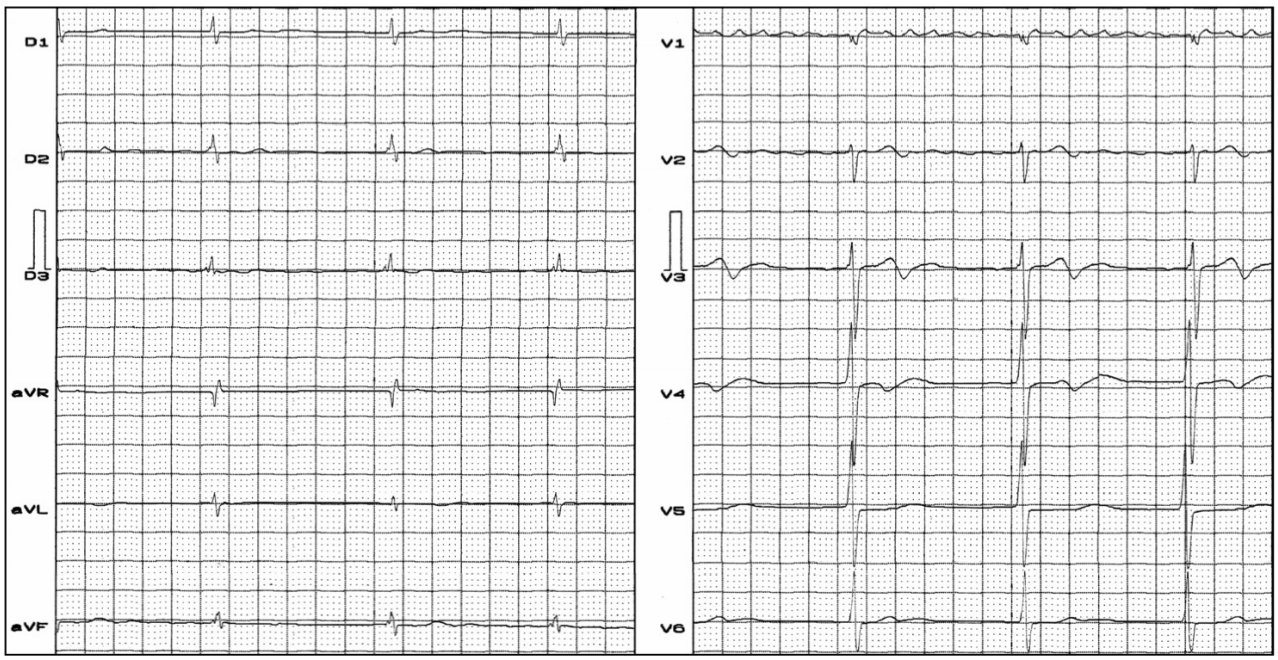
Old man with angina – 2009

Dr. Andrés R. Pérez Riera

Dearest friends which is the ECG diagnosis?
¿Queridos amigos cuál es el diagnóstico de este ECG?



Clinical Data: 84 years old man diagnosed with unstable angina
Which is the ECG diagnosis?

OPINIONES DE COLEGAS

Dear Potro

Thanks for this beautiful case.

My interpretation:
1. Junctional rhythm
2. Long QT with big U-waves
3. Flat T-waves with +/- component

ID: Electrolyte Disorder: Hypokalemia (with possible Hypomagnesemia)

Happy New Year!

Adrian Baranchuk

This is a fascinating ECG. I have a different approach, whether presented with a 12 lead ECG as in this case or a simple (or complex) rhythm strip. In addition to arriving at a provisional diagnosis which is a fun intellectual exercise, I try to take this to the next step and decide on what I would do for the patient, whether this is simply the added tests needed to establish a diagnosis or perhaps, initiate therapy.

I have seen this phenomenon on a repeated basis but more than 20 years ago when I was active as a general cardiologist. I presented an abstract to theof Chest Physicians in 1989 but never got around to writing it up for formal publication. The reference is:

Levine P.A., Klein, M.D., *Bradycardia exacerbated angina, therapy with a combination of drugs and pacing, Chest 1989, 134S, Presented at the XVI World Congress on Diseases*

of the Chest and 55th Annual Scientific Assembly, American College of Chest Physicians, Nov 1, 1989; Boston, MA.

