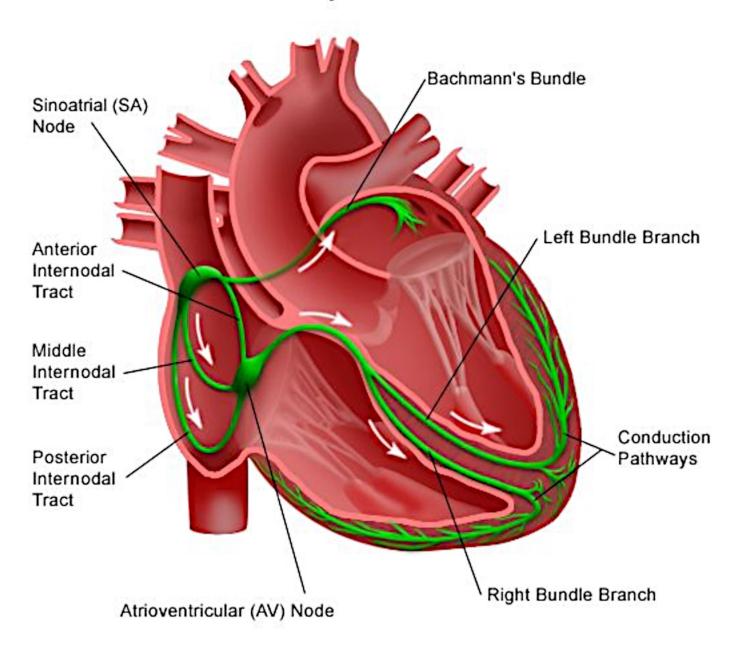
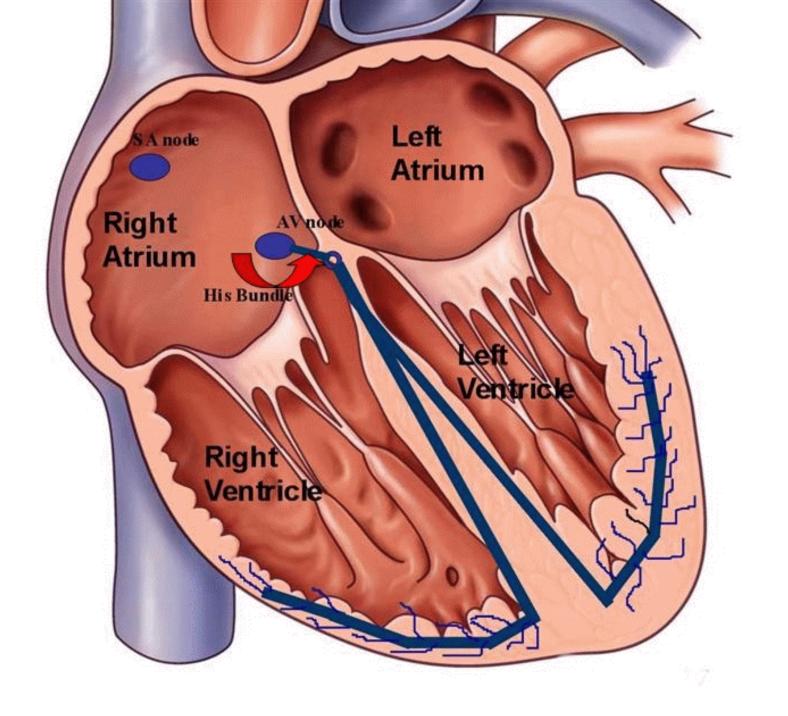
ECG and non ECG hints for the type of arrhythmia

Ilan Lashevsky MD
IWAS 14
Collingwood

Electrical System of the Heart





The peculiar behavior of AVNRT

TABLE 1
Percentage of Symptoms Present by Diagnosis

Symptoms	AVNRT	AVRT	VT	A Flutter	AF
	n = 152	n = 80	n = 25	n = 36	n = 30
Neck pounding	60.5%	43.8%*	36.0%*	13.9%*	26.7%*
Chest pounding	85.5%	88.8%	60.0%	52.8%	66.7%
Palpitations Shirt flapping Dizziness	95.4%	92.5%	68.0%	69.4%	86.7%
	58.6%	43.8%*	32.0%*	16.7%*	13.3%*
	69.7%	63.8%	60.0%	36.1%	56.7%

AVNRT = atrio-ventricular nodal reentry tachycardia; AVRT = atrio-ventricular reentry tachycardia; VT = ventricular tachycardia; A Flutter = atrial flutter. *P < 0.05 versus AVNRT.

