

QRS AXIS


Frontal
-90°
aVR
aVL
180°
0°
I
X
Y
III
aVF
II
QRS AXIS +120°
aVR
aVL
D1
D3
D2
aVF


Horizontal -90°
180°
V1
V2
V3
V4
V5
V6

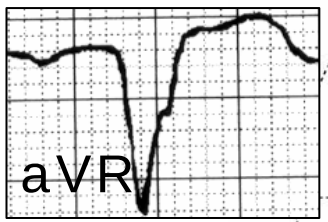
SIGNIFICANCE OF rS PATTERN IN LEFT PRECORDIAL LEADS IN PRESENCE OF CLBBB



In presence of CLBBB with Rs or rS pattern in left precordial leads(V5 -V6) may indicate:

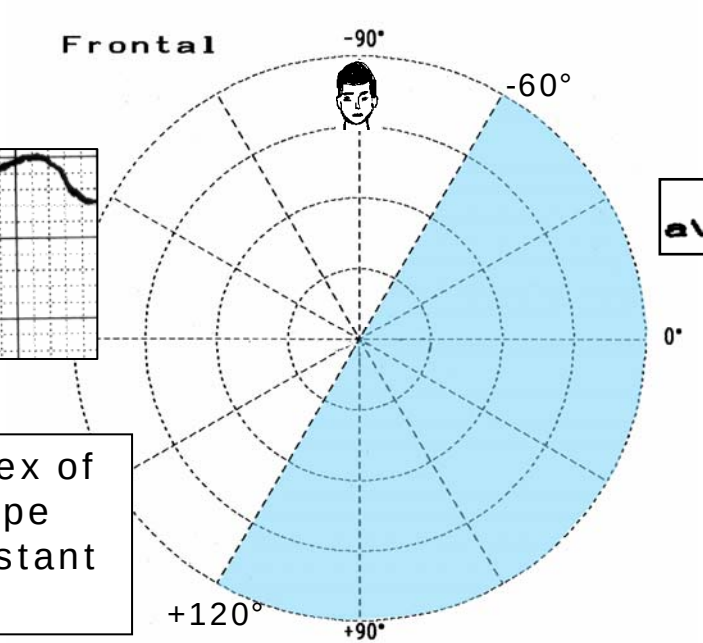
1) Displaced transition zone of QRS complex to left (Left Ventricle Posterior)
2) Association Right Ventricular Hypertrophy (RVH) or Right Ventricular Enlargement (RVE)
3) Associated left anterior fascicular block (LAFB)
4) Complete LBBB complicated with left ventricular free wall myocardial infarction.

COMPLETE LBBB: QRS PATTERN IN aVR LEAD



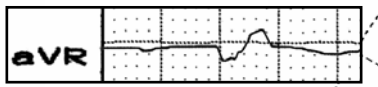
QS

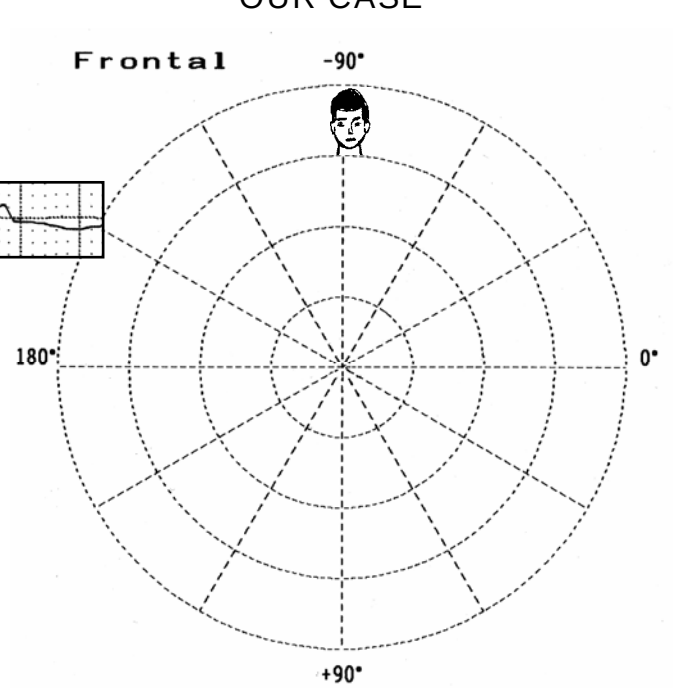
QRS complex of the QS type almost constant in aVR.



