Elderly Woman Carries Symptomatic Sinus Node Dysfunction (SND) or Sick Sinus Syndrome (SSS)

Mulher idosa portadora de Doença do Nó Sinusal Sintomática

Which is the appropriate approach?

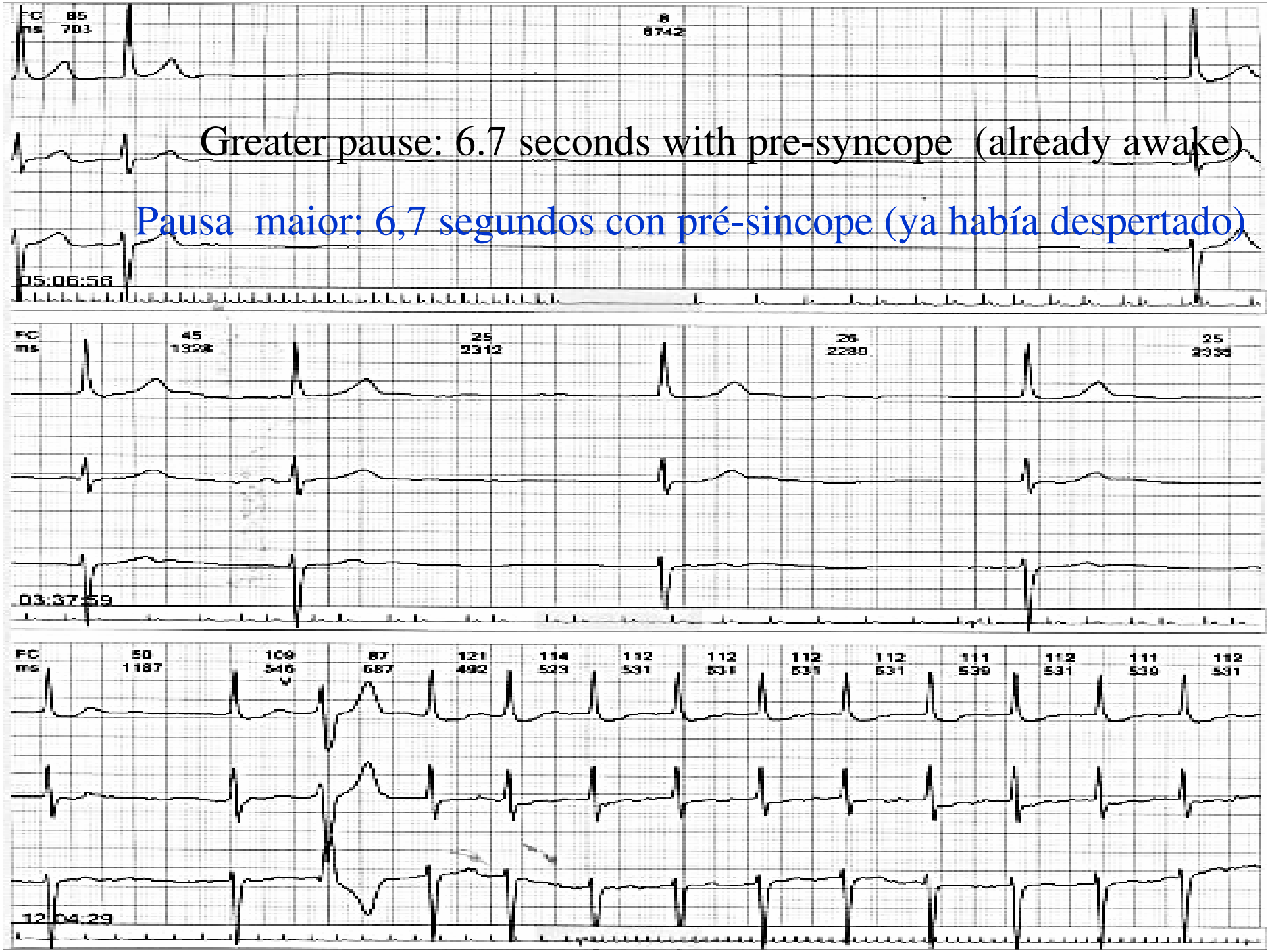
Raimundo **Barbosa-Barros M.D.**
raimundobb@uol.com.br

Specialist in Cardiology by the Brazilian Society of Cardiology (SBC).
Specialist in Intensive Care by the Sociedade Brasileira de Terapia Intensiva.
Chief of the Coronary Center of the Hospital de Messejana Dr. Carlos Alberto Studart Gomes.
Fortaleza - Brazil.