TABLE 2

Arterial and Left Atrial Pressures During Sinus Rhythm, Native AVRT, Simulated AVRT, and Simulated AVNRT

Pressures (mmHg)	Sinus Rhythm	Native AVRT	Simulated AVRT	Simulated AVNRT
Mean left atrial pressure Peak left atrial pressure Mean arterial pressure Systolic blood pressure	7.3 ± 2.4 10.5 ± 3.2 96.2 ± 14.8 126.6 ± 21.5	$16.3 \pm 4.2^*$ $21.7 \pm 3.9^*$ $81.4 \pm 17.0^*$ $99.8 \pm 19.7^*$	$13.7 \pm 3.9^*$ $19.3 \pm 4.8^*$ $78.9 \pm 11.9^*$ $96.1 \pm 17.1^*$	$19.4 \pm 4.8^{*\dagger}$ $27.2 \pm 6.0^{*\dagger}$ $70.4 \pm 16.9^{*}$ $83.7 \pm 22.1^{*}$

^{*}P < 0.05 versus sinus rhythm; $^{\dagger}P$ < 0.05 versus simulated AVRT.



"Pacemaker Syndrome"

- Fatigue, dizziness, hypotension
- Caused by pacing the ventricle asynchronously, resulting in AV dissociation or VA conduction
- Mechanism: atrial contraction against a closed AV valve and release of atrial natriuretic peptide
- Worsened by increasing the ventricular pacing rate, relieved by lowering the pacing rate or upgrading to dual chamber system
- Therapy with fludrocortisone/volume expansion NOT helpful

A case of AF

- 38 yo male
- Recurrent AF from 2007, currently 5-6 per year
- Starting with physical activity, bearing down stops arrhythmia
- Symptoms dizziness
- Echo low normal LVEF
- ER ECG atrial fibrillation

A case of AF – which is the most plausible trigger of AF?

Atrial fibrillation

Psycological stress leading to AF

SVT

Brugada syndrome

Atrial flutter

AF in < 65y

Modifiable Atrial Fibrillation Risk Factors

Obesity

Obstructive Sleep Apnea

Hypertension

Diabetes Mellitus

Alcohol Consumption

Incidence of Symptomatic Atrial Fibrillation in Patients With Paroxysmal Supraventricular Tachycardia

MARK E. HAMER, MD,* WILLIAM E. WILKINSON, PhD, WALTER K. CLAIR, MD, RICHARD L. PAGE, MD, FACC, ELIZABETH A. McCARTHY, RN, EDWARD L. C. PRITCHETT, MD

Durham, North Carolina

Objectives. This study was performed to determine the incidence of symptomatic, sustained atrial fibrillation in a group of patients with paroxysmal supraventricular tachycardia. The effects of the mechanism of paroxysmal supraventricular tachycardia (atrioventricular [AV] node reentry vs. AV reentry through an accessory pathway) and heart rate during the tachycardia on the occurrence of atrial fibrillation were also assessed.

Background. There is a substantial incidence of atrial fibrillation in patients with paroxysmal supraventricular tachycardia, but the precise incidence and the factors that determine it are unknown.

Methods. One hundred sixty-nine patients with paroxysmal supraventricular tachycardia were followed up by regular clinic visits and transtelephonic electrocardiographic monitoring during symptomatic episodes of arrhythmia. The Kaplan-Meier product-limit method was used to estimate the proportion of patients remaining free of atrial fibrillation during the observation period. The Cox proportional hazards model was used to assess the effect of mechanism and heart rate during paroxysmal supraventricular tachycardia on the atrial fibrillation—free period.

Results. Thirty-two (19%) of the 169 patients had an episode of atrial fibrillation during a mean follow-up period of 31 months. The cumulative percent of patients experiencing an episode of atrial fibrillation was 6% within 1 month, 9% within 4 months and 12% within 1 year. The mechanism of paroxysmal supraventricular tachycardia was not associated with the time to occurrence of atrial fibrillation; the hazard ratio corresponding to classification in the AV node reentry group was $0.8 \ (p > 0.6)$. The heart rate during paroxysmal supraventricular tachycardia was not associated with the time to occurrence of atrial fibrillation; the hazard ratio associated with an increase in heart rate of 50 beats/min during the tachycardia was $1.15 \ (p > 0.5)$.

Conclusions. This study suggests that atrial fibrillation will develop in ~12% of patients with paroxysmal supraventricular tachycardia during a 1-year follow-up period. The occurrence of atrial fibrillation is not related to the mechanism or heart rate of the paroxysmal supraventricular tachycardia.

(J Am Coll Cardiol 1995;25:984-8)

Lone Atrial Fibrillation in the Young – Perhaps Not So "Lone"?

Scott R. Ceresnak, MD¹, Leonardo Liberman, MD², Eric S. Silver, MD², Steven B. Fishberger, MD³, Gregory J. Gates, PhD⁴, Lynn Nappo, RN¹, Joseph Mahgerefteh, MD¹, and Robert H. Pass, MD¹

Objective To determine if pediatric patients with a history of lone atrial fibrillation (AF) have other forms of supraventricular tachycardia (SVT) that may potentially trigger AF.

