Paciente do sexo masculino, 69a, hipertenso, com história de pré-sincope - 2009

Dr. Raimundo Barbosa Barros

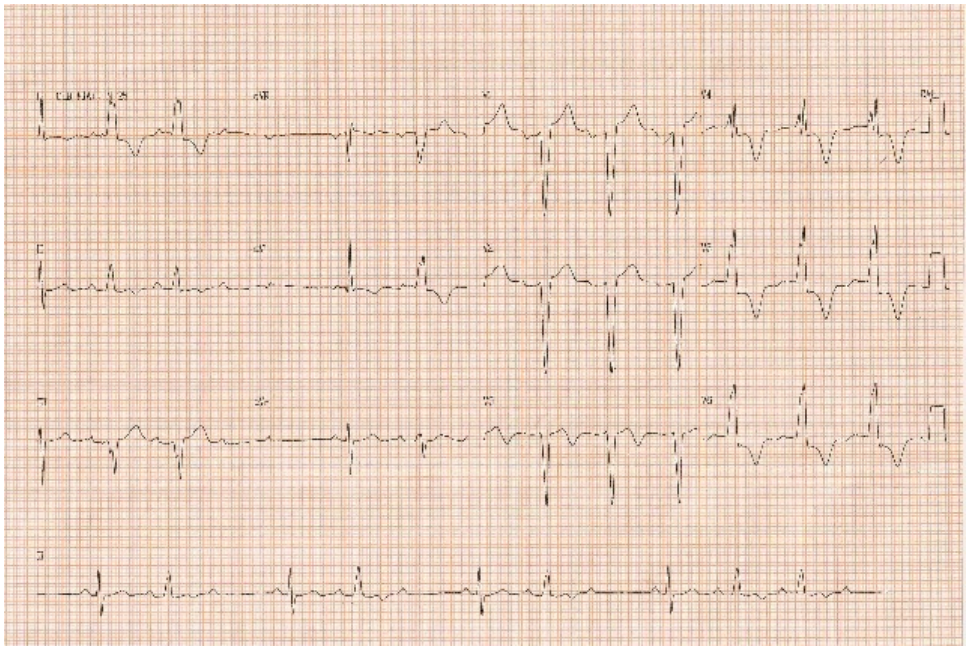
Paciente do sexo masculino, 69a, hipertenso, com história de pré-sincope.

Perguntas:

1) Qual o diagnóstico Eletrocardiográfico?

2) Possui indicação de implante de marcapasso definitivo?

Um abraço
Raimundo Barbosa de Barros



OPINIONES DE COLEGAS

Andres and everyone to whom this response will be shared (my apologies, I should have replied to all)

The ECG shows two phenomena. One is Wenckebach 2nd degree AV block. The key feature being the PR interval prior to the blocked P wave being longer than the PR interval following the block.

The second is the intermittent BBB (left bundle morphology). The rate is a little slow for Ashman's functional aberration although one setting in which Ashman's aberration occurs is Wenckebach and then, commonly, the second conducted complex aberrates with the third conducted complex normalizing intra-ventricular conduction. The last 6 complexes in the 12 lead section of the ECG shows consistent BBB such that Ashman's phenomenon is unlikely. LBBB does not exclude Ashman's for while the usual pattern is RBBB, that is 80% while LBBB can occur in 20%.

Hence, I think that this is a rate-dependent (phase 3) aberration. This is more common in CAD and with a history of hypertension, CAD is highly likely.

If this patient was totally asymptomatic, I would follow this patient prospectively and not even consider implantation of a permanent pacemaker. However, given the recent history of "near-syncope", I am still not ready to rush to implant a pacemaker. I would, however, recommend a further cardiac evaluation. I would start with an Echo and an ETT. Anticipating that the patient will develop LBBB at higher sinus rates, some other imaging modality will be required as one cannot rely on the standard ECG with ST segment changes. IF there are signs of ischemia, I would proceed with a cardiac catheterization and if there are significant lesions, I would consider revascularization.

If there is no sign of significant ischemic disease or after revascularization, I would take the patient to the EP lab evaluating both AV Conduction (significant infra-His disease would

warrant a pacemaker) and inducibility of monomorphic VT. If VT can be induced, I would favor implantation of an ICD.

If VT cannot be induced and I cannot confirm significant infra-His disease I would follow the patient without implanting a pacemaker or ICD.

It might be appropriate to implant a subcutaneous loop recorder if a definite explanation is not forthcoming from the EP lab, particularly if the catheterization demonstrated no evidence of significant CAD.

If the patient had significant CAD and underwent a revascularization procedure with a subsequent non-diagnostic EP study, I would prospectively follow him and only consider implant of a loop recorder if there are recurrent near-syncopal or overt syncopal episodes.

