BRADIARRITMIA NOTURNA EM HOMEM ATLETA JOVEM RONCADOR

NOCTURNAL BRADYARRHYTHMIA IN SNORER YOUTH ATHLETE MAN

Case Report from Raimundo Barbosa Barros MD

Chief of coronary center Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza-Ceará-Brazil

Caso del Dr Raimundo Barbosa Barros Chefe do centro coronariano do Hospital de Messejana Dr. Carlos Alberto Studart Gomes Fortaleza- Ceará- Brasil.

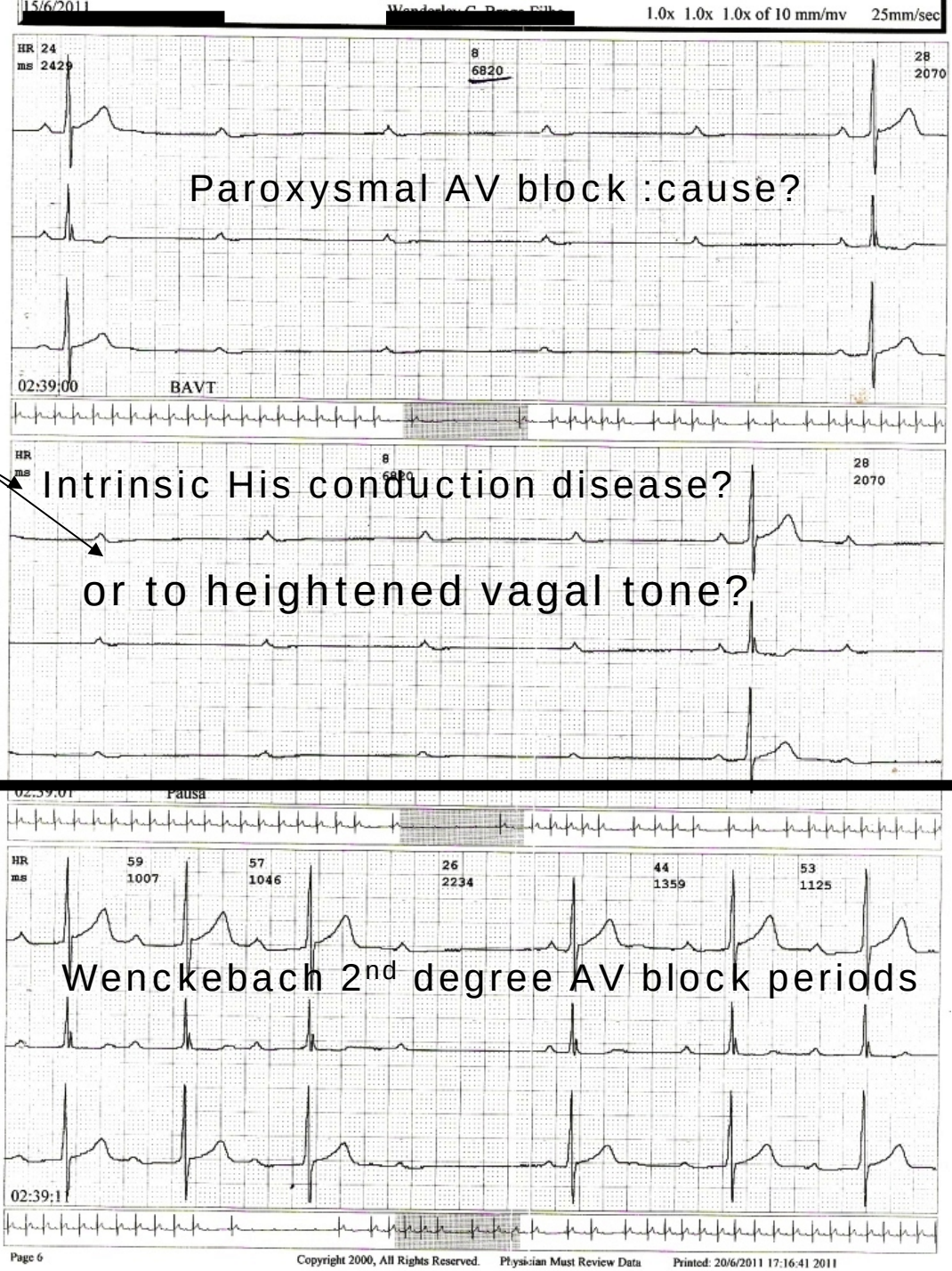
Conclusions and commentaries Andrés Ricardo Pérez-Riera MD PhD

Prezado Professor Andrés gostaria de ouvir a opinião dos integrantes do foro em relação este caso.