Study design A multicenter review of patients with lone AF who underwent electrophysiology (EP) study from 2006-2011 was performed. Inclusion criteria: age ≤21 years, normal ventricular function, structurally normal heart, history of AF, and EP study and/or ablation performed. Exclusion criteria: congenital heart disease or cardiomyopathy. Patient demographics, findings at EP study and follow-up data were recorded.

Results Eighteen patients met inclusion criteria. The mean age was 17.9 ± 2.2 years, weight was 82 ± 21 kg, body mass index was 27 ± 6 , and 15 (83%) were males. Eleven (61%) were overweight or obese. Seven (39%) had inducible SVT during EP study: 5 atrioventricular nodal re-entry tachycardia (71%) and 2 concealed accessory pathways with inducible atrioventricular re-entry tachycardia (29%). All 7 patients with inducible SVT underwent radiofrequency ablation. There were no complications during EP study and/or ablation for all 18 patients. The mean follow-up was 1.7 ± 1.5 years and there were no recurrences in the 7 patients who underwent ablation. There were 2 recurrences of AF in patients with no other form of SVT during EP study.

Conclusions Inducible SVT was found in 39% of pediatric patients undergoing EP study for Ione AF. EP study should be considered for pediatric patients presenting with Ione AF. (*J Pediatr 2013;162:827-31*).



How many atrial fibrillation ablation candidates have an underlying supraventricular tachycardia previously unknown? Efficacy of isolated triggering arrhythmia ablation

Luigi Sciarra^{1*}, Marco Rebecchi¹, Ermenegildo De Ruvo¹, Lucia De Luca¹, Lorenzo Maria Zuccaro¹, Alessandro Fagagnini¹, Leonardo Corò², Giuseppe Allocca², Ernesto Lioy¹, Pietro Delise², and Leonardo Calò¹

¹Cardiology Department, Via Montaione 20 00139, Policlinico Casilino, Rome, Italy; and ²Cardiology Department, Conegliano Hospital, Conegliano Veneto, Italy Received 20 May 2010; accepted after revision 9 August 2010; online publish-chead-of-print 10 September 2010

Supraventricular tachycardia may trigger atrial fibrillation (AF). The aim of the study was to evaluate the prevalence of supraventricular tachycardia (SVT) inducibility in patients referred for AF ablation and to evaluate the effects of SVT ablation on AF recurrences.

Methods and results

Two hundred and fifty-seven patients (185 males; mean age: 53.4 ± 14.6 years) referred for AF ablation were studied. In all patients only AF relapses had been documented in the clinical history. Twenty-six patients (10.1%; mean age: 43.4 ± 13.3 years; 17 males) had inducible SVT during electrophysiological study and underwent an ablation targeted only at SVT suppression. Ablation was successful in all 26 patients. The ablative procedures are: 12 slow-pathway ablations for atrioventricular nodal re-entrant tachycardia; 9 concealed accessory pathway ablations for atrioventricular re-entrant tachycardia; and 5 focal ectopic atrial tachycardia ablations. No recurrences of SVT were observed during the follow-up (21 \pm 11 months). Two patients (7.7%) showed recurrence of at least one episode of AF. Patients with inducible SVT had less structural heart disease and were younger than those without inducible SVT (interventricular septum thickness: 8.4 ± 1.6 vs. 11.0 ± 1.4 mm, P < 0.01; left atrial diameter: 37.0 ± 3.0 vs. 44.0 ± 2.2 mm, P < 0.01; age: 43.4 ± 13.3 vs. 57.3 ± 11.2 years, P < 0.01). Prevalence of paroxysmal AF was higher in patients with inducible SVT when compared with those with only AF (84.6 vs. 24.6%, P < 0.01).

Conclusion

A significant proportion of candidates to AF ablation are inducible for a SVT. SVT ablation showed a preventive effect on AF recurrences. Those patients should be selected for simpler ablation procedures tailored only on the triggering arrhythmia suppression.

Keywords

Atrial fibrillation • Supraventricular tachycardia • Ablation



How many atrial fibrillation ablation candidates have an underlying supraventricular tachycardia previously unknown? Efficacy of isolated triggering arrhythmia ablation

 26 patier electroph Luigi Sciarra^{1*}, Marco Rebecchi¹, Ermenegildo De Ruvo¹, Lucia De Luca¹, Lorenzo Maria Zuccaro¹, Alessandro Fagagnini¹, Leonardo Corò², Giuseppe Allocca², Ernesto Lioy¹, Pietro Delise², and Leonardo Calò¹

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Received 20 May 2010; accepted after revision 9 August 2010; online publish-ahead-of-print 10 September 2010

• 2/26 (7.7

Aims

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Keywords

Atrial fibrillation . Supraventricular tachycardia . Ablation

A case of AF – which is the most plausible trigger of AF?