I do not think there is sufficient evidence based on the ECG alone to rush to implant a pacemaker. This patient his highly likely to have other cardiac disease. The ECG and clinical history provide sufficient evidence to prompt a more extensive evaluation.

Paul A. Levine MD (QEPD)

Hola!

Muy interesante registro. En mi opinión impresiona ser un fenómeno de Wenckebach de la Rama Izquierda con periodicidad variable (3:2 , 4:3 , etc). Creo que en este caso y con antecedentes de episodios pre-sincopales o mareos merecería la colocación de un MPD. Saludos cordiales.

Damián Longo

Greetings

I totally agree that this is Wenckebach second degree AVB and rate dependent BBB. Prof.'s Levine prospective plan is great, but I would like to start this evaluation by doing an exercise test to see how the underlying block will behave before taking him to EP lab.

My question to Prof. Levine, would you try to induce VT in this patient if he had a normal or near normal heart? or LVEF > 40%?

Thank you

Raed Abu Sham'a, MD
Jerusalem – Palestine

Raed,

There was a fascinating study years ago by Ken Rose (University of Chicago) who did serial (once a year) EP studies in asymptomatic patients with BBB in various manifestations. No therapy was prescribed. If the patient then developed symptoms, they were re-studied at that time. The general results were that 1/3 had further increase in AV conduction disease (increase HV interval, overt 2nd degree AV block at lower atrial paced rates) and then received a pacemaker, 1/3 had inducible VT and were treated with either drugs or an ICD and 1/3 had no identifiable electrical cause of their symptoms.

My first two tests would be an echo to evaluate chamber size and an estimate of EF and ventricular function and an ETT. If the ETT showed evidence of ischemia, I would pursue this line of evaluation before an EP study. If the ETT failed to show signs of ischemia and did not unmask further signs of AV conduction system disease over and above a rate dependent BBB, I would proceed with an EP study independent of the EF. A more detailed history might also suggest the need for a head-up tilt table test.

If I did the EP study prior to a cardiac cath and demonstrated inducible VT but then identified significant ischemia, I would treat the ischemia before considering an ICD. After resolution of the ischemia (angioplasty +/- stent or bypass surgery), I would repeat the EP study the patient with respect to inducible VT. This is the reason that I deferred the EP

study until after the cardiac cath.

Paul A. Levine MD (QEPD)

BAV 2° tipo I, bloqueo de rama intermitente, no tengo claro si ya han podido correlacionar los sìntomas con el transtorno de conducción encontrado, con estos datos clínicos no indicaría pace ahora, descartando otras causas como cardiopatía isquémica, evaluaría muy bien las características de los síntomas, otra cosa sencilla de hacer es un MSC y también evaluaría función ventricular. Faltan datos, si los tienen que los manden, con lo que hay no pongo un pace por ahora

Saludos

Francisco Femenia

Hola Francisco y amigos del foro

Excelente ECG!
Creo que hay algo más que un Wenckebach 3:2. Y NO se trata de bloqueo de rama intermitente, ya que existe una secuencia fija: el primer latido conducido es angosto (más visible en la tira de ritmo y en aVF y aVR) mientras que el segundo es más ancho.
El bloqueo de rama intermitente en la mayoría de los casos requiere MP por severa enfermedad distal. Este NO parece ser el caso.
Lo que creo sucede en este caso es que el primer latido de la serie encuentra el nodo AV ya repolarizado (por la pausa previa) y el segundo latido, con PR más largo encuentra el período refractario funcional de la rama izquierda, el tercer latido se bloquea en el nodo AV, genera la pausa y permite que nodo AV y rama izquierda se recuperen, facilitando conducción normal. Y la secuencia vuelve a iniciarse.

La última secuencia de la tira es 4:3 donde el fenómeno se prolonga sobre un tercer latido conducido (para Nicolás y los demás estudiantes: fíjense cómo el RR se acorta, confirmando Wenckebach en el nodo AV).

SAludos
Dr Adrian Baranchuk, MD FACC

Hola a todos! Muy interesante ECG, la verdad que este foro es una verdadera biblioteca donde se puede ver de todo, y mejor tutelada, imposible!
Nunca había visto un Wenckebach de este tipo. Edgardo: ¿Sería posible ver el registro con un poco más de definición?
Muchas gracias Adrian por todos tus comentarios y por acordarte de los que recién nos iniciamos en este fascinante mundo! Se aprende mucho de cada comentario tuyo! Quizás sea mi falta de ojo o que veo la imagen muy pequeña por la definición de mi monitor, pero ¿me podrías aclarar en qué parte el RR se acorta? Aprecio bien la prolongación del PR hasta la aparición de una P no conducida y tu explicación del fenómeno (refractariedad primero de la rama izquierda y luego del nodo AV). Muchas gracias a todos!