Trata-se de um atleta jovem, branco, (32 anos) homem com e queixa de ronco noturno. Sem antecedentes familiares de importância.
Ele veio a nosso serviço para realização uma avaliação pré-participativa com o intuito de correr uma mini-maratona.
Exame físico normal. índice de massa corpórea normal
ECG, ECO e Ergoespirometria(teste cardiopulmonar) normais.
No Holter esta alteração no dromotropismo durante o sono.
Qual o diagnóstico da arritmia? Qual a conduta apropriada?
O paciente está apavorado porque um outro colega indicou-lhe implante de marcapasso definitivo!!!!
Raimundo Barbosa MD

Dear Professor Andres I would like to hear opinions from the members of forum about this case.
It is a young Caucasian men athlete (32 years old) with complaints of nocturnal snoring consequence of deviated nasal septum with episodes of nasal obstruction and frequent use of decongestant. Negative family background. He came to our accomplishments to an pre-participatory evaluation in order running a mini-marathon.
Physical Examination: normal. Normal body mass index
ECG, ECO and cardiopulmonary exercise test were normal.
In this Holter(see next slide) we observe this dromotropic disorder during sleep.
What is the ECG diagnosis of this arrhtyhmia? What is the appropriate approach?
The patient is terrified because another colleague pointed to a definitive pacemaker!!
Raimundo Barbosa MD

Dramatic paroxysmal episode of high degree AV block with third degree AV block on the top rhythm strip:
Cause?
Paroxysmal AV block Cause?
Probably hypoxia secondary to sleep apnea syndrome (SAS), Obstructive Sleep Apnea Syndrome (OSAS) or Obstructive Sleep Apnea (OSA).
Intrinsic His conduction disease? Because narrow QRS (Very rare)



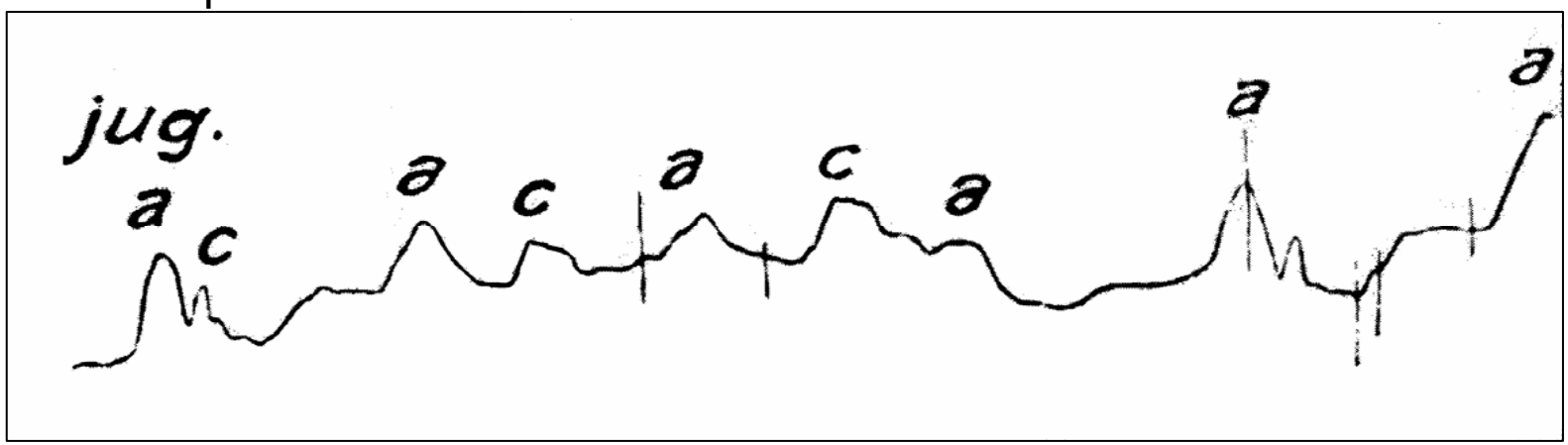
If the first beat of the Wenckebach cycle presents prolonged PR interval, it is considered that there is association with first degree AV block.