Atrial fibrillation

Psycological stress leading to AF

SVT

Brugada syndrome

Atrial flutter

SVT – in patient with history of AF

15 Mar 1978

CARESCAPE B850 V2.0.6.2 12SL

		ECG 12 Lead Analysis	1/1 Age Gender	38 years Male
Medical Record Number: Second ID:	M00515454	Identification:	Height Weight	
Bed:	09B1		Ethnicity Ventricular Rate	193 /min
Unit:	ED		PR Interval QRS Duration	ms 84 ms
Printed: Measurement Time:	25 Sep 2016 17:51:31 25 Sep 2016 17:51:29	Notes:	QT/QTc P-R-T Axis	246 / 441 ms 95 -85
* Unconfirmed ECG Report SUPRAVENTRICULAR TACH RIGHTWARD AXIS MARKED ST ABNORMALITY, MARKED ST ABNORMALITY,	YCARDIA POSSIBLE INFERIOR SUBENDOCARI POSSIBLE ANTERIOR SUBENDOCARI	ABNORMAL ECG DIAL INJURY DIAL INJURY		
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N5				

Gain: 10mm/mV Filter Band: Diagnostic (0.05 - 150 Hz)

Case of recurrent syncopes

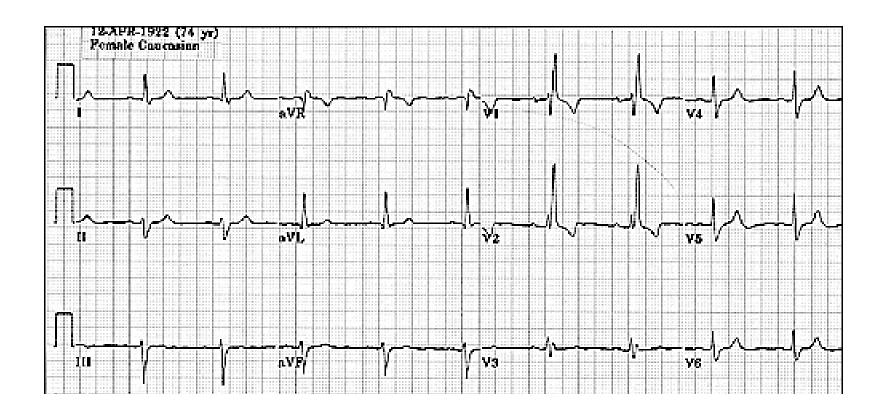
• 65yo

Intermittent syncope when walking

Immediate recovery

No warning signs

Bifascicular block



Work up

Reassurance

Repeat ECG

Holter monitor

Loop recorder

Pacemaker

Recommendations for Permanent Pacing in Chronic Bifascicular Block

CLASS I

- Permanent pacemaker implantation is indicated for advanced second-degree AV block or intermittent third-degree AV block. (Level of Evidence: B) (63–68,101)
- Permanent pacemaker implantation is indicated for type II second-degree AV block. (Level of Evidence: B) (73,75,79,123)
- Permanent pacemaker implantation is indicated for alternating bundle-branch block. (Level of Evidence: C) (124)

CLASS IIa

- Permanent pacemaker implantation is reasonable for syncope not demonstrated to be due to AV block when other likely causes have been excluded, specifically ventricular tachycardia (VT). (Level of Evidence: B) (102–111,113–119,123,125)
- Permanent pacemaker implantation is reasonable for an incidental finding at electrophysiological study of a markedly prolonged HV interval (greater than or equal to 100 milliseconds) in asymptomatic patients. (Level of Evidence: B) (109)
- Permanent pacemaker implantation is reasonable for an incidental finding at electrophysiological study of pacinginduced infra-His block that is not physiological. (Level of Evidence: B) (118)

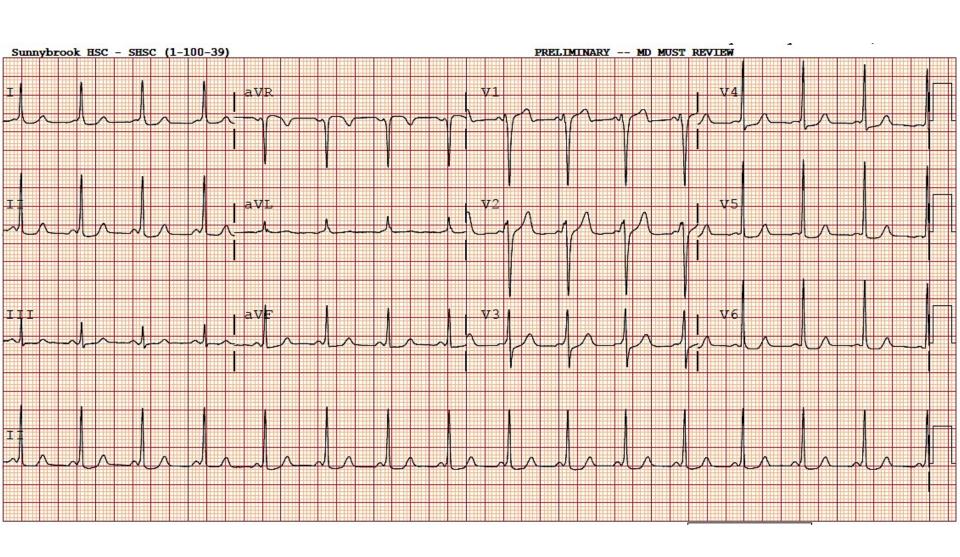
A case of chest pains

- 43yo F
- Chest pains for many years
- 2009 diagnosed with CAD based on ECG changes
- Over the years short episodes of chest pains
- Seen by a cardiologist conservative treatment
- Recent visit to ER with severe chest pains sent home the next day
- What would you do?
 - continue medical treatment
 - stress test
 - add on plavix
 - something else