Nicolás Bonantini. Estudiante de Medicina

Hola Nicolas

Como van esos exámenes?

El fenomeno de Wenckebach en el nodo AV se caracteriza por:

1. Prolongación progresiva y decremental del intervalo PR hasta que una onda P se bloquea
2. Acortamiento progresivo del intervalo RR con intervalo PP constante
3. El 90% de los bloqueos son de origen suprahisiano

4. El 90% de los casos son benignos y no requieren marcapasos
5. Extra bonus (no se encuentra en los libros de texto, o como diría el Ratón Ayala: ...En Europa no se consiguen...): el intervalo PR que inicia cada ciclo es constante (o fijo)

Una vez entendido esto, debierás preguntarte qué quiere decir progresivo y decremental. Para esto te voy a dar un ejemplo ficticio:
El 1er PR mide 120 ms (normal), el 2do PR mide 180 ms (normal, pero más largo), el 3er PR mide 220 ms (mas largo), el 4to PR mide 240 ms y el quinto se bloquea. Esto representa un Wenckebach 5:4 (hasta acá OK?).

Si te fijas bien, el PR cada vez se alarga más (eso explica el término progresivo), pero el delta de prolongación (es decir el porcentaje de prolongación cada vez es menor: en el ejemplo, ente el primer y segundo latido el PR se prolongo 60 ms, entre el 2do y el 3ro se prolongo 40 ms, y entre el tercero y el cuarto solo 20 ms). Este acortamiento del delta (o porcentaje de prolongación) explica el término decremental y a su vez es la razón del acortamiento de los RR, mientras que los PP se mantienen constantes.

En el ejemplo del foro, vos tenes al inicio un Wenckebach 3:2, esto quiere decir que habrá 2 intervalos RR en esa secuencia. Si te fijas, el segundo intervalo RR es más corto, confirmando Wenckebach.

Ojalá esté lo suficientemente claro, pero no dudes en preguntar más.

Ojalá haya muchos Nicolases!

Adrián Baranchuk

Adrian:
Tu explicación ha sido clara y concisa, y cada renglón fue respondiendo mi última pregunta... No quiero seguir para no sonar como un "lamebotas", solo elogiarte por tu espíritu docente y decirte que con cada comentario tuyo se aprende un poco más (al igual que con otros integrantes del foro como el Profe Riera, para no despertar celos..🫠).
Muchas gracias a todos los Profes del foro por aguantarnos, enseñarnos y dejarnos participar! Y gracias Adrian por el elogio, aunque no me considero un ejemplo, solo

un estudiante con vocación y ganas de aprender... Una vez más, muchas gracias a vos y a todos!!!

Nicolás Bonantini

My ECG diagnosis in this case is:

1) Acquired second-degree AV block (Mobitz type II?) with constant 2:1 rate. The criteria are: Each two P waves, one is blocked, the conducted P waves have a constant and normal PR interval, the first conducted P wave is followed by a narrow QRS complex (R=S in II: - 30 degree: LAFB? Minimal degree?) and the second one has complete intermittent LBBB pattern rate-dependent: tachycardia-dependent or in "phaseblock. It is impossible to determine if this is Mobitz type I or II by surface ECG. The changes in the rate of conduction may indicate if this is type I or II; or if in the change the PR interval remains constant, it will be type I. In terms of percentage, 35% are supra-Hisian (nodal), 15% Hisian and 50% infra-Hisian. The 2:1 block with narrow QRS complex, nearly always indicates supra-Hisian nodal location.
2) Complete LBBB pattern with strong suspected anterolateral ischemia: plus-minus T waves in V3 and symmetrical, deep and broad-base T waves from V4 to V6. Mainly in V4.

ACQUIRED CAUSES OF AV BLOCK

1) Idiopathic senescent AV block: Lev disease related to aging: it is an acquired heart block due to idiopathic fibrosis and calcification of the electrical conduction system of the heart. Lev's disease is most commonly seen in the elderly, and is often described as senile degeneration of the conduction system.
2) Coronary artery disease: Probably our case. Imperative Cardiac Catheterization with coronariography.
3) Postoperative or traumatic
4) AV node ablation
5) Irradiation of the chest
6) Infectious
7) Collagen vascular: systemic lupus erythematous, rheumatoid arthritis, scleroderma, dermatomyosistis, ankylosing spondylitis, polyarteritis nodosa, Marfand syndrome.
8) Infiltrative: sarcoidosis, amyloidosis, hemochromatosis, lymphomatous or solid tumor.