If I can locate it, I will send it separately since I will not start looking until I complete this response.

The underlying rhythm is atrial fibrillation. There is a very slow ventricular response.

There are only 4 cycles that can be measured and these are not absolutely the same although the variation is minimal.

There is at least high grade 2nd degree AV block although it cannot be characterized as either Mobitz I or Mobitz II in light of the underlying atrial fibrillation. This is most likely 3rd degree AV block in the presence of chronic atrial fibrillation. Whether the focus is a junctional escape rhythm or, based on the narrow QRS morphology, a fascicular escape rhythm, I cannot say without a baseline ECG clearly showing either sinus with intact AV nodal conduction or atrial fibrillation with a more rapid and more irregular ventricular response to indicate that it is clearly conducted from the atria.

As to QRS morphology, there is low voltage in the limb leads although not in the precordial leads. There is an initial "q" wave in Lead V1 and an absence of the expected normal small q in the lateral leads (D1, aVL, V5-6) raising concerns about a prior septal MI however if the ventricular escape focus is arising from one of the ventricular fascicles and not the AV node, any specific diagnosis would be suspect.

There is terminal T wave inversion in leads V2-V4 consistent with anterior wall ischemia. There is also a prominent U wave in the anterior precordial leads which is consistent with the marked bradycardia although I cannot rule out electrolyte abnormalities or other causes of U waves.

Now to return to my earlier statement and the referenced abstract. I cared for a series of patients when I was atwho presented with the following scenario.

Unstable angina, an increase in beta blockade or calcium channel blocking therapy was associated with a significant slowing of the heart rate which caused anxiety on the part of the physicians caring for the patient and was associated with a worsening of the angina. The medications (either a calcium channel blocker and/or a beta blocker) were discontinued, the heart rate increased and at faster heart rates the patient also experienced angina.

We finally implanted a pacemaker (usually a dual chamber although in a patient such as the subject of this challenge, I would have chosen a single chamber ventricular pacemaker) to prevent the rate from getting too slow while allowing us to increase the beta blocker and/or calcium channel blocker with a marked clinical improvement.

My patients had one other factor directing our approach. The patients were not candidates for coronary artery bypass surgery because of the extent of the coronary artery disease based on coronary angiography or their general medical status condition precluded major

open heart surgery. Please remember that this was more than 20 years ago and some of those patients might be a candidate for surgery or another intervention today.

In the management of ischemic heart disease we routinely use medications to slow the ventricular rate in an effort to reduce the metabolic demand thus restoring a balance between myocardial blood supply and myocardial oxygen demand. In my patients and this case, the presenting heart rate was too slow and although the metabolic demand was reduced, the ischemia actually increased again due to an imbalance between supply and demand.

The hypothesis was based on the fact that the majority of coronary blood flow occurs during diastole. In a patient with high grade obstructive lesions in the coronary arteries (atin the late 1980's we had started doing coronary artery angioplasty.

As part of our own learning curve, we advanced very thin catheters into the coronary arteries and measured the gradient across the obstructing plaque.)

The gradient across a stenotic lesion was commonly > 50 mm Hg and not uncommonly above 100 mm Hg. With a long diastolic filling period associated with a marked bradycardia, the diastolic pressure would fall below the required perfusion pressure and the segment of the myocardium distal to the obstructing lesion would have no perfusion during systole and again during late diastole creating a mismatch between supply and demand. As such, even with a slow rate, the net effect was an increase in ischemia and anginal symptoms.

By increasing the rate using pacing therapy, I was able to improve myocardial perfusion while not further increasing metabolic demand.

I have tried to diagram this but would ask your indulgence for my hand-drawn diagram. Knowing that cyberspace sometimes markedly distorts diagrams, both a word and pdf file of the same diagram is attached.

There is also one additional consideration. When treating atrial fibrillation, I used to try to push for rates similar to a normal resting sinus rate.