A case of chest pains

- 43yo F
- Chest pains for many years
- 2009 diagnosed with CAD based on ECG changes
- Over the years short episodes of chest pains
- Seen by a cardiologist conservative treatment
- Recent visit to ER with severe chest pains sent home the next day
- What would you do?
 - continue medical treatment
 - stress test
 - add on plavix
 - something else

ECG of the "chest pains" 43yo F



A case of palpitations

- 43yo F
- Palpitations for many years
- 2009 diagnosed with WPW based on ECG changes
- Over the years short episodes of palpitations
- Seen by an electrophysiologist conservative treatment
- Recent visit to ER with SVT at 200bpm sent home the next day
- What would you do?
 - continue observation
 - Holter monitor
 - add AAD
 - something else

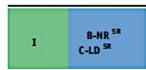
6.2.2. Asymptomatic Patients With Pre-Excitation: Recommendations

Recommendations for Management of Asymptomatic Patients With Asymptomatic Pre-Excitation

COR

LOE

Recommendations



See Online Data Supplements 11 and 12. 1. In asymptomatic patients with pre-excitation, the findings of abrupt loss of conduction over a manifest pathway during exercise testing in sinus rhythm.^{294–297} (Level of Evidence: B-HR) ^{SR} or intermittent loss of pre-excitation during ECG or ambulatory monitoring²⁹⁷ (Level of Evidence: C-LD) ^{SR} are useful to identify patients at low risk of rapid conduction over the pathway. Noninvasive testing has been shown to identify patients at low risk of developing rapid conduction over the accessory pathway and life-threatening ventricular arrhythmias in response to AF. The noninvasive findings that identify a pathway not capable of maintaining rapid conduction during AF include intermittent loss of conduction over the accessory pathway on the resting ECG or during ambulatory monitoring, or abrupt loss of pre-excitation during exercise testing (Figure 16).^{294–297} The ECG should be evaluated closely to make certain the delta wave is truly absent, as accessory pathways, especially left lateral pathways, may demonstrate varying degrees of pre-excitation because of fusion between conduction over the accessory pathway and

IIa B-NR ^{SR}

See Online Data Supplements 11–15. An EP study is reasonable in asymptomatic patients with pre-excitation to risk-stratify for arrhythmic events. ^{254,256,298–301}

value for identifying pathways with life-threatening properties. 294,295,297

through the AV node. This may give the appearance of loss of pre-excitation if the subtle delta wave is not identified. Noninvasive tests have an approximately 90% positive predictive value and 30% negative predictive

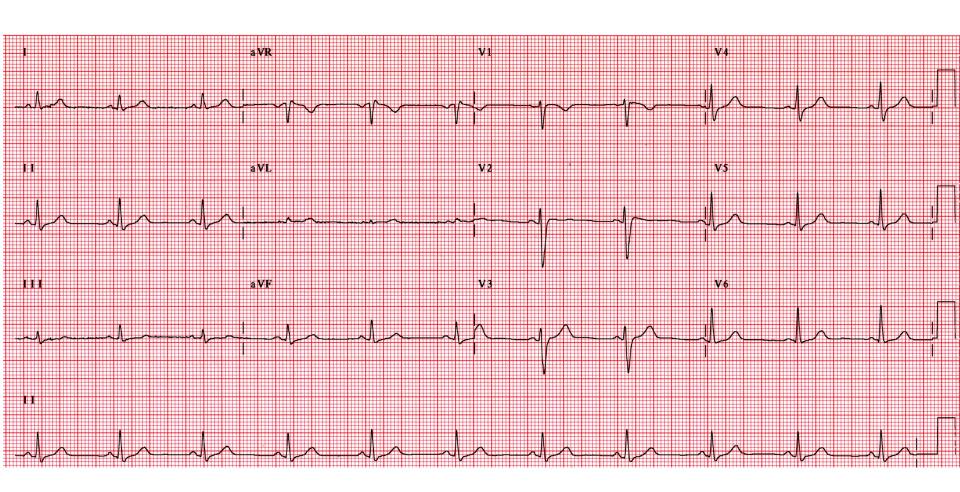
In the absence of symptoms, a clinical priority is identifying accessory pathways at increased risk of arrhythmic events, including rapid conduction during AF and development of life-threatening ventricular arrhythmias, with the most useful findings being the following: an R-R interval < 250 ms between 2 pre-excited complexes during induced AF; the presence of multiple accessory pathways; the ability to induce sustained AVRT; the finding of AVRT precipitating pre-excited AF; and an accessory pathway refractory period < 240 ms. ^{254,256,298,299,301} Malignant arrhythmias correlate more with the EP properties of the accessory pathway than with the presence or absence of symptoms. This approach is supported by the low risk of complications observed in an EP study in which complication rates among 2,169 patients ranged from 0.09% to 1% and included pneumothorax and access site complications. ²⁵⁴