Diagnosis Mobitz type ICharacterized by cycles that present progressive prolongation of PR interval until a P wave appears, not followed by its corresponding QRS complex (blocked P wave). The cycle is called Wenckebach, which will always have one P wave more than the QRS complexes. The ratio is expressed, for instance: 4:3, which indicates that there are four P wave each three QRS complexes in each cycle.

The degree of PR interval prolongation is increasingly lower (decreasing increases), which explains the progressive shortening of RR intervals. This rule may not be found in cases of: sinus arrhythmia and non-homogeneous conductions in AV node and/or His fascicle.
The length of the Wenckebach cycles may not be uniform; i.e. vary in a rate: 4:3, 5:4 etc. Prolonged cycles indicate nodal location. If the first beat of the Wenckebach cycle presents prolonged PR interval, it is considered that there is association with first degree AV block. What is fantastic is that Wenckebach described this block before the discovery of the ECG, just by observing the veins of the neck.

History

In 1899, Karel Frederik Wenckebach published an article where he analyzed the irregular pulse, describing the difficulties of AV conduction with progressive widening in toads. Later, the Wenckebach type block (Mobitz type I) would be known as "Wenckebach phenomenon."



This is the original tracing of jugular venous pulse made by Wenckebach. Note the progressive widening of the a-c interval (corresponding to the PR interval) until the a wave is no longer followed by a c wave. The brilliant Wenckebach described the arrhythmia before the discovery of ECG!!![1]

1. **Pérez-Riera AR, Femenía F, McIntyre WF, Baranchuk A. Karel Frederick Wenckebach (1864-1940): A giant of medicine.Cardiol J. 2011;1:337-339.**

ATRIOVENTRICULAR BLOCKS

Concept: dromotropic disorders located at any level of the sino-ventricular conduction system by conduction "slowing" in the atria (PA interval), AV node (AH interval), His bundle and its branches (HV interval) or association of the former.

CLASSIFICATION CRITERIA OF ATRIOVENTRICULAR BLOCKS

I) BY DEGREE

(I-1)1st degree AV block;
(I-2) 2nd degree AV block:
a) Mobitz type I or Wenckebach
b) Mobitz type II
(I-3) Fixed 2:1 AV block;
(I-4) High-degree or advanced AV block;
(I-5) 3rd degree, complete or total AV block.

II) BY TOPOGRAPHY RELATED TO THE HIS BUNDLE

(II-A) Supra-hisian or pre-hisian.may extend PA and/or AH intervals: conduction slowing in the atria (PA) and/or AV node (AH).
(II-B) Hisian and infra-hisian.
IIB-1) Hisian: His bundle.
IIIB-2) Infra-hisian, fascicular or divisional: branches and divisions.
(II-C) Mixed: they affect the PA, AH and HV intervals.

POSSIBLE LOCATIONS OF AV BLOCK

	SUPRA-HISIAN OR PRE-HISIAN	HISIAN AND INFRA-HISIAN
LOCATION: **PERCENTAGE:**	AV node 75%	His bundle or divisions. 5% and 20%.
QRS DURATION: (QRSd)	Up to 100 ms.	Hisian narrow QRS. Infra-His QRSd 120 ms or more: branch block morphology
ELECTROGRAM	Prolongation of AH or PA intervals	Infrahisian: 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.

1. Supra-hisian or pre-hisian: 75%. associated to narrow QRS.
2. Hisian: 5%.
3. Infra-hisian, fascicular or divisional: 20%. 1 and 2 associated to wide QRS.