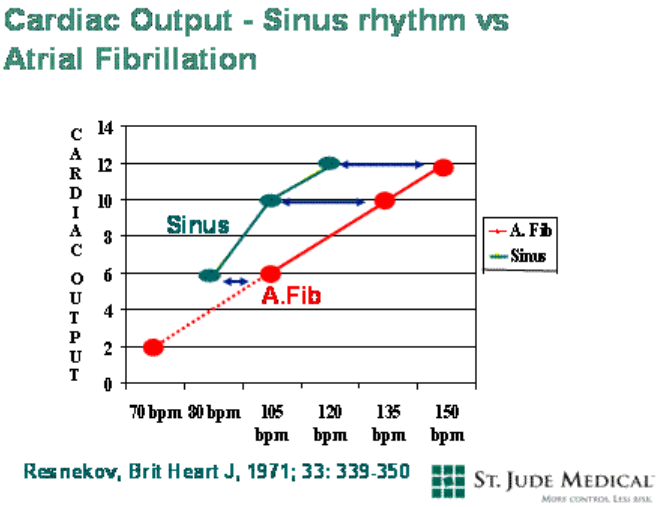
When invited to give a presentation on the limitations of the Automatic Mode Switch algorithm at Dr. Santini's conference inin 1996, I discovered a very interesting phenomenon that has changed the way that I practice with respect to controlling the ventricular response to AFib.

I now aim to have a resting ventricular rate around 80-90 bpm.

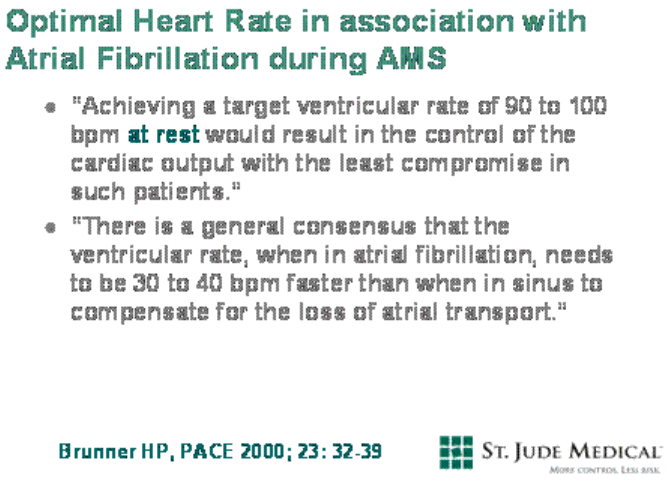
For that presentation, I extracted the data presented in the paper by Resnekov and McDonald (1971) and reformatted it as sinus rhythm vs atrial fibrillation with respect to cardiac output.

For similar cardiac outputs, the sinus rate is, on average 20 - 40 bpm slower than the rate in atrial fibrillation. The extrapolation to what I had always considered the desired resting rate in a patient with chronic atrial fibrillation actually resulted in a low cardiac output state which doesn't help coronary perfusion or perfusion of the other organs in the body.

The patient whose ECG was circulated has an even slower rate so I suspect a marked decrease in cardiac output and a serious limitation to coronary perfusion.

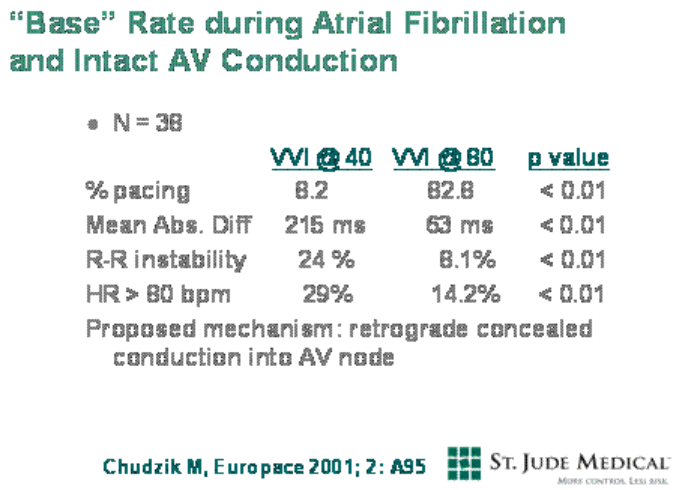


The benefit of a higher (paced) rate during atrial fibrillation was subsequently shown by Brunner and colleagues utilizing a metabolic stress test. These studies were performed in patients with a DDD pacemaker during automatic mode switch in the presence of atrial fibrillation.