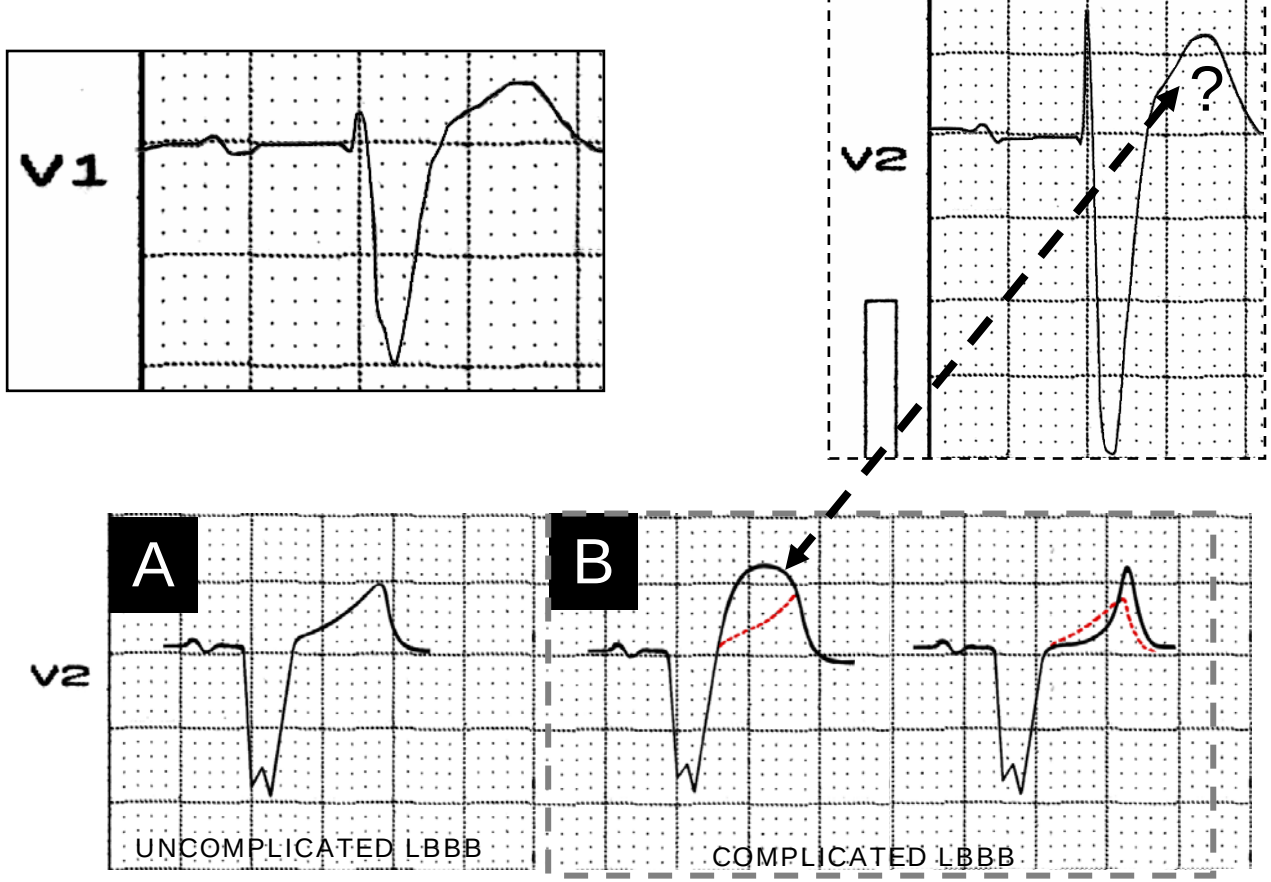
In presence of LBBB with QRS axis between -60°/+120° in aVR the QRS complex has always QS pattern.

OUR CASE





In this case, aVR has minus-plus isodiphasic QRS complex.
A diphasic complex whose positive and negative deflections are approximately equal. It is indicative of paradoxical LBBB of Lepeschkin.



- **A:** The elevated ST segment has a straight upward slope, or an upward slope that is minimally concave-upwards.
  The T wave is upright, with asymmetrical limbs and a relatively blunt apex.

- **B:** With coronary insufficiency or AMI the ST segment elevation is exaggerated (≥5 mm) in the right precordial leads and becomes coved, convex-upward.
  The T wave becomes inverted and/or its limbs tend to become more symmetrical.

Modified from Leo Schamroth. The Electrocardiology of Coronary Artery Disease. Blackwell Scientific Publications. Oxford London Edinburgh Melbourne. 1975; pg 86.

Final ECG diagnosis

- **Sinus rhythm: Supraventricular command**
- **First-degree AV block: prolonged PR interval = 280ms**
- **Atypical Left Bundle Branch Block (LBBB): Paradoxical Lepeschkin type because QRS axis is located beyond + 90 degree (+120 degree = isodiphasic aVR lead).**
- **This ECG pattern (LBBB + extreme right axis deviation beyond + 90 degree) has an ominous prognosis because it is a a marker of advanced congestive cardiomyopathy(1**).
- **In a patient with LBBB, the occurrence of both first-degree A-V block and a QRS duration ≥ 160ms strongly suggested the likelihood of H-V prolongation (2**).