COLLEAGUES OPINIONS

Andres, These rhythm strips are of major concern. The dramatic episode of high grade AV block on the top rhythm strip is frightening although the ACC/AHA/HRS Guidelines for device implant discount marked pauses while asleep. I disagree and have had patients with classic intermittent marked sinus node dysfunction or AV block while awake (hence, clearly needed a pacemaker) and following implantation of the pacemaker, reported that they woke feeling more rested. Their intermittent AV block that also occurred during the night was also effectively treated facilitating their getting a better rest. Indeed there is also a sinus bradycardia accompanying the high grade AV block favoring a diagnosis of increased vagal or parasympathetic tone but I really would have expected the sinus node slowing to be equally marked along with the AV block if the vagal input to the heart was balanced. If there is no reversible cause, I would not argue with someone who wanted to implant a permanent dual chamber pacemaker however, I would first look for Obstructive Sleep Apnea (OSA) with a formal sleep study given the history of snoring. If this is demonstrated, I would try this relatively young man on a CPAP unit either in the sleep lab or at least with another Holter Monitor while he is wearing the CPAP unit. IF he has OSA and these frightening looking rhythms resolve with the use of the CPAP unit, I would just do that UNLESS either the CPAP doesn't work for him or for a variety of reasons, he doesn't tolerate it and hence, even if it were beneficial, he does not use it. Then I would protect these profound periods of asystole with a permanent pacemaker. Since the basic manifestation is AV block, I would program a low rate and allow for P wave synchronous ventricular pacing. There are a variety of surgical techniques to treat OSA but I am not familiar with those – still that is something to consider if he has OSA and refuses to use a CPAP unit. If Dr. Barbosa Barros elects to proceed with a surgical intervention (presuming a diagnosis of OSA is made), I would try to hold off on implanting a permanent pacemaker until the surgery is completed for OSA and it is determined that it is working or not working.

Since he is described as being athletic, that would also increase his vagal tone. There have been some interesting studies over the years. In superbly fit athletes, either professional athletes or amateur athletes who are in the Olympics, there is marked resting sinus bradycardia, junctional rhythms and even Wenkebach 2nd degree AV block. When the Olympic games are over and the athletes back-off on their rigorous exercise, they become a little deconditioned and the slow heart rates and low grade 2nd degree AV block resolves (it makes you wonder what really is normal).

1. Meytes I, Kaplinsky E, Yahini JH, Hanne-Paparo N, Neufeld HN, Wenckebach AV block: a frequent feature following heavy physical training, Amer Heart J 1975; 90: 426-430
2. Zaman L, Moleiro F, Rozanski JJ, et al, Multiple electrophysiologic manifestations and clinical implications of vagally mediated AV block, Amer Heart J 1983; 106: 92-99
3. Kinoshita S, Konishi G, Atrioventricular Wenckebach periodicity in athletes: influence of increased vagal tone on the occurrence of atypical periods, J Electrocardiol1987; 20: 272-279.

While neurocardiogenic syncope is associated with either a cardioinhibitory component (both sinus arrest and AV block if one paces the atrium) and vasodepression (vasodilation), there is a subgroup of neurocardiogenic syncope patients who experience syncope in association with physical exercise, usually just when they stop and this is commonly AV block with minimal if any sinus node dysfunction or slowing.

1. Talwar KK, Edvardsson N, Varauskas E, Paroxysmal vagally mediated AV block with recurrent syncope, Clin Cardiol 1985; 8: 337-340
2. Hirata T, Yano K, Okui T, Mitsuoka T, Hashiba K, Case Report: asystole with syncope following strenuous exercise in a man without organic heart disease, J Electrocardiol 1987; 20: 280-283
3. Oswswald S, Brooks R, O'Nunian SS, Curwin JH, Roelke M, Radvany P, Ruskin JM, McGovern BA, Asystole after exercise in healthy persons, Ann Int Med 1994; 120: 1008-1011
4. Sakaguchi S, Shultz JJ, Remole SC, Adler SW, Lurie KG, Benditt DG, Syncope associated with exercise, a manifestation of neutrally mediated syncope, Am J Cardiol 1995; 75: 476-481

Based on the second rhythm strip, the AV block appears to be Wenckebach 2nd degree AV block. The level of block is probably within the AV junction but with the increased vagal tone (we do not know what was happening while the first rhythm was recorded - was this during a profound apneic episode?). There is also a preferential distribution of the parasympathetic innervations to the heart. Coming from the right side, there is a predilection to the SA node. Coming from the left side involves a predilection to impact the AV junction. He probably has preferential vagal stimulation via the left vagus nerve.
I await your input and that of other members of this conference. I will go out on a limb and attribute the intermittent AV block and sinus node slowing while sleeping to OSA and suspect that it might be "cured" with a CPAP unit to treat the OSA.