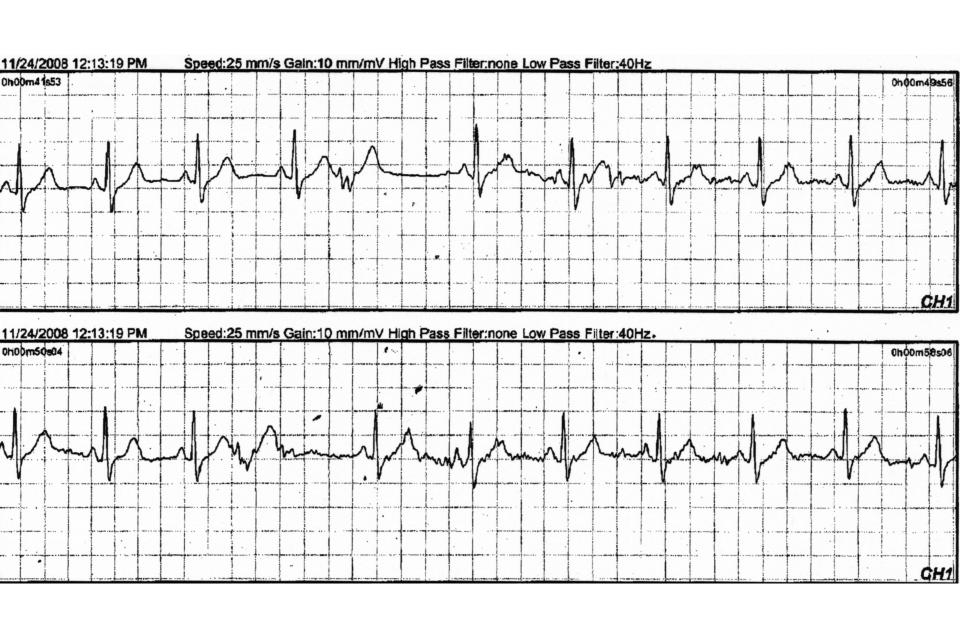
IIa B-NR ^{SR}

See Online Data Supplements 11–15. Catheter ablation of the accessory pathway is reasonable in asymptomatic patients with preexcitation if an EP study identifies a high risk of arrhythmic events, including rapidly conducting pre-excited AF.^{254,302,303}

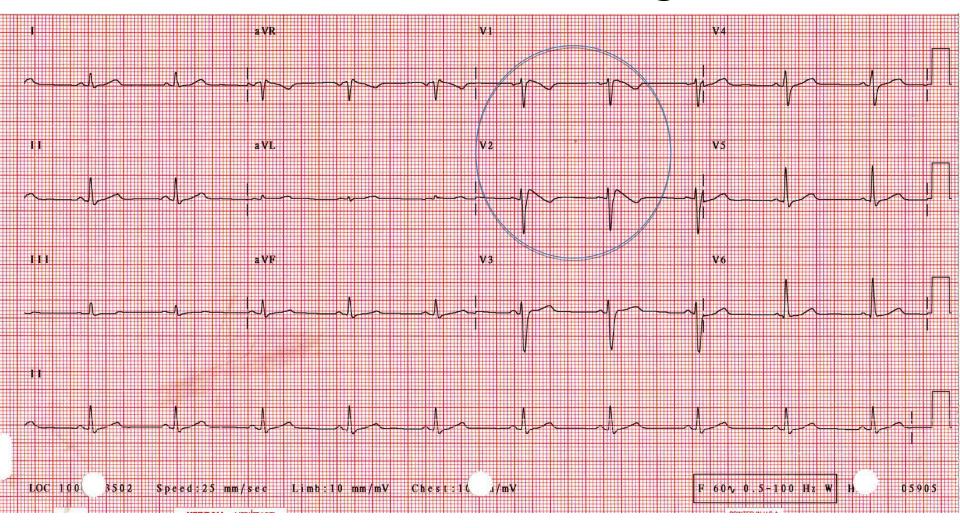
In a large prospective cohort study of 756 asymptomatic patients with close to 8 years of follow-up, 9% of patients developed malignant AF (shortest R-R interval ≤250 ms), and 2% developed ventricular fibrillation. Habition arrhythmias correlated more with high-risk EP properties of the accessory pathway than with the presence or absence of symptoms. Ablation of the accessory pathway(s) in high-risk patients was also examined in 1 RCT that enrolled 76 patients, showing that arrhythmic events (defined as symptomatic SVT, AF, and ventricular fibrillation in this study) occurred in 7% of patients who underwent ablation versus 77% who did not undergo ablation. Another study that examined patients on the basis of whether an ablation was performed reported that none of the asymptomatic patients who had undergone ablation of the accessory pathway developed a malignant arrhythmia during 8 years of follow-up. The risk of complications with ablation ranged from 0.1% (complete heart block) to 0.9% (ablation-induced right bundle-branch block). The risks and benefits of proceeding with ablation of pathways found not to have high-risk characteristics should be discussed thoroughly with patients in advance of the EP procedure.