Case report English

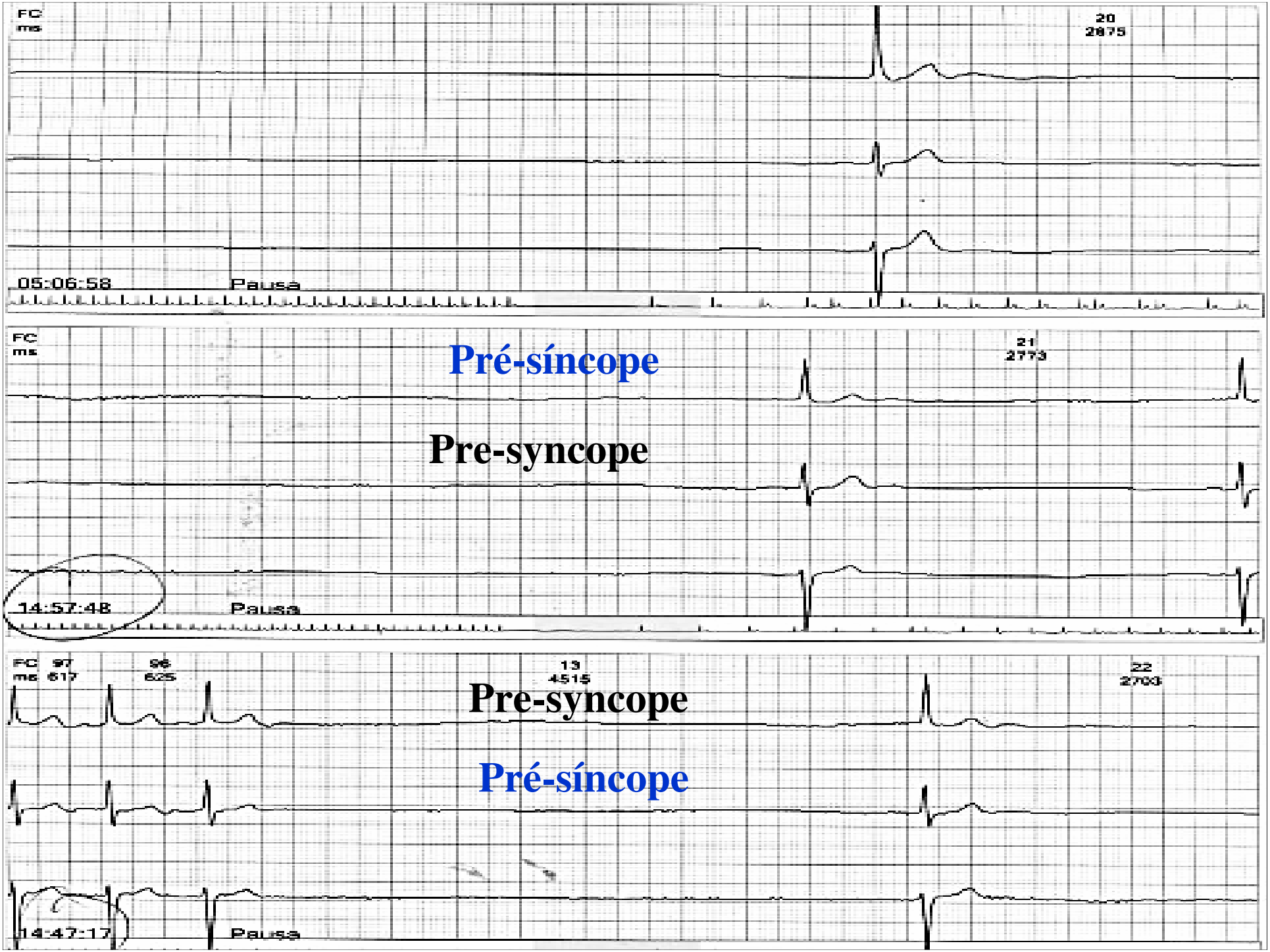
Master, whenever you have a spare minute, present this case to the forum. 71-year-old woman with (biological) mitral valve replacement from 2 years ago. History of pre-syncope and palpitations for 2 months. Taking warfarin, enalapril maleate and espironolactone.
Should she be indicated ablation of atrial tachycardia with the aim of improving sinus node function? Or, Should we indicate permanent pacemaker and then try to stabilize the atrium with drugs?
Raimundo Barbosa-Barros M.D. Fortaleza Ceará

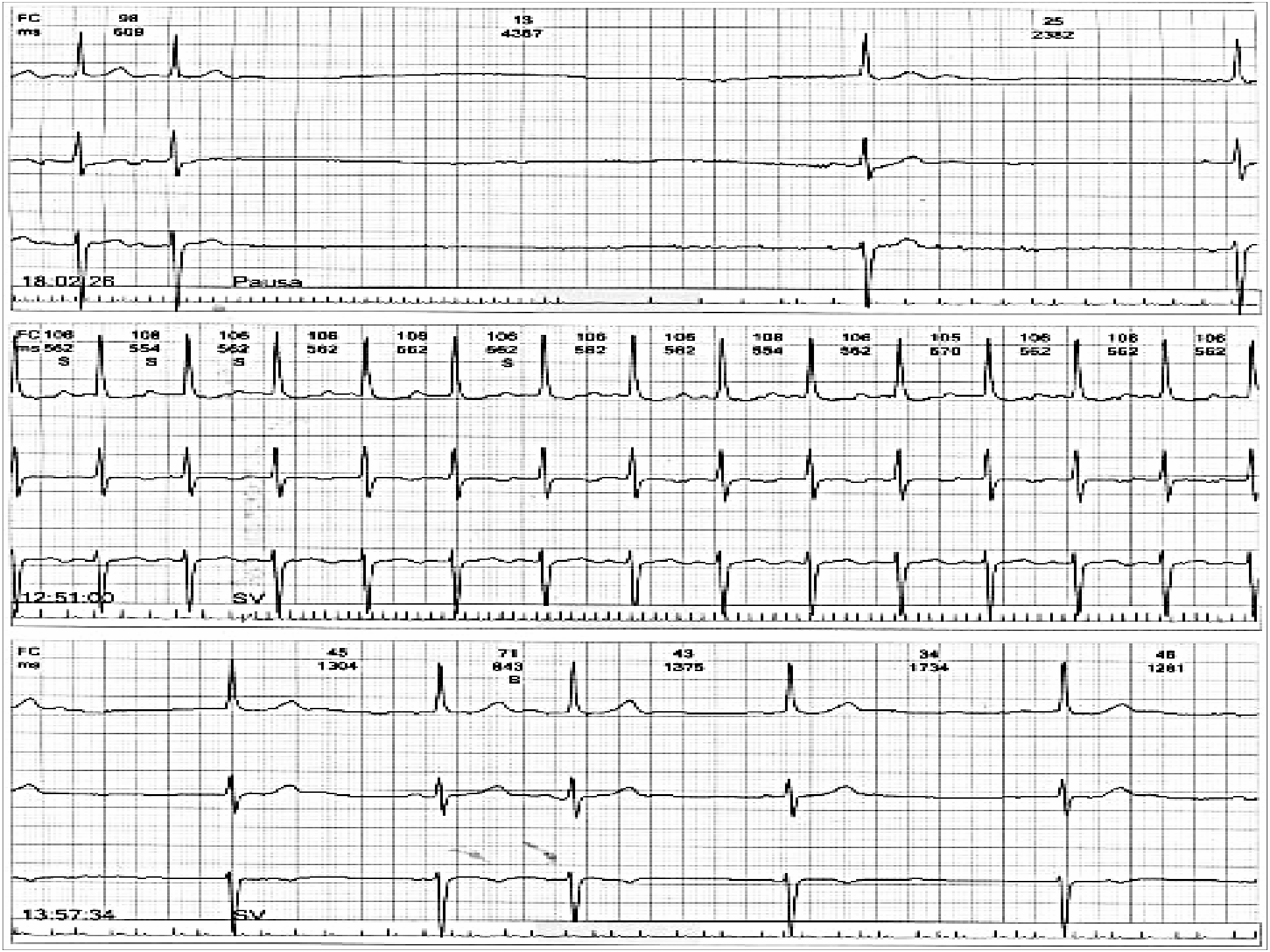
Maestro,quando tiver tempo suba para o foro este caso. Mulher de 71 anos con substituição da válvula mitral (por prótese biológica) há 2anos. História de pré-síncope e palpitações por 2 meses. Em uso regular de warfarina, enalapril e aldactone.
Têm indicação de ablação da taquicardia atrial com o intuito de melhorar a função do Nó sinusal? ou melhor indicar implante de marcapasso definitivo e depois tratar de estabilizar o átrio com fármacos?
Raimundo