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Classic vagotonic block as described by Massie and myself many years ago[1]. Note the slowing of the sinus rate as block ensues. In our series in most particularly the young athletes this finding was benign. Although several did require pacemakers.Would be inclined to wait and see if he becomes symptomatic. Note a very recent report in JACC from Brignole et al [2] on paroxysmal AV block as a cause of syncope without concomitant sinus slowing. I have seen pts who may show a vagal response with predominant effect on the node.

Clássico bloqueio vagontónico como descrevera Massie[1] y eu mesmo muito anos atrás. Observe a súbita desaceleração da taxa sinusal subseqüente manifestada como um bloqueio seguinte. Em nossa série, e mais particularmente em jovens atletas este achado foi benigno, embora vários exigiram implante de marcapasso. Estaria inclinado a esperar e ver se ele se torna sintomático. Note um caso reportado muito recentemente no JACC por Brignole e col2 sobre el bloqueio AV como causa de síncope sem concomitante alentecimento sinusal. Eu tenho visto pacientes que podem mostrar uma resposta vagal com efeito predominante sobre o Nó SA.

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1. **Massie B, Scheinman MM, Peters R, et al. Clinical and electrophysiologic findings in patients with paroxysmal slowing of the sinus rate and apparent Mobitz type II atrioventricular block.**Circulation**. 1978 Aug;58:305-314.**
2. **Brignole M, Deharo JC, De Roy L, et al. Syncope Due to Idiopathic Paroxysmal Atrioventricular Block Long-Term Follow-Up of a Distinct Form of Atrioventricular Block.J Am Coll Cardiol. 2011 Apr 29. [Epub ahead of print]**

Dear Andres,

In this asymptomatic patient I will favor supra nodal AV block. There is a clear slowing of the sinus rhythm, the QRS are not wide, the J point elevation is slight and the morphology of the P wave is normal. An exercise test could be done. I will investigate whether bradyarrhythmias parallel breathing episodes.

No pace maker in this case.

Kind regards,

Prezado Andrés Em este paciente assintomático penso que se trata de um bloqueio AV supra-nodal. Há claro alentecimiento do ritmo sinusal, os QRS não são largos, o ponto J está levemente elevado e a morfologia das ondas P são normais.
Uma prova de esforço poderia estar indicada. Investigaria se as bradiarritmias são concomitantes a os episódios pausa respiratória.

Não indicaria marcapasso em este caso.

Professor Philippe Chevalier MD PhD Département de Statistique des Hospices Civils de Lyon, **Lion France.**

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To our dear forums's friends '; my porpose is to discuss the interesting case from our dear friend Dr Raimundo Barbosa Barros This a seldom case of a young athletic male with a paroxysmal complete bradycardic A-V block during sleeping

During sleeping could be recorded 3 different types of bradycatdic block

!) Variable P-R segment , thise pattern is a londitudinal dissociation of the upper A-V node This dissociation is due o a vagal effect , in patient with , most probably , with a mutation in one of a miriad of receptors or a structural anomaly in the very complicate tissue, .working as an electrical transformer

2) Atipical Wenchebach (let me remaind the 5 electrocardiographic characteristics of typical Wenchebach A) progressive elongation of P-R segment B) progressive shortening of R-R, C) The first P-R after the drop beat must be normal , D)The R-P segment is progressivly shorten

E) the sinus drop beat is only one) This typical Wenchebach is seen ,mostly in the metabolic reperfused inferior wall infarction (as we have discribed in AHJ 1987)

Every second degree A-V block witch doesn't fulfill the 5 criteria is an atypical Wanchebach with different electrophisiology meaning from the typical one

3) Complete bradycardic block , like the case presented by Raimundo. This pattern is a seldom one

Why ? Because the sinus node is more sensitive to te vagal effect than the upperA-V node , More frecuent is to record in Holter prolong sinus arrest , sometime until 15 msec

The Raimundo's case is a physilogical anomaly, in witch the upper A-V node is more sensitive to the vagal effect than sinus onel recommend to introduce a ventricular pacemaker with 45 bpm , Why? because no medical treatment is avaible , and it is known that there are accumulating small brain demage , witch can be manifested in the elderly

Diffential diagnosis ; there are another paroxysmal block with narrow QRS , but very danger and life threteaning THE HIS , IMFRANODAL BLOCK