9) Neuromuscular: myotonic muscular dystrophy, peroneal muscular atrophy, scapuloperoneal syndrome, linb-gird dystrophy.
10) Drugs effects: digoxin, beta blockers, calcium antagonist, amiodarone, procainamide, class IC agents (propafenone, encainide, flecainide) taxol.

MY THERAPY APPROACH PROPOSSAL

1) Because there are clear ECG signs of ischemia, (in a senior man) we think that it is not necessary another non-invasive approach. Cardiac catheterization is first indicating, and if there are coronary significant lesions, I would consider stents or revascularization.

2) After this approach: EPS

3) Permanent pacing in acquired AV block class I

4) Recommendation for type II second degree AV block. Level of evidence B.(1)

TYPE AV BLOCK	PACING NECESSARY	PACING PROBABLY NECESSARY	PACING NOT NECESSARY
Third or Complete Heart Block (CHB)	**Symptomatic congenital CHB Acquired symptomatic CHB Acquired asymptomatic CHB**		
Second	**Symptomatic type I Symptomatic type II**	**Asymptomatic, type II at intra-His or infra-His level Hemodynamically symptomatic due to loss AV synchrony**	**Asymptomatic, type I, at supra-His (AV nodal) level. An exception may be in the elderly with asymptomatic type I AV block.**
First		**Hemodynamically symptomatic due to effective loss of AV synchrony with markedly prolonged PR interval (e.g.>300ms)**	**Asymptomatic**

1) Braunwald E, Zipes DP, Libby DIEASE A TEXTBOOK OF CARDIOVASCULAR MEDICINE. 6th edition pp777. 2001.

Addendum:

In reference to first etiology (Lev disease) unfortunately the literature is confused and wrong. Why? Because several authors put together Lev and Lenègre disease. They are absolutely different. Both entities, called **Progressive Cardiac Conduction Defects (PCCD),** are grouped unfortunately together as primary conduction diseases (Lev-Lenègre).

Both Lenègre disease —known as "primary" PCCD[1]— as well as the secondary mechanic lesion—sclerosis of the left "cardiac skeleton" or Lev disease[2]— usually cause LBBB or RBBB, frequently associated with divisional blocks.

Occasionally, they develop into more advanced degrees of block with a potential to cause SCD due to total AV block, to the extent that they represent the most important cause of pacemaker implantation in the first world: 0.15 per 1,000 inhabitants a year.

The same mutation in novel single SCN5A missense mutation can lead either to Brugada syndrome or to an PCCD. Modifier gene(s) may influence the phenotypic consequences of a SCN5A mutation. A G-to-T mutation at position 4372 was identified by direct sequencing and was predicted to change a glycine for an arginine (G1406R) between the DIII-S5 and DIII-S6 domain of the Na+ channel protein[3]**.**

DIFFERENCES BETWEEN LENÈGRE AND LEV DISEASE[4]

	LEV DISEASE	**LENÈGRE DISEASE**
Pathologic anatomy	Mechanical progressive fibrosis of the left "cardiac skeleton." Calcification of the mitral valve ring, fibrous central body, membranous part of the aorta base, apex muscular septum, and direct Hisian system and antero-superior fasciculus of the left branch.	Progressive sclerosis of the intraventricular His-Purkinje conduction system.
Etiology	Idiopathic. Mechanical acceleration of the aging process.	Allelic heterozygotic mutation with Brugada syndrome located in the alpha subunit of the sodium channel in the SCN5A gene.
Identified genetic defect	No	1) Substitution of the serine amino acid by

		glycine (G298S) in the domain of the I S5-S6 loop. 2) Substitution of asparagine by aspartic acid within the IV domain of S3 (D1595N). 3) Substitution of 514 cysteine by glycine (G514C). 4) Substitution of glycine by threonine in the 4372 position and glycine by arginine (G1405R) between the DIII-S5 domains of the sodium channel. 5) Others.

References

1) Lenègre J.The pathology of complete atrioventricular block. Progr Cardiovasc Dis 1964; 6:317-323
2) Lev M. Anatomic basis of atrioventricular block. Am J Med 196437:742.
3) Kyndt F, Probst V, Potet F, et. al.Novel SCN5A Mutation Leading Either to Isolated Cardiac Conduction Defect or Brugada Syndrome in a Large French Family. Circulation 2001; 104: 3081-3086.
4) Tan HL, Bink-Boelkens MT, Bezzina CR, et al. A sodium-channel mutation causes Isolated cardiac conduction disease. Nature 2001; 409:1043-104**7**

All the best for all
Andrés R. Pérez Riera