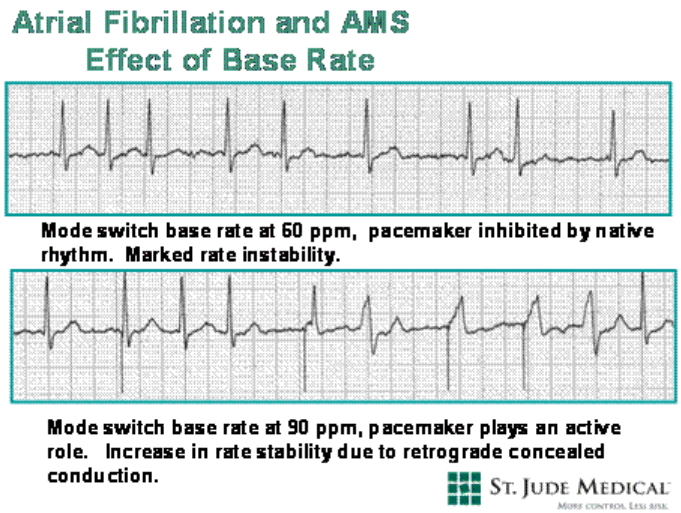


When pacing is involved, maintaining a higher base rate in the setting of a poorly controlled ventricular response also helps to stabilize the ventricular rate by the mechanism of concealed retrograde conduction into the AV node.

Below is the data presented by Chudzik and colleagues at Europace in 2001.



When I attempted a higher base rate in my patients with atrial fibrillation who basically had intact AV nodal conduction, the following is an example:



To return to the 84 year old man with unstable angina, he has atrial fibrillation with a very slow ventricular response, possibly complete heart block. I propose that the very slow ventricular response is contributing to his unstable angina and based on the ECG, there is, at least, anterior wall ischemia.

While the preferred management would be revascularization, he might still require permanent pacing. If he is not a candidate for revascularization, pacing may be of additional benefit and if the lead is placed in the RVOT, there should be minimal distortion of the paced QRS complex.

I have found the abstract that I discussed below with respect to this case. It is attached.

BRADYCARDIA-EXACCERBATED ANGINA, THERAPY WITH A COMBINATION OF DRUGS AND PACING

P.A.Levine,MD, FCCP*, M.D.Klein, MD, FCCP
University Hospital, Boston, MA, USA

Usual antianginal therapy may be limited in some patients due to induction of symptomatic bradycardia. Despite this slow rate, episodes of angina may continue or worsen. A series of 23 pts who were not candidates for revascularization yet could not tolerate either beta or calcium channel blocking agents were treated with temp pacing to enable medications to be given. 16 (70%) demonstrated a dramatic reduction in frequency and severity of angina. A permanent pacemaker was implanted. Mean follow-up is 19 months (range 4 - 35) with continued good anginal control in 9, 3 have increasing angina despite initial good results and 3 died due to MI and one due to CHF. Two theories are offered: The marked bradycardia allows the coronary perfusion pressure (CPP) to fall below the critical CPP increasing ischemia out-of-proportion to the decreased metabolic demand engendered by the slower HR. The slow HR increases wall stress by increasing end diastolic volume. Pacing prevents this inappropriate rate slowing while allowing the safe administration of meds.

SPONTANEOUS PNEUMOTHORAX IN PNEUMOCYSTIS CARINII PNEUMONIA.

S. GARAY,* FCCP, J. LOWY, FCCP, D. KAMELHAR, FCCP. New York University Medical Center, N.Y.

Spontaneous pneumothorax is a recently documented manifestation of Pneumocystis carinii pneumonia (PCP). Although it is unusual, it is being seen with increasing frequency because of the large numbers of AIDS patients with PCP. We have diagnosed 18 patients with this manifestation. All patients presented with fever, cough and dyspnea. Initial CXR findings revealed unilateral pneumothorax in 14 and bilateral pneumothoraces in 4. All had bronchoscopy proven PCP. Resolution of the pneumothoraces was variable. Nine patients re-expanded either spontaneously (3) or following chest tube insertion (6). Nine patients had persistent air leak. Sclerosis therapy was attempted in all 9, but was successful in only 2. Seven patients died with persistent air leak. Conclusion: Spontaneous pneumothorax in PCP may present a more complicated course with significant difficulties in re-expansion.