The differnces between the two enteties are ; the infranodal block appear in generally with sinus tachycardia , in every time of the day and motly in postmenopausal female , and is the most frecuent cause of syncope in postmenopausal female with persistent first degree A-V block . and prolong HV , Sometime we see this type of block and syncope in female with P-R 18- 200 msec. In this cases emmergency pacemaker is mandatory and life saving

In the Raimundo's case the paroxysmal block is seen with sinus bradycardia , atypical Wenchebach , and logitudinal dissociation , only during night , mostly in young male 18-45 years old, no syncope episodes , pacemaker is not mandatory a not life saving

My kindly regard

Samuel Sclarovsky

Spanish Dr Samuel Sclarovsky

Queridos amigos del forum analizaré el caso de nuestro querido amigo Dr Raimundo Barbosa Barros Este es un caso raro de bloqueo paroxístico del nódulo AV(NAV) ocurrido durante el sueño. El mismo habitualmente se observa durante la noche y existen varios tipos:

1)Con intervalos PR de longitud diferentes por disociación longitudinal del NSA por efecto vagal

2)Bloqueo de segundo grado con Wenkebach atípico

3)Bloqueo paroximal completo (muy raro)

Los patrones 1 y 2 son muy frecuentes en jovenes durante el sueño y se deben a un hipertono vagal El efecto vagal nocturno se manifiesta en el NAV y en la parte superior del NAV.

El NAV es el mas sensible al este efecto , por lo tanto vemos asístoles muy prolongadas que pueden extenderse hasta 15 milisegundos.

En el Holter pueden ocurrir varios episodios durante la noche .,Este fenómeno no acarrea peligro de vida pero ocasiona microlesiones cerebrales acumulativas. que pueden llegar a ser significativa en la vida adulta (este fenomeno habitualmente se observa en varones entre entre 18 a 45 años)

El caso presentado es raro porque indica que el Nódulo AV es mas sensible al efecto vagal que el NSA, y esto es una desviación fisiológica, Tambien en este caso se observa un Wenkebach atipico, que es el mas frecuente en estos casos El período de Wenkebach tipico tiene 5 condiciones obligatorias 1) prolongación progresiva del intervalo PR 2) acortamiento progresivo del RR 3) intervalo PR póst P bloqueda normal 4) existencia de una sola onda P bloqueada 5) intervalo RP con acortamiento progresivo.

TODO BLOQUEO QUE NO CUMPLE ESTAS 5 CONDICIONES ES ATIPICO Y SE DEBE EXPLICAR PORQUE ES ATIPICO

Por ejemplo en este caso indica que hay una disociación longitudal dentro del Nódulo AV por la existencia de 2 vias con velocidad conduccion y periodos refractarios diferentes acentuado por el efecto vagal. La anatomia ,fisiologia y la cantidad de receptores ,y moleculas que hace a este tansformador electrico asombroso y por supuesto como ocurre en la biologia y todo tejido complicado ven muhas mutaciones de todo tipo a difererencia del sistema de conduccion intraventricular , que son simples cables

El diagnóstico diferencial de este bloqueo completo paroxístico es de crítica importancia. El otro bloqueo paroxístico con QRS angostos son de muy alto riesgo y es el bloqueo paroximal del haz de HIS, que tiene patrón electrocardiografico En este caso , yo aconsejo un marcapaso ventricular en 45latigos por minuto convencer a este joven atleta que esto no lo va impedir a intervenir en triaton , dedicarde a toda actividad profesional y cortejar a toda bella dama , que en Brasil hay una acumulacion extraordinaria de este genero

Disculpen companieros por la larga discusion de un cardiologo jovato que vio mucho pacientes en 50 años de profesión y es un lector persistente de la literatura cardiológica

Un fraternal abrazo a todos nuestros queridos amigos del forum

Samuel Sclarovsy

Mi opinion
Bloqueo AV de 2do grado con periodicidad Wenchebach y bloqueo SA 3er grado completo paroxÍstico nocturno que en contexto de antecedentes de ser atleta y roncador puede interpretarse como hipertono vagal asociado a posible sindrome de apnea del sueño (SAS).

Conducta: Solicitar polisomnografia para confirmar diagnóstico de SAS que puede ser la causa de alteraciones dromotrópicas.