Greater pause: 6.7 seconds with pre-syncope (already awake)

Pausa maior: 6,7 segundos con pré-sincope (ya había despertado)


FC
ms
20
2875
05:06:58
Pausa
Pré-síncope
Pre-syncope
FC
ms
21
2773
14:57:48
Pausa
FC 97
ms 617
96
625
13
4515
Pre-syncope
22
2703
Pré-síncope
14:47:17
Pausa


FC
ms
98
609
13
4287
25
2382
18.02.26
Pausa
FC 108
ms 562
S
108
554
S
106
562
S
106
562
106
562
106
562
S
106
562
106
562
108
554
106
562
105
570
106
562
106
562
106
562
12:51:00
SV
FC
ms
45
1304
71
843
S
43
1375
34
1734
46
1281
13:57:34
SV

ECOCARDIOGRAMA 1 BIDIMENSIONAL COM DOPLER

Medidas e Calculos			Vl. Referencia	
Peso : 59.00 KG	Altura : 1.55 m		ASC : 1.57 m2	
Aorta	31.00 mm	De	20.00 a	37.00 mm
Atrio Esquerdo	52.00 mm	De	20.00 a	40.00 mm
Atrio Esquerdo/Aorta	1.68 mm	Até	1.40 mm	
Ventriculo Direito	14.00 mm	De	7.00 a	26.00 mm
Septo Interventricular	9.00 mm	De	7.00 a	11.00 mm
Parede Posterior	9.00 mm	De	7.00 a	11.00 mm
Septo IV/Parede Posterior	1.00 mm	Até	1.40 mm	
VE - Diametro Diastolico	47.00 mm	De	35.00 a	56.00 mm
VE - Diametro Sistolico Final	36.00 mm	Até	39.00 mm	
VE - Volume Diastolico Final	102.36 ml	Até	97.34 ml	
VE - Volume Sistolico Final	54.43 ml	Até	29.55 ml	
Massa VE	142.11 g	masc até 224 e fem até 162		
Ind. Massa VE	90.30 g/m2	masc até 115 e fem até 95		
Relacao V/M	0.7			
Fracao de Encurtamento	23,404 %	> 27 %		
Fracao de Ejecao	46,823 %	> 53 %		

Bioprosthesis in the mitral position with good mobility of mobile elements. Valve area by PHT = 2.3cm2. Discrete central regurgitation. Left atrium with moderate increase, mild aortic regurgitation and mild to moderate tricuspid. Maximum gradient left atrium / left ventricle of 16 mm Hg and a mean gradient of 6 mm Hg LVEF 46%: Mild LV disfuction

Portuguese Conclusão do Ecocardiograma

Conclusão:

- Prótese biológica em posição mitral apresenta boa mobilidade dos elementos móveis, área valvar pelo PHT = 2,3cm², gradiente máximo AE - VE = 16,0mmHg, gradiente médio AE - VE = 6,0mmHg, regurgitação discreta central.
- Moderado aumento do átrio esquerdo.
- Movimento aplainado do septo interventricular (pós cirurgia).
- Regurgitação aórtica leve.
- Regurgitação tricúspide de leve a moderada.

Colleagues opinions

Spanish

Si no estaba tomando drogas frenadoras del automatismo y conducción creo que tiene indicación de MPD y lo antes posible. El ritmo de escape es muy lento y por lo tanto con mucho riesgo, mas en un paciente con cardiopatía estructural, FEVI disminuída y operado.
No tengo elementos concluyentes en el Holter para hablar de taquicardia auricular.
Si no tengo otros registros con otra arritmia le pondría un MP bicameral.
Saludos:
Alejandro Cuesta
arritmia@yahoo.com

English

If she is not taking automatism and conduction blocking drugs, I think the indication is permanent PM and as soon as possible. The escape rhythm is very slow and so, highly dangerous, all the more so in a patient with structural heart disease, decreased LVEF and operated.
I see no conclusive elements in Holter about atrial tachycardia. If I have no other recordings with another arrhythmia, I would implant a dual chamber PM.
Regards,
Alejandro Cuesta M.D.Ph.D. Montevideo Uruguay
arritmia@yahoo.com

English

Dear Andrés and Raimundo,

In the Holter recording, the multiple symptomatic prolonged pauses do not present blocked P waves and nodal escape beats are evident. I see first degree AVBlock. I don't know if in the ECG it is present. I quote "I have the impression that the patient does not have cardiomyopathy, since the LV diastolic diameter is normal and type A paradoxical septal motion is mentioned (post-surgery). Consequently, LV systolic function should be good and the report of the operator could be mistaken. It has not been reported if the prosthetic valve is functioning normally, neither the LV function, nor if she presents mitral annulus calcification or not, nor if the degree of LA dilatation is moderate."

I interpret that associated hypersensibility of the carotid sinus has been ruled out, considering that with the atrial dilatation that she presents, the evolution to rapid atrial arrhythmias (atrial fibrillation, atrial flutter, or atrial tachycardia) would be expected.

I would not make a tilt test or an EPS to show dysfunction. My management would be to implant permanent pacemaker and management with antiarrhythmic medication. I would not make her undergo an invasive procedure, because it entails risks in a scenario of dilated atrium, unless she does not respond to the prescribed drugs. My only doubt is whether to ablate or to indicate antiarrhythmic drugs, since the indication of pacemaker is class IA. The pacemaker would be useful because it would allow using beta blockers for her cardiomyopathy that she cannot receive currently.

The prognosis in the long term would be given by the evolution of cardiomyopathy; the addition of a beta blocker would contribute to the improvement of survival and hospitalizations.