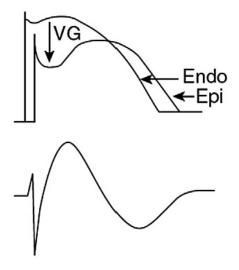
Patient: palpitations, Family history of SCD



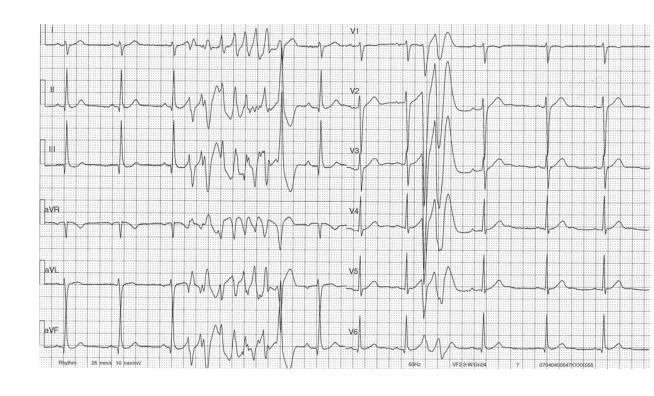


Procainamide challenge test

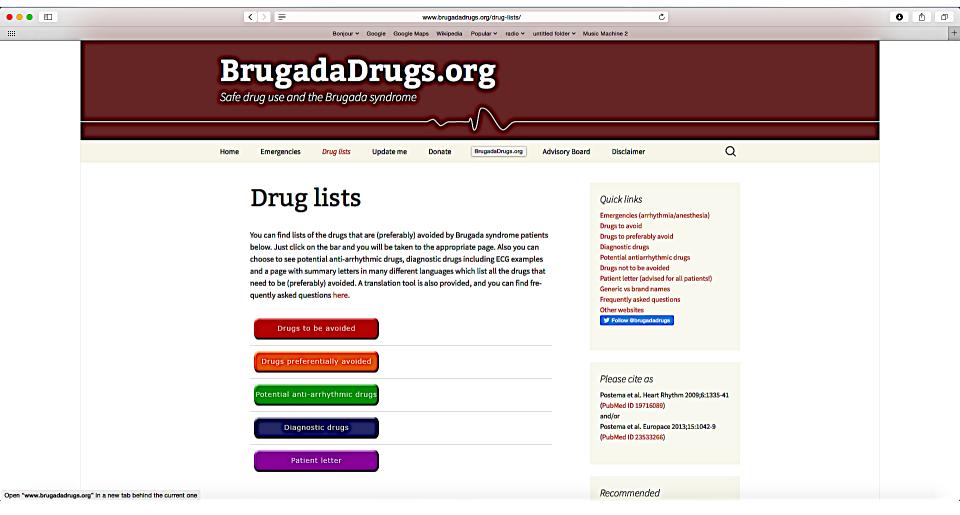




Brugada's pattern



Brugadadrugs.org



"fast" bradycardia – should we worry?