Pruebas de disautonomia para confirmar hipertonia vagal por corazón de atleta donde pueden presentarse estas alteraciones de la conduccion Ergometria

Con estas pruebas recien establecer una conducta racional que en principio a mi parecer no es eléctrica (marcapaso) y no alarmar inutilmente ni angustiar a este joven atleta

Un abrazo

Juan Sirena

Santiago del Estero-Argentina

Estimado Maestro Perez Riera y Dr Barbosa:
En las tiras de Holter enviadas evidencia un bloqueo de segundo grado tipo I (o Wenckebach). En deportistas entrenados suelen ser un hallazgo y no denota enfermedad estructural sino funcional, predominio del tono vagal que se exacerba en el descanso, con pronóstico benigno. En el primer trazado presenta BAV completo transitorio, con una pausa de mas de 6 seg. La história refiere se tratar de un paciente roncador. No indicaria marcapasos, solicitaria una polisomnografia. me encantaria que el amigo Adrian nos ilumine con sus conocimientos en este tema. Interpeto las pausas provocadas por hipoxemia asociadas con apneas del sueño. Estudio: polisomnografia. Si son desaturaciones de acuerdo al resultado de la polisomnografia se podria indicar VPAP, o corrección quirúrgica si presenta anomalias estructurales otorrinológicas, de acuerdo a los resultados de la misma.
Me pregunto porque se le solicitó el estudio Holter?? no presentaba en lo descrito criterios para su realización.
Saludos y me encantaria oir la opinion del profesor Adrian un experto en esta materia.
Martin Ibarrola

Rev Esp Cardiol. 1998;51:498-501. - Vol.51 no. 6

Bloqueo auriculoventricular avanzado de presentación atípica tras infarto agudo de miocardio en el síndrome de apnea del sueño Francisco José Morales Poncea, Francisco López Pardoa, María del Mar Méndez Corteganoa, Adrián Revello Bustosa, Gonzalo Barón Esquiviasa, Alonso Pedrote Martínezaa, Francisco Errazquin Sáenz de Tejadaa and José Burgos Cornejoa

http://www.revespcardiol.org/sites/default/files/elsevier/pdf/25/C510613.PDF

Apnea del sueño y arritmias cardíacas: asociación y mecanismos involucrados. Adrian Baranchuk, Christopher S. Simpson, Damian P. Redfearn, Kevin Michael, Mike Fitzpatrickhttp://www.electrofisiologia.org.ar/joomla/volumen-i-numero-i/apnea-del-sueno-y-arritmias-cardiacas

http://www.nlm.nih.gov/medlineplus/spanish/ency/article/000811.htm

FINAL COMMENTS

Many nocturnal cardiac arrhythmias and conduction defects have been reported in the adult sleep apnea syndrome (SAS), Obstructive Sleep Apnea Syndrome (OSAS) or Obstructive sleep apnea (OSA). **The most original is the great variability of the heart rate which is cyclical and related to the apneic episodes, and easily differentiated from simple respiratory sinus arrhythmia. It is characterized by an initial bradycardia followed by rebound tachycardia.**

The bradycardia is vagally dependent (inhibited by atropine) probably secondary to carotid chemoreceptor stimulation by the hypoxemia. The tachycardia is mainly attributed to the cessation of vagal hypertonicity although catecholamine stimulation has been suggested.

The origin of these changes is purely functional, regressing with treatment of apnea (waking, tracheotomy), the maintenance of arterial oxygen concentrations with oxygen therapy and parasympathetic blockade (atropine).

The intensity of the phenomenon is related to the degree of arterial desaturation, which is itself related to basal arterial saturation (SaO_2) and the duration of the apneas. Prolonged asystole due to paroxysmal SA or AV block may be observed at night in these patients**. The influence of vagal overactivity is confirmed (suppression of vagotomy) with no organic pathology (diurnal absence, tracheotomy, normal electrophysiological testing) in favour of a relationship with apnea. Though less common than conduction abnormalities, atrial arrhythmias (extrasystoles, flutter, fibrillation) are also possible complications of SAS. The absence of an organic substrate is indicated by their regression post-tracheotomy and the efficacy of atropine (again in favour of a vagally-induced mechanism). Finally, nocturnal ventricular hyper-excitabilty is sometimes observed, the probable mechanism being the association of severe hypoxemias (SaO2 < 60%) and the increased sympathetic tone at the end of the apnea.** OSA) is a common medical condition that occurs in approximately 5% to 15% of the population. The pathophysiology of OSA is characterized by repetitive occlusions of the posterior pharynx during sleep that obstruct the airway,

followed by oxyhemoglobin desaturation, persistent inspiratory efforts against the occluded airway, and termination by arousal from sleep.