Warm regards to both,

Martin Ibarrola M.D. Argentina

Spanish

Estimado Potro y Raimundo: en el registro Holter las multiples pausas prolongadas sintomáticas no presentan ondas P bloqueadas y se evidencian latidos de escape nodales. Observo BAV de primer grado. Desconozco si en el ECG lo presenta.
Tengo la impresión que la paciente no tiene una miocardiopatia porque el diámetro diastólico del VI es normal y se refiere movimento septal paradójico tipo A (póst-cirúrgico) Consequentemente, la función sistólica del VI debe ser buena y el informe del operador puede estar errado. No se ha informado si la válvula protésica es normo-funcionante, y si tiene o no calcificación del anillo mitral. interpreto han descartado hipersensibilidad del seno carotideo asociado, dado que con la dilatación auricular que presenta la evolución a arritmias auriculares rapidas (FA, fluter auricular, o taquicardias auriculares) seria lo esperable. No realizaria estudios de tilt test ni EEF para evidenciar la disfunción. Mi conducta seria implantación de marcapasos definitivo y manejo con medicación antiarritmica. No la someteria a un procedimiento invasivo no exento de riesgos en una auricula dilatada a no ser que no responda a los fármacos indicados, la unica diferencia que encuentro es si ablacionan o indican antiarritmicos, ya que la indicacion de marcapasos es clase IA.Ademas encuentro que seria de utilidad el mismo ya que permitiria utilizar BB para su miocardiopatia que no puede recibir en el momento actual. El pronóstico a largo plazo estará dada por la evolución de la miocardiopatia, el agregado de BB contribuiria a mejoria de sobrebrida y hospitalizaciones.
Un abrazo a ambos
Martin Ibarrola

Prezados Masters El Potro e The Fox Decidir implante de Marca-passo em Doença do Nó Sinusal naturalmente já é dificil a quando acompanhada da bradi-taqui mais complicada ainda. Minha impressão é que a taquiarritmia é bradicardica dependente, portanto só o marca-passo estabilizaria a paciente.

Dears Masters "The Mustang" and " Fox"
Put a device in Sick Sinus Syndrome must be made with caution and more attention when are brady-tachy arrhythmia. In this case I find that only pace is sufficient because the tachyarrhythmia seems be bradycardia dependent.
Adail Paixao Almeida Vitoria da Conquista Bahia Brazil

Final comments

Andrés Ricardo **Pérez-Riera M.D.PhD.**

Sick Sinus Syndrome (SSS) or Sinus Node Dysfunction (SND), is a relatively uncommon syndrome characterized by dysfunction of the SA-node that is often secondary to senescence of the SA node and surrounding atrial myocardium. The term "sick sinus syndrome" was first used in 1967 to describe the sluggish return of SA nodal activity in some patients following electrical cardioversion, and was later applied to a clinical syndrome characterized by chronic SND, a sluggish or absent SA nodal pacemaker after electrical cardioversion, frequently depressed escape pacemakers, and/or AV nodal conduction disturbances (1). These abnormalities can result in profound sinus bradycardia, sinus pauses, sinus arrest, SA nodal exit block, and inappropriate responses to physiological demands during exercise or stress. Bradycardia-tachycardia syndrome is a variant of SSS in which slow arrhythmias and fast arrhythmias alternate. SND or SSS is an umbrella term that covers three heart rhythm problems (arrhythmias):

1. Sinus bradycardia, which causes a slow heart rate
2. Tachycardias, which cause fast heart rates, often followed by a very slow heart rate. Types of tachycardias include atrial fibrillation, atrial flutter/tachycardia, and supraventricular tachycardia
3. Bradycardia-tachycardia, which causes alternating slow and fast heart rhythms

SND or SSS is more common in elderly adults, where the cause is often a non-specific, scar-like degeneration of the cardiac conduction system. Cardiac surgery, especially to the atria, is a common cause of SSS in children. Coronary artery disease, high blood pressure, and aortic and mitral valve diseases may be associated with SSS, although this association may only be incidental.

What causes SSS is not completely understood, but we do know that disorders that cause scarring, degeneration, or damage to the heart can cause SND or SSS . These include: Certain medications can make abnormal heart rhythms worse. These include the following drugs, which are all prescribed for various heart problems: digitalis, calcium channel blockers, dysopiramide, lidocaine, beta-blockers anti-arrhythmic drugs and Lithium. The last drug induced SND at therapeutic levels. Lithium is used as an antimanic and mood-stabilising drug. It can cause various adverse effects such as nausea, vomiting, polyuria, fine tremors, myocarditis and arrhythmias. Lithium induced SND with serum lithium levels in therapeutic range**.(2)**

Gradual loss of sinus rhythm occurs after the Mustard, Senning, and all varieties of the Fontan operation. This is thought to be secondary to direct injury to the Sinus Node during surgery and also due to later, chronic hemodynamic abnormalities. Paroxysmal atrial tachycardias are frequently associated with SND or SSS , and loss of sinus rhythm appears to increase the risk of sudden death. Patients with transposition of the great arteries now undergo the arterial switch operation, which avoids the extensive atrial suture lines that lead to SN damage.

SND or SSS was described in 15% of patients who had undergone the Ross operation for aortic valve disease or complex left-sided heart disease, 2.6-11 years earlier. Other arrhythmias, such as complete AV block and VT, were present as well after the Ross operation.

When repairing ASDs, especially sinus venosus ASDs, SND or SSS frequently occurs because of the proximity of the defect with SN tissue.

Other surgically related causes of SND or SSS include the following:

Patients who have undergone surgery for endocardial cushion defects may later develop SND

SND or SSS may be caused by a Blalock-Hanlon atrial septectomy

SND or SSS may occur after repair of partial or total anomalous pulmonary venous return

Cannulation of the superior vena cava, usually performed for cardiopulmonary bypass or extracorporeal membrane oxygenation, may damage Sinus Node tissue.

Ischemic cardiac arrest may cause SND or SSS .

SND or SSS has (3;4;5;6;7) been documented in patients with symptomatic Brugada Syndrome (BrS), which suggests it is not a rare concomitant. The only accepted treatment of BrS is today implantation of an ICD. In the future studies should evaluate if PM in some cases of symptomatic BrS can be used instead of ICDs in patients with a loss-of-function SCN5A mutations.