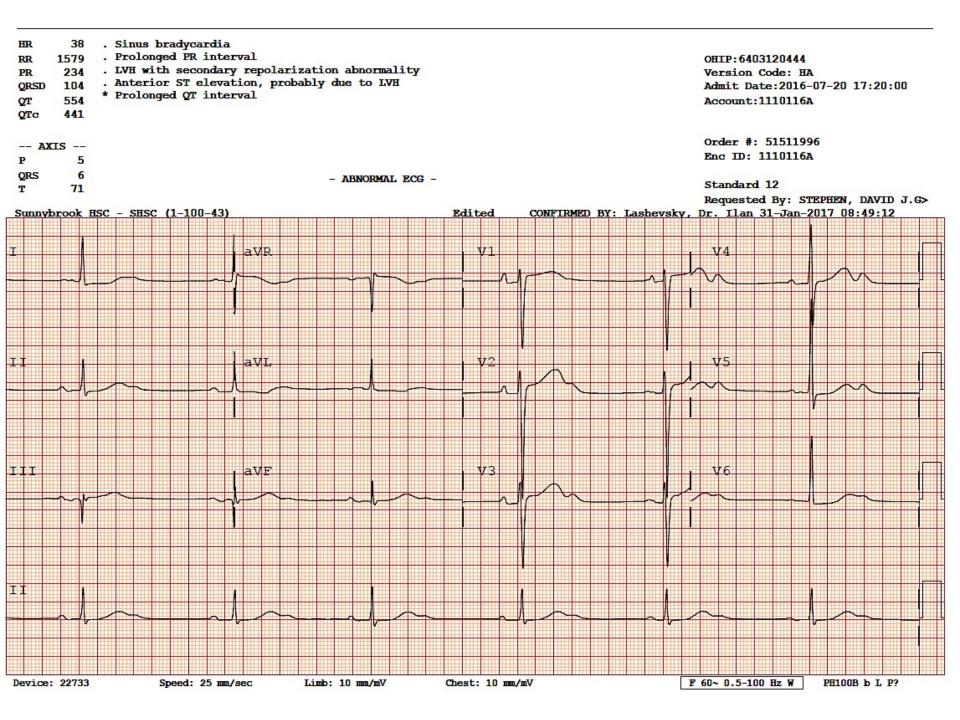


Table 2

Cutoff Values That Best Distinguished Patients With Bradyarrhythmias Complicated by Torsade de Pointes From Patients With Uncomplicated Bradyarrhythmias

Parameter	Cutoff Value	Sensitivity	Specificity	PPV	NPV
T _{peak} -T _{end}	117 ms	96.6%	98.2%	93.3%	99.1%
QTc interval	480 ms	96.6%	92.0%	75.7%	99.0%
QT interval	570 ms	90.0%	86.7%	64.3%	97.0%

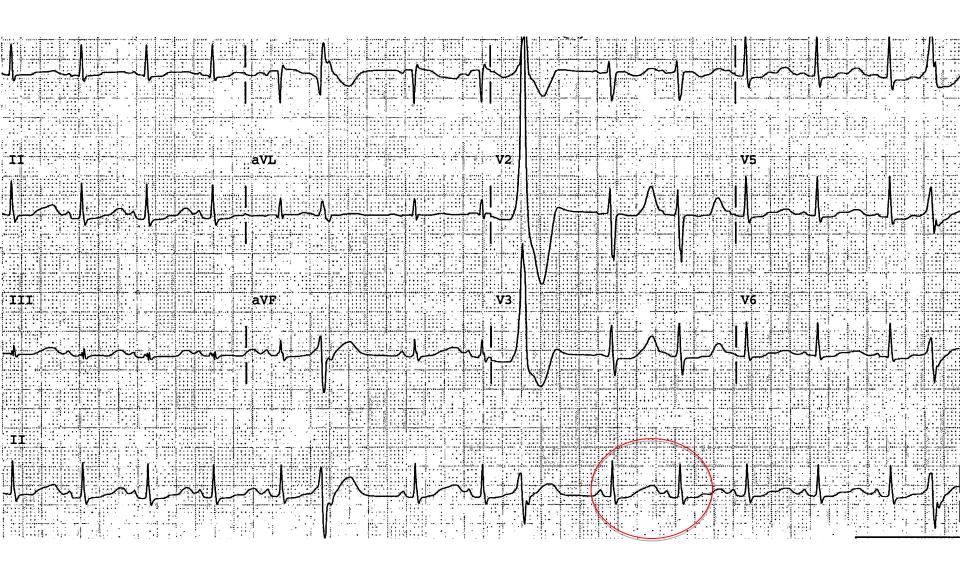
PPV for TdP

• 84% - QT ≥ 510ms + LQT2 morphology

• 94% - Tp-e ≥ 85ms + LQT2 morphology

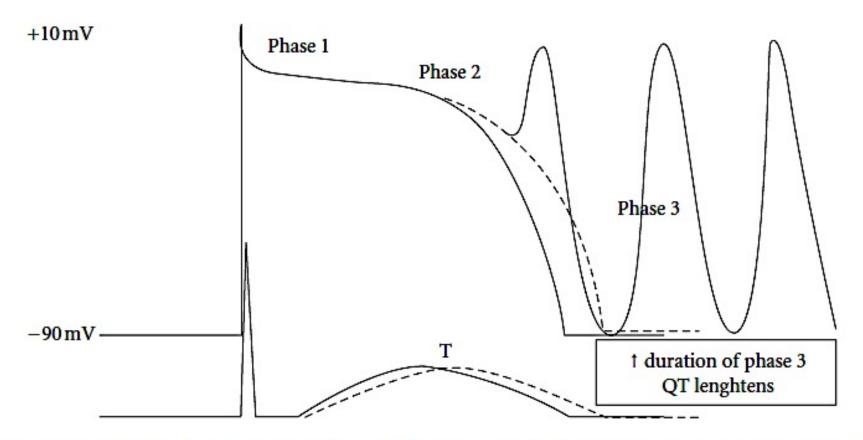
Recurrent syncope

- 68 years F
- Atrial fibrillation
- Sotalol started 2 weeks ago
- Rrecurrent syncope