AIDS I (S)
Wednesday, November 1, 8:30 - 10:00 AM

INHALED VERSUS INTRAVENOUS PENTAMIDINE (PENT) FOR THE TREATMENT OF PNEUMOCYSTIS CARINII PNEUMONIA (PCP) IN AIDS.

GW Soo Hoo,* Z Mohsenifar, RD Meyer. Cedars-Sinai Medical Center; Los Angeles, California.

We prospectively compared the efficacy of inhaled (IH)(8mg/kg) to intravenous (IV) PENT (4mg/kg) for the treatment of PCP. Gay males (n=17) with mild to moderate PCP were randomly assigned to receive 21 days of daily IV (n=9) or IH (n=8) PENT delivered with a Respirgard-II nebulizer. The two groups were comparable as outlined below:

Group	PaO_2	$P(A-a)O_2$	LDH
IV (n=9)	75±17 mmHg	36±22 mmHg	315±113 U/L
IH (n=8)	68±13 mmHg	39±18 mmHg	438±234 U/L

(mean ± SD; $p>0.05$ for all)

All patients in the IV group improved but 5/9 (56%) experienced dysglycemia and hypotension, 4/9 (44%) hematologic toxicity and 1/9 (11%) rash; 4 patients in the IH group (50%) responded to therapy ($p<0.05$ compared with IV). The remaining 4 patients deteriorated on IH therapy and were switched to IV trimethoprim-sulfamethoxazole or PENT and two eventually died. This 25% mortality rate, although not significant, occurred in the 2 patients with the highest LDH or greatest $P(A-a)O_2$ difference. We conclude that IH PENT deserves further evaluation in mild PCP, and may not be indicated in patients with moderate to severe disease.

PNEUMOCYSTIS CARINII BINDS TO THE CELL BINDING DOMAIN OF FIBRONECTIN.

ST Pottratz* and WJ Martin II FCCP. Indiana University, Indianapolis IN.

Pneumocystis carinii (PC) pneumonia is a major cause of morbidity and mortality in immunocompromised hosts. Attachment of PC to alveolar epithelial cells is a prerequisite to the development of pneumonia. The mechanism of this attachment is unknown. A previous study showed that PC trophozoites bind to fibronectin (Fn) in a saturable and specific manner. The purpose of the current study was to discover whether PC trophozoites bind to Fn via the known cell binding site on the Fn molecule. This site has been localized to the tetrapeptide sequence Arg-Gly-Asp-Ser (RGDS). Using an ^{125}IFn binding assay, Fn binding to PC trophozoites was inhibited by addition of RGDS. This inhibition was comparable to that obtained with intact Fn, but required a 1000 fold higher molar concentration. The false peptide Arg-Gly-Glu-Ser (RGES) had no effect on Fn binding to PC. (* = $p<.03$)

	^{125}IFn alone	^{125}IFn +1uM Fn	^{125}IFn +1mM RGDS	^{125}IFn +1mM RGES
Bound ^{125}IFn(ng)	903±109	305±146*	292±89*	1045±184

Thus PC trophozoites recognize and bind to the well characterized cell binding site on the Fn molecule, localized to the tetrapeptide sequence RGDS. Modulating PC adherence to alveolar epithelium through the use of Fn analogs may provide novel therapy in the treatment of Pneumocystis carinii infection.

Mi impresión diagnóstica es FA con BAV completo y ritmo del haz de His. SCA con QT prolongado.

En el ECG del paciente de 84 años:

1. No visualizo onda P, si una línea de base en serrucho en V1 y V2 que podría corresponderse con una Fibrilación auricular, como el ritmo ventricular es regular de 53 latidos minutos (eso es lo que logro medir). Interpreto con respecto a esto que se trata de una FA con BAV completo y un ritmo nodal o del haz de His. Digo esto porque el QRS no se encuentra desviado ni con aumento del tiempo de conducción. Me gustaría conocer la opinión de los especialistas acerca del eje eléctrico y las S profundas en V5 y V6.

2. Intervalo QT prolongado, estoy plenamente de acuerdo y se presenta en pacientes con síndrome coronario agudo. El QTc lo estimo en 560 mseg. No refiere el Dr que envió el ECG sintomatología como debilidad muscular etc que me puedan hacer pensar en un trastorno electrolçitico como causa de la prolongación del QT.