Congenital SSS is due to mutations of gene responsible for formation of Alpha subunit of sodium channel. An study showed that the heterozygous variant of connexin 40 polymorphism gene(8) variant is more frequent among patients with SND or SSS and their healthy relatives than in persons of control group, suggesting a link between the gene and the disease

Coronary artery disease

Coronary artery disease is believed to be a common contributory cause of SND, probably through atherosclerotic changes in the SN artery.

In rare cases, SND or SSS *may be associated with conditions such as:*

1. ***Diphtheria*** *(an infection that can damage the heart muscle)*
2. ***Hemochromatosis*** *(excess iron in the blood)*
3. ***Muscular dystrophy*** *(an inherited condition in which the body's muscles are damaged and weak)*
4. ***Amyloidosis*** *(a condition in which a protein called amyloid is deposited in tissues or organs) (8) Cardiac amyloidosis results in severely symptomatic heart failure that has a poor prognosis because of the development of a restrictive cardiomyopathy. The diagnosis of cardiac amyloidosis is often delayed because of nonspecific signs and symptoms.*
5. **Atrial giant cell myocarditis:** it is a clinical condition characterized by acute heart failure, severe atrial dilatation, mitral/tricuspid regurgitation,atrial mural thrombus, atrial wall thickening, and atrial hypokinesis whit normal ventricular function(9)
6. ***Rheumatic fever:*** it is another cause of SND or SSS .
7. **CNS disease,** which is usually secondary to increased intracranial pressure with subsequent increase in the parasympathetic tone.
8. **Endocrine-metabolic diseases**:hypothyroidism and hypothermia
9. **Electrolyte imbalances:** hypokalemia and hypocalcemia.
10. **Kawasaki disease** SND or SSS and AV block have been found to occur more frequently in pediatric patients with Kawasaki disease with moderate to severe coronary artery disease than in the general population. This is believed to be secondary to myocarditis or abnormal microcirculation in the SN artery and the AV-node artery.

A routine ECG may provide further information in such patients. However, the symptoms are nonspecific and the ECG changes may not be diagnostic, thereby necessitating either noninvasive or electrophysiologic studies (EPFs) to confirm the diagnosis.

Epidemiology

Occurrence in the United States

The exact incidence of SND or SSS is unknown. The syndrome occurs in approximately 1 in 600 cardiac patients older than 65 years.(10)

International occurrence

Due to its relationship with advanced age, SND or SSS is more prevalent in countries where citizens have a longer life expectancy.

Age-related demographics

SND may develop at any age but it is primarily a disease of the elderly, with the average age of occurrence being about 68 years.(11) SND or SSS in young patients is often related to underlying heart disease or associated with SCN5A mutation Age-related changes are believed to be the most common cause of SND or SSS and are related to fibrosis in the SN. These fibrotic changes also occur in the atrium and the conduction system of the heart and are believed to contribute to the association among SND or SSS, tachy-brady syndrome, conductive system disease, and an inappropriately slow escape rhythm.

The diagnostic evaluation should also include a search for reversible causes of SA nodal depression, such as drugs eg, beta blockers, calcium channel blockers, digoxin, ischemia, and autonomic The initial clues to the diagnosis of SND or SSS are often clinical, as patients may present with symptoms of Even though many types of SSS produce no symptoms, patients may present organ hypoperfusion and pulse irregularity. Such signal and symptoms include the following:

1. **Slower than normal pulse (bradycardia)**
2. **Stokes-Adams attacks - fainting due to asystole or ventricular fibrillation**
3. **Fall**
4. **Dizziness or light-headedness Fainting or near fainting lightheadedness, presyncope, syncope,**
5. **A sensation of rapid, fluttering heartbeats (palpitations)**
6. **Chest pain or angina**
7. **Shortness of breath or dyspnea on exertion,.**
8. **Fatigue**
9. ***Weakness***
10. **Headache**
11. **Nausea**
12. **Interrupted sleeping**
13. **Confusion or difficulty remembering things**

Risk Factors for Sick Sinus Syndrome

While the exact cause of SND or SSS *is unknown, some factors, however, often are associated with the condition, such as:*

Age

1. ***Previous heart attack (myocardial infarction)***
2. ***Medications to treat high blood pressure and other heart diseases***
3. ***Hyperkalemia (too much potassium in the blood)***
4. ***Thyroid disease***
5. ***Sleep apnea***
6. ***Heart surgery***
7. ***Atrial fibrillation (AF) :*** *The risk of stroke in patients with AF can be assessed by use of the CHADS2 and the CHA2DS2-VASc score system.* CHADS2 and CHA2DS2-VASc score are associated with increased risk of stroke and death in patients paced for SND or SSS irrespective of the presence of AF**.(12)**

Prognosis factors

Atrioventricular block, chronic AF, and systemic embolism are major pathologic conditions that affect the outcome of the syndrome.

The incidence of sudden cardiac death in patients with SND or SSS is very low.(13) Mortality in patients with SNDis primarily determined by underlying heart disease.

Pacemaker therapy does not appear to affect survival in patients with SND(14, 15, 16) and is, therefore, used primarily for the alleviation of symptoms. Symptomatic patients with normal systemic ventricular function and SND or SSS have an overall good prognosis with atrial (rate-responsive) pacing.

Patients with tachy-brady syndrome have a worse prognosis than do patients with isolated SND or SSS The overall prognosis in patients with SND and additional systemic ventricular dysfunction (eg, numerous postoperative Mustard and Fontan patients) depends on their underlying ventricular dysfunction or degree of congestive heart failure (CHF).

Patients who have undergone a Fontan surgery and developed SND or SSS , endocardial atrial leads can be implanted relatively safely and can permit low-energy thresholds for as long as 5 years after implantation.(17)

Treatment

Treatment of SSS is directed at symptoms, which may include lightheadedness, presyncope, syncope, and, less often, dyspnea on exertion or worsening angina. In addition, patients with tachycardia-bradycardia syndrome may present with palpitations and other symptoms associated with a rapid heart rate. However, it is not uncommon for patients to develop clinical manifestations of SND or SSS insidiously. While some individuals present with frank syncope, patients more commonly report progressive development of the symptoms described above and often equate this with natural "aging".

Treatment for SSS focuses on eliminating or reducing unpleasant symptoms. For people who are bothered by symptoms, the treatment of choice is usually an implanted electronic pacemaker.