1. **Nikolic G, Marriott HJ. Left bundle branch block with right axis deviation: a marker of congestive cardiomyopathy. J Electrocardiol. 1985 Oct;18:395-404**
2. **Rosen KM, Ehsani A, Shahbudin H, Rahimtoola MB, Clinical and Electrocardiographic Correlations H-V Intervals in Left Bundle-Branch Block *Circulation*. 1972;46:717.**

COMMENTARIES

- In patients with chronic bundle branch block ≈ 33% show progressive AV conduction system disease and AV nodal and infranodal disease progress independently.
- Progression of infranodal disease is more common in feminine gender.
- AV nodal disease progress independently. AV nodal disease is a common cause of AV block and can occur without further prolongation of the AH interval once a critical level of disease is attained, whereas infranodal block is usually accompanied by progressive lengthening of the HV interval.
- Progression of AV conduction disease is not readily predictable from clinical and electrophysiologic variables (1).
- In LBBB mortality is higher in patients with associated left axis deviation than in those with a normal axis, although the incidence of progression of AV block is low.
- In symptomatic patients with prolonged His to ventricular intervals, the incidence of progression of AV block is higher (12%).
- Preexisting LBBB in the absence of clinical evidence of heart disease is rare, yet carries with it a slightly increased mortality.
- Newly acquired LBBB carries a 10-fold increase in mortality
- The incidence of SD as the first manifestation of heart disease is increased 10-fold(2).

- **Peters RW, Scheinman MM, Dhingra R, Rosen K, McAnulty J, Rahimtoola SH, Modin G. Serial electrophysiologic studies in patients with chronic bundle branch block. Circulation. 1982 Jun;65:1480-14485.**
- **Flowers NC. Left bundle branch block: a continuously evolving concept. J Am Coll Cardiol. 1987 Mar;9:684-689.**

Patients with NYHA class III or IV, LVEF ≤ 35% and a QRS interval ≥120ms may benefit from bi-ventricular pacemaker (CRT) placement or surgical remodelling of the heart. These treatment modalities may make the patient symptomatically better, improving quality of life and in some trials have been proven to reduce mortality.

- In the COMPANION trial, CRT (pacing the LV well as the RV) has been shown to improve survival in individuals with NYHA class III or IV (moderate to severe HF) HF with a widened QRS complex on ECG.
- The CARE-HF trial, showed that patients receiving a Medtronic bi-ventricular pacemaker (CRT) and optimal medical therapy benefit from a 36% reduction in all cause mortality, and a reduction in cardiovascular related hospitalization.
- Additionally, patients with NYHA class II, III or IV, LVEF of 35% (without a QRS requirement) may benefit from an Implantable Converter Defibrillator (ICD), a device that is proven to reduce all cause mortality (death) by 23% compared to placebo. This mortality benefit was observed in patients who were already optimally managed on drug therapy.
- Another current treatment involves the use of left ventricular assist devices (LVADs). LVADs are battery-operated mechanical pump-type devices that are surgically implanted on the upper part of the abdomen. They take blood from the LV and pump it through the aorta. LVADs are becoming more common and are often used by patients who have to wait for heart transplants.
- Acorn Cardiovascular, based in St. Paul, Minnesota, recently created the CorCap Cardiac Support Device (CSD), also known as the "heart sock." It is a dacron mesh that is placed around the heart. The elastic CSD works by mechanically restoring the contractility of the expanded heart. The CorCap CSD recently failed to be approved by the FDA.
- The ultimate treatment is cardiac transplant surgery (heart transplant) or implantation of an artificial heart.

Cardiac resynchronization therapy (CRT) lowered morbidity and mortality in patients with moderate to severe HF. Gervais et al. examined whether baseline and follow-up ECGs characteristics might predict long-term outcome.

CARE-HF(1) randomly assigned 409 patients to medical therapy (MT) plus CRT, and 404 patients to MT alone.

ECG measurements were made at baseline during SR, and at 3 months during paced or spontaneous rhythm depending on treatment assignment.

Favorable outcome was defined as freedom from death, urgent transplantation, or cardiovascular hospitalization.

Among patients assigned to CRT, 39% had unfavorable outcomes including 55 deaths. By single variable analysis:

1. Prolonged PR interval
2. Left QRS axis (but not QRS duration)
3. LBBB) at baseline

HR, PR, and QRS duration at 3 months predicted unfavorable outcome. By multiple variable analysis, treatment assignment, PR, and RBBB at baseline predicted outcome, whereas baseline JTc and QRS duration at 3 months predicted all-cause mortality and HF hospitalization.

In CARE-HF, QRS duration at baseline did not predict outcome, but QRS at 3 months was a predictor by single variable analysis. Patients with prolonged PR interval and the 5% of patients with RBBB had a particularly high event rate.

1. **Gervais R, Leclercq C, Shankar A, Jacobs S, Eiskjaer H, Johannessen A, Freemantle N, Cleland JG, Tavazzi L, Daubert C; CARE-HF investigators. Surface electrocardiogram to predict outcome in candidates for cardiac resynchronization therapy: a sub-analysis of the CARE-HF trial. Eur J Heart Fail. 2009 Jul;11:699-705.**