3. Presenta transtornos de la repolarización en las derivaciones de V2 a V4.

4. Presenta onda U en las mismas derivaciones.

Creo se trata de una **FA con BAV completo**, en el contexto de un paciente de 84 años con un Síndrome coronario agudo. Ya que el paciente según refieren en el envío del ECG tiene ángor.

Me gustaría saber los valores enzimáticos de marcadores de isquemia cardiaca. Obviamente ayudaría en las conductas a seguir.

Desconozco porqué en los datos enviados si se encontraba tomando medicamentos que puedan provocar BAV, ya que si padecía previamente de FA probablemente estuviera tomando BB, amiodarona, o algún medicamento para ésto.

Si estaba tomando medicamentos bradicardizantes y las enzimas cardíacas son normales, se podría tomar la decisión de mantener una conducta expectante y medicar para su síndrome coronario agudo.

En el caso de no estar tomando medicamentos de este tipo previos, se podría evaluar la colocación de un marcapasos transitorio y conducta expectante de acuerdo a evolución.

Y luego con los estudios diagnósticos de ecodoppler (digo ecodoppler porque permite valorar todo lo que ya conocen todos acerca de la utilidad del eco para la evaluación de síndromes coronarios agudos y a mi juicio el doppler siempre brinda información adicional complementaria interesante, función diastólica y en este caso en particular observar la ausencia de onda E lo que confirmaría el diagnóstico de FA y también me permite estimar el volumen minuto no solo la FEy. ya que en este paciente en particular el ángor podría corresponder a un BAV completo con bradicardia sinusal e hipoflujo y ángor secundario a esto.

Si presenta alteraciones de la motilidad en el ecocardiograma o marcadores enzimáticos cardíacos positivos, evaluar de acuerdo a evolución y respuesta al tratamiento CCG. Esto de acuerdo al riesgo de arritmias en el contexto de un paciente con QT prolongado en el escenario de un IAM.

S. Ahnve

- QT Interval Prolongation in Acute Myocardial Infarction
European Heart Journal Advance Access published on November 2, 1985, DOI 10.1093/eurheartj/6.suppl_D.85.
Eur Heart J 6: 85-95.

Paventi, Saverio, Bevilacqua, Umberto, Parafati, Maria A., Di Luzio, Enza, Rossi, Francesco, Pelliccioni, Patrizia R., Paventi, Saverio

- QT Dispersion and Early Arrhythmic Risk During Acute Myocardial Infarction
Angiology 1999 50: 209-215

Martín Ibarrola

Hola amigos:

Mi participación en esta excelente Lista es rutinariamente como "lector", ya que no soy arritmólogo, sino cardiólogo clínico.

Y, como tal, me voy a animar a opinar, no tanto sobre la arritmia en sí misma (destacados colegas ya han opinado), sino sobre la impresión general que tengo al ver este hermoso ECG:

a- Ritmo "lento", regular, con ondas de Fibrilación auricular en V1.

b- En las derivaciones del plano frontal tendencia al bajo voltaje, con un eje eléctrico no bien determinado.

c- En V1, comienzo con q, y bloqueo de rama derecha.

d- Diferencia de voltaje entre V1 y V2 (sobrecarga auricular derecha).

e- La repolarización en cara anterior,con signos compatibles de "repolarización no homogénea" y/o cardiopatía isquémica. (QT largo, ondas T +/- y -/+).

Y en el plano frontal la "casi ausencia) de ondas T.

f- Persistenia de ondas S llamativas en V5 y V6

Mi primera impresión "clinico-ECG?":

Puede tratarse de un paciente con EPOC (enfermedad pulmonar obstructiva crónica), con sobrecarga derecha,+ fibrilación auricular crónica + bloqueo AV, tratado crónicamente con amiodarona, y, consecuentemente, algún grado de hipotiroidismo por la droga,+ cardiopatía isquémica.

Gracias por permitirme opinar (total, mañana termina el 2009, y déjenme el derecho a equivocarme....),

Y Feliz Año Nuevo a todos!!, y en especial a Andrés y a Edgardo.