Medication changes

Antiarrhythmic therapy of patients with disturbed automatism of the SA node and impaired AV conductance may be complicated by hemodynamically significant bradycardias and contraindications for pacemaker placement for SND or SSS If current medications to see if any of them could be interfering with the function of SA node. Medications used to treat high blood pressure or heart disease — such as beta blockers or calcium channel blockers — can worsen abnormal heart rhythms. In some cases, adjusting these medications can relieve symptoms.

Pacing the heart

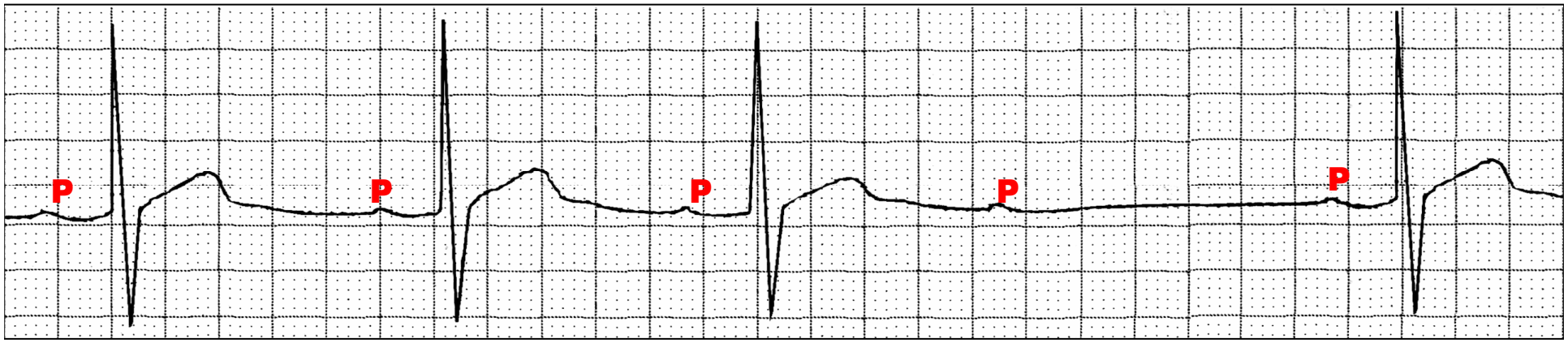
Most people with SSS eventually need a permanent artificial pacemaker (AP) to maintain a regular heartbeat. This small, battery-powered electronic device is implanted under the skin near collarbone during a minor surgical procedure. The AP is programmed to stimulate or "pace" heart as needed to keep it beating normally. The type of AP depends on the type of irregular heart rhythm. Some rhythms can be treated with a single-chamber pacemaker, which uses only one wire (lead) to pace one chamber of the heart - in this case, the atrium. However, most people with SSS benefit from dual-chamber pacemakers, in which one lead paces the atrium and one lead paces the ventricle. The risk of complications, such as swelling or infection in the area where the pacemaker was implanted, is small. Cardiac pacing is the most powerful therapy; physiologic pacing (atrial or dual-chamber) has been shown definitively to be superior to ventricular pacing. According to the 2008/2012 ACC/AHA/HRS guidelines, pacemaker therapy has the following indications: .(18)

- **Class I indication** - For patients with documented symptomatic sinus bradycardia, sinus pause, and chronotropic incompetence; this includes patients who have iatrogenic SND secondary to essential medications for which no acceptable alternatives exist
- **Class IIa indication -** For patients with SND and a sinus rate below 40 bpm when a clear association between symptoms (ie, symptoms consistent with bradycardia) and bradycardia has not been documented
- **Class IIa indication -** For patients with syncope of unexplained origin when clinically significant abnormalities of SN are discovered or provoked in EP studies
- **Class IIb indication -** For patients with minimal symptoms and a chronic heart rate of less than 40 bpm while awake.
- **Class III indication -** Pacemaker therapy is contraindicated in patients with asymptomatic SND or symptomatic bradycardia due to medications that are not essential. Sinus pauses occurring during sleep are generally not considered indications of pacing, May patients , particularly trained athletes, have high levels of vagal tone and have significant pauses greater than 3 seconds and periods of sinus bradycardia, these patients do not require permanent pacing See next sequence two slides

Holter Recording

2nd Degree Av Block, Mobitz Type II
With Narrow QRS

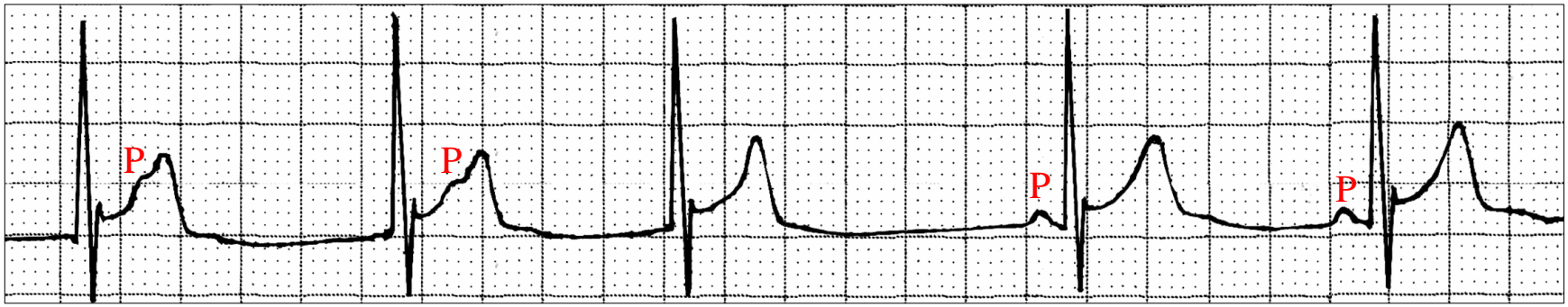
Name: E . J. **Sex:** Male **Age:** 26 **Race:** White **Weight:** 70 Kg.
Height: 1.72 m **Biotype:** Athletic **Date:** 25/01/2001
Time: 1:52:10 AM Patient sleeping **Profession:** Long distance runner