Obstructive sleep apnea syndrome (OSAS) refers to recurring episodes of upper respiratory track obstruction and frequent decreases in arterial oxygen saturation due to repetitive occlusions of the posterior pharynx during sleep. Its prevalence in adult population is 4% in men and 2% in women. The most important causes of morbidity and mortality in affected patients are traffic accidents and cardiovascular complications including systemic arterial hypertension, coronary artery disease, congestive heart failure, and cardiac arrhythmias. Pulmonary hypertension may be associated with OSA, especially in patients with preexisting pulmonary disease. The initial phases of apnea are associated with a transient increase in the parasympathetic activity resulting in bradyarrhythmias, followed by tachycardias due to increased sympathetic activity and arousal after the end of apnea episodes. The most frequent arrhythmia in OSAS is cyclic variation of heart rate. Most of the arrhythmias seen in OSAS are secondary to OSAS and disappear with OSAS treatment, without any electrophysiological conduction system abnormalities[1].

OSA is associated with daytime sleepiness and fatigue, likely due to fragmented sleep from recurrent arousals. Substantial evidence shows that patients with OSA have an increased incidence of hypertension compared with individuals without OSA and that OSA is a risk factor for the development of hypertension.

Although the exact cause that links OSA with cardiovascular disease is unknown, there is evidence that OSA is associated with a group of proinflammatory and prothrombotic factors that have been identified to be important in the development of atherosclerosis. OSA is associated with increased daytime and nocturnal sympathetic activity.

1. **Bayram NA, Diker EObstructive sleep apnea syndrome and cardiac arrhythmias. Turk Kardiyol Dern Ars. 2008 Jan;36:44-50.**

Autonomic abnormalities seen in patients with OSA include increased resting heart rate, decreased R-R interval variability, and increased blood pressure variability. Both atherosclerosis and OSA are associated with endothelial dysfunction, increased C-reactive protein, interleukin 6, fibrinogen, and plasminogen activator inhibitor, and reduced fibrinolytic activity. OSA has been associated with enhanced platelet activity and aggregation. Leukocyte adhesion and accumulation on endothelial cells are common in both OSA and atherosclerosis. Clinicians should be aware that OSA may be a risk factor for the development of cardiovascular disease[1].

QT dispersion (QT(d)) measures the variability of the ventricular recovery time. QT(d) may identify patients at risk for ventricular arrhythmias and sudden cardiac death (SCD). Patients with OSAS and no structural heart disease have a higher QT(d)/QT(c)(d) compared to an overtly healthy patient population, possibly serving as a marker for an increased risk of SCD[2].

Cardiac resynchronization therapy (CRT) reduces the severity of sleep apnea. Major effects are seen in patients with Central sleep apnea. The presence of sleep apnea may be an additional consideration when deciding on which HF patients will receive CRT[3]. Older age and moderate-severe OSA are predictors of interatrial block. P-wave dispersion is increased in patients with moderate-severe OSA. This may partly explain the high prevalence of atrial arrhythmias in patients with OSA[4].

OSA may increase the risk of stroke in AF patients: refining the CHADS2 score[5].

1. Parish JM, Somers VK.Obstructive sleep apnea and cardiovascular disease. Mayo Clin Proc. 2004 Aug;79:1036-46.
2. Voigt L, Haq SA, Mitre CA, et al. Effect of obstructive sleep apnea on QT dispersion: a potential mechanism of sudden cardiac death. Cardiology. 2011;118:68-73.
3. Lamba J, Baranchuk A. Cardiac resynchronization therapy for the treatment of sleep apnoea: a meta-analysis. Europace. 2011 May 11. [Epub ahead of print]
4. Baranchuk A, et al. Interatrial block in patients with obstructive sleep apnea. Cardiol J. 2011;18:171-175.
5. Yazdan-Ashoori P, Baranchuk A. Obstructive sleep apnea may increase the risk of stroke in AF patients: refining the CHADS2 score. Int J Cardiol. 2011 Jan 21;146:131-133.