Un abrazo,

Dr. Mario A. Heñin

ECG. que no presenta onda P; por las características en V1 parece una fibrilación auricular con bradicardia por ritmo nodal, la onda q en V1, ondas t neg de V2 a V4 hace pensar en un cuadro coronario, el QT se encuentra prolongado, desconozco medicación que tenía el paciente, si estaba con amiodarona, o BB o no; le realizaría un ecodopler, y una cámara gamma.

¡Felices fiestas para todo el foro!

PD: me olvidé de enviar fibrilación auricular con bloqueo AV completo.

Manuel Salvador Cano

Dear Dr. Paul Levine: I wiould like to discuss the very interesting case of old man with angina

I agree with your approach related to intractable angina syndrome with srevere bradycardia.

This ECG, is probably a non coronary disease patient

As you remarked in your discussion the ECG shows small limb complexes, but with large one in the precordial leads

In clinical practice this pattern very frecuent indicate sleft ventricular hypertrophy and dilatation

Mostly is very extensive transmural anterior myocardial infarction with deep S waves in right precordial leads

I suspected that this case is a hypertrophic cardiomyopathy witch evolved to dilatation

The atrial flutter / fibrillation seen in V1 is aquencense in all other leads suggesting a long standing atrial fibrillation in a severe fibrotic and dilated left atrium.

The second problem is the severe impaired conduction delay in the upper node and in the infranode junctional tissue

This pattern is not observe in chronic ischemic syndrome

Why?

Because this area is suply by all 3 arteries

1) from the right coronary in 80% of the cases

2) the infranodal is supliedby the circunflex and the first ramus of septal arteries

This tissue is very important to regulate de heart rate toward the ventricle. Mather nature give not overflow to any tissue in the heart, but almost only to the sinus node and to the AV node.

Coronaries event can not destroy these tissues as occur in active myocites and intraventricular conduction system.

I think that a primary disease affect the septo areas.

The ventricular area is also severly affected the wideness of V3 is about 120ms, indicating most probably a severe intraventricular conduction delay.

The inverted waves seen in right precordial leds is the junction between the T and U waves

In a case of an octagenarium patient in whitch the ECG shows all the cardiac areas (atrium, supra and infranode AV node sand the left vent as well) the first clinical diagnosis is infiltrative disease,probably amiloidosis

Why amiloidosis?

Because this infiltrative heart disease has the biological mechanism as Alzeimher

In the hypertrophic heart there are an hyperproduction of protiens, about 50% of the proteins are imperfect structered or unfolded (see cardiovascular research january 2010)

There are a series of proteins that destroy the miss folded

The first line are the chaperones, second line ubiquitine (Nobel award 2007) the third line are the proteosome

This system avoid the entrance of miss folded protein to the myocytes, but is the production is very high or defende system is exauted the imperfect protein enter in the sarcomers

The angina could be provoked by the soffocated microcirculation due to the fibrotic extracelular matrix

The only study I recommend is MRI, witch can I help for the diagnosis

I agree with your therapeutical approach

My kindly regard and happy new years!

Samuel Sclarovsky

Queridos colegas:
Minha análise do ECG;
FC = 48 bpm
Ausencia de onda P
Presença de onda F em VI típicas de fibrilação atrial c/ freq = 300 pm
Presença de onda U nas precordiais (sinal de isquemia?)
Sâp no plano frontal = P não visualizadas
Sâqrs = 40º
sât = indeterminado
Presença de onda q em aVL e QS em V1

Diag = ritmo hissiano alto
Fibrilação atrial c/ frequencia ventricular baixa
Alteração difusa da repolarização ventricular

Dr. Adail Paixao Almeida - Bahia – Brasil

Agregando algunos detalles si es que no lo escribieron, diria que el paciente tiene una sobrecarga de VD por los complejos bajos en plano frontal, signo de Tranchesi áeñaloza, quizas auricular derecha grande, Eje en plano horizontal hacia atras y a la derecha con S profunda hasta V6. Si tiene una Presión aumentada del lado derecho es un motivo más para dolor precordial por disminución de flujo. No hay onda Q ni en D1, aVL ni V5 ni V6. Seguramente el vectocardiograma nos dirá si tiene un área inactiva septal o no, y lo relativo a la sobrecarga.

Un abrazo a todos

Emilio Marigliano