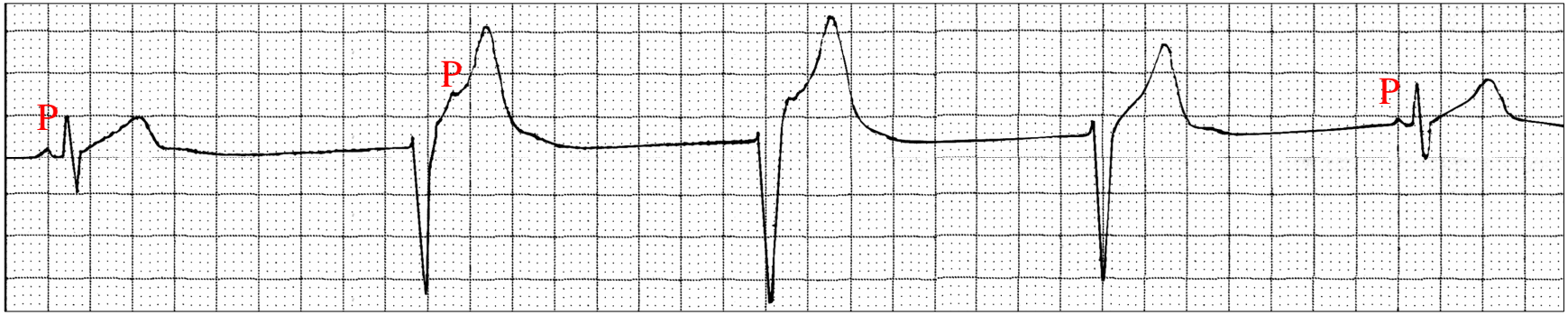
PR interval remains constant until a P wave is not conducted. This type of block is observed in 7% of the cases in athletes of enduro. Fixed or constant PR interval: it does not exist, progressive prolongation of PR, with the block occurring suddenly. In general, 2nd degree AV block type II with narrow QRS is observed in 35% of the cases and in the remaining 65%, the QRS is long.

Holter Recording

Atrioventricular Dissociation (Dissociation By Interference) With Junctional Scape Rhythm



Atrioventricular Dissociation (Dissociation By Interference) With Scape Ventricular Rhythm



Atrioventricular dissociation (dissociation by interference) with junctional scape rhythm and atrioventricular dissociation (dissociation by interference) with scape ventricular rhythm in an elite athlete in Holter.

In patients who have type II second-degree AV block, the demonstration of His-Purkinje block, even in the minimally symptomatic or possibly asymptomatic person, may be sufficient evidence to conclude that pacemaker theraphy is indicated because of the risk for progression to complete AV block

Electrophysiology Study value

EPS of SND or SSS have low sensitivity but relatively high specificity.

EPS is indicated only when a causal relation between the appearance of bradycardia and the patient's symptoms cannot be established despite repeated noninvasive evaluations!

Single- Versus Dual-Chamber Pacemakers

In patients with SND, the annual incidence of complete AV block is about 0.6%.(19) In the United States, the implantation of dual-chamber pacemakers is preferred in practice because their use anticipates the possible subsequent development of conducting system dysfunction. This practice is supported by data from the Danish Multicenter Randomized Trial on Single Lead Atrial Pacing versus Dual Chamber Pacing in Sick Sinus Syndrome (DANPACE) trial, in which 9.3% of patients with single-lead atrial pacing (AAIR) required upgrade to a dual-chamber pacemaker (DDDR) over 5.4 years follow-up due to new development of significant AV conduction abnormalities. This was necessary despite the fact that these patients had no significant intraventricular conduction abnormality, PR intervals below 260ms, and no Wenckebach AV block with atrial pacing at 100 bpm at baseline.(20) In addition, patients in AAIR mode had more AF than did patients in DDDR mode. Importantly, however, no significant mortality difference between AAIR and DDDR mode was noted.

Arguably, a single-chamber atrial pacemaker with AAI mode is an acceptable alternative in patients with SND and normal AV and intraventricular conduction because of the added expense of and the potential for more lead extraction with a dual-chamber pacemaker.

In patients with SND and known AV conduction abnormality (including bundle branch block and bifascicular block), a dual-chamber pacemaker should be used due to the high risk of AV block (about 36% in a 5-year follow-up study).

Pacemaker Programming

Chronic right ventricular pacing has been shown to be associated with an increased incidence of AF, stroke, heart failure, and probably death.(13;21;22) A study suggested that RV pacing is detrimental to LV function even in patients with a normal LVEF.(21) Therefore, avoiding RV pacing is advantageous in patients with SND treated with pacemaker therapy. However, using the intrinsic AV conduction in patients with a very long intrinsic PR interval may not be beneficial clinically, as suggested by a trial in patients with an ICD.(20) Theoretically, a very long PR interval may result in pacemaker syndrome during sinus tachycardia or fast atrial pacing rhythm.

In the DANPACE trial,(12) about 65% of patients with a moderate AV delay setting in DDDR mode with mean RV pacing had less AF and no increased rate of heart failure, as compared with patients in AAIR mode. Clearly, the optimal AV delay settings in patients with SND are still unknown, although various programming algorithms from different pacemaker companies are very effective in reducing RV pacing.
Mode switch is an important feature to monitor atrial flutter and AF events. Because more than 50% of patients with SND may over time develop tachy-brady syndrome,(11) it is very important to identify these patients through pacemaker monitoring and anticoagulate them to reduce their risk of stroke. However, the most appropriate anticoagulant therapy is still uncertain for patients in whom AF is detected only as an incidental finding on pacemaker or ICD diagnostics.
Pacemakers with a rate drop response program may benefit some patients with neurocardiogenic syncope. Studies have suggested that closed-loop stimulation technique in the Biotronik pacemaker may be quite helpful in reducing syncope in patients with neurocardiogenic syncope.(24, 25)
Rate response features have been used in patients with SND, especially in the presence of chronotropic incompetence. However, the clinical benefits of this program feature are still controversial.(26)

Additional treatments for fast heart rate

With rapid heart rate as part of SND or SSS, it is necessary additional treatments to control these rhythms: warfarin (Coumadin) or dabigatran (Pradaxa).
AV node ablation. This procedure can also control fast heart rhythms in people with pacemakers. It involves applying radiofrequency energy through a long, thin tube (catheter) to destroy (ablate) the tissue around the AV node between the atria and the ventricles. This stops fast heart rates from reaching the ventricles and causing problems.
Radiofrequency ablation of atrial fibrillation. This procedure is similar to AV node ablation. However, in this case, ablation targets the tissue that triggers AF. This actually eliminates AF itself, rather than just preventing it from reaching the ventricle

References

1. Ferrer MI. The sick sinus syndrome in atrial disease.JAMA. 1968;206(3):645.
2. Shetty RK, Vivek G, Parida A, et al. Lithium-induced sinus node dysfunction at therapeutic levels. BMJ Case Rep. 2013 Jan 22;2013. pii: bcr2012008107. doi: 10.1136/bcr-2012-008107
3. Risgaard B, Bundgaard H, Jabbari R, et al. Pacemaker implantation in a patient with brugada and sick sinus syndrome. World J Cardiol. 2013 Mar 26;5:65-67
4. Smits JP, Koopmann TT, Wilders R, et al. A mutation in the human cardiac sodium channel (E161K) contributes to sick sinus syndrome, conduction disease and Brugada syndrome in two families. J Mol Cell Cardiol. 2005 Jun; 38: 969-981.
5. Sumiyoshi M, Nakazato Y, Tokano T, y col. Sinus node dysfunction concomitant with Brugada syndrome. Circ J. 2005 Aug; 69: 946-950.
6. Fazelifar AF, Haghjoo M, Emkanjoo, et al. Brugada-type ECG association with unexpected sick sinus syndrome. Pacing Clin Electrophysiol. 2006 Feb; 29: 204-206
7. Wilde AA, Brugada R Phenotypical manifestations of mutations in the genes encoding subunits of the cardiac sodium channel. Circ Res. 2011 Apr 1; 108: 884-897 Chernova AA, Nikulina SIu, Shul'man VA, Polymorphism of connexin 40 gene-- a novel genetic marker of the sick sinus node syndromeKardiologiia. 2011;51:17-19.
8. Gilotra NA, Chow GV, Cingolani OH. Cardiac Amyloidosis Presenting with Prolonged QT Interval and Recurrent Polymorphic Ventricular Tachycardia. Tex Heart Inst J. 2013;40:193-195.
9. Larsen BT, Maleszewski JJ, Edwards WD, et al. Atrial giant cell myocarditis: a distinctive clinicopathologic entity. Circulation. 2013 Jan 1;127:39-47
10. Rodriguez RD, Schocken DD. Update on sick sinus syndrome, a cardiac disorder of aging. *Geriatrics*. Jan 1990;45:26-30, 33-36.
11. Adán V, Crown LA. Diagnosis and treatment of sick sinus syndrome. *Am Fam Physician*. Apr 15 2003;67:1725-1732.

12. Lamas GA, Lee KL, Sweeney MO, Silverman R, Leon A, Yee R, et al. Ventricular pacing or dual-chamber pacing for sinus-node dysfunction. *N Engl J Med*. Jun 13 2002;346:1854-1862.
13. Menozzi C, Brignole M, Alboni P, Boni L, Paparella N, Gaggioli G, et al. The natural course of untreated sick sinus syndrome and identification of the variables predictive of unfavorable outcome. *Am J Cardiol*. Nov 15 1998;82:1205-1209.
14. Simon AB, Janz N. Symptomatic bradyarrhythmias in the adult: natural history following ventricular pacemaker implantation. *Pacing Clin Electrophysiol*. May 1982;5:372-383.
15. Alt E, Völker R, Wirtzfeld A, Ulm K. Survival and follow-up after pacemaker implantation: a comparison of patients with sick sinus syndrome, complete heart block, and atrial fibrillation. *Pacing Clin Electrophysiol*. Nov 1985;8:849-855.
16. Shah MJ, Nehgme R, Carboni M, Murphy JD. Endocardial atrial pacing lead implantation and midterm follow-up in young patients with sinus node dysfunction after the fontan procedure. *Pacing Clin Electrophysiol*. Jul 2004;27:949-954
17. Epstein AE DiMarco JP, Ellenbogen KA, et al; 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society. American College of Cardiology Foundation; American Heart Association Task Force on Practice Guidelines; Heart Rhythm Society. J Am Coll Cardiol. 2013 Jan 22;61(3):e6-75. doi: 10.1016/j.jacc.2012.11.007. Epub 2012 Dec 19.
18. Rosenqvist M, Obel IW. Atrial pacing and the risk for AV block: is there a time for change in attitude?. *Pacing Clin Electrophysiol*. Jan 1989;12:97-101.
19. Nielsen JC, Thomsen PE, Hojberg S, Moller M, Vesterlund T, Dalsgaard D, et al. A comparison of single-lead atrial pacing with dual-chamber pacing in sick sinus syndrome. *Eur Heart J*. Mar 2011;32:686-696.
20. Andersen HR, Thuesen L, Bagger JP, Vesterlund T, Thomsen PE. Prospective randomised trial of atrial versus ventricular pacing in sick-sinus syndrome. *Lancet*. Dec 3 1994;344:1523-1528.

20. Svendsen JH, Nielsen JC, Darkner S, et al; DANPACE Investigators. CHADS2 and CHA2DS2-VASc score to assess risk of stroke and death in patients paced for sick sinus syndrome. Heart. 2013 Jun;99:843-848.
21. Sweeney MO, Bank AJ, Nsah E, Koullick M, Zeng QC, Hettrick D. Minimizing ventricular pacing to reduce atrial fibrillation in sinus-node disease. *N Engl J Med*. Sep 6 2007;357:1000-1008.
22. Yu CM, Chan JY, Zhang Q, Omar R, Yip GW, Hussin A. Biventricular pacing in patients with bradycardia and normal ejection fraction. *N Engl J Med*. Nov 26 2009;361:2123-2134.
23. Kanjwal K, Karabin B, Kanjwal Y, Grubb BP. Preliminary observations on the use of closed-loop cardiac pacing in patients with refractory neurocardiogenic syncope. *J Interv Card Electrophysiol*. Jan 2010;27:69-73.
24. Occhetta E, Bortnik M, Audoglio R, et al. Closed loop stimulation in prevention of vasovagal syncope. Inotropy Controlled Pacing in Vasovagal Syncope (INVASY): a multicentre randomized, single blind, controlled study. *Europace*. Nov 2004;6:538-547.
25. Lamas GA, Knight JD, Sweeney MO, Mianulli M, Jorapur V, Khalighi K. Impact of rate-modulated pacing on quality of life and exercise capacity--evidence from the Advanced Elements of Pacing Randomized Controlled Trial (ADEPT). *Heart Rhythm*. Sep 2007;4:1125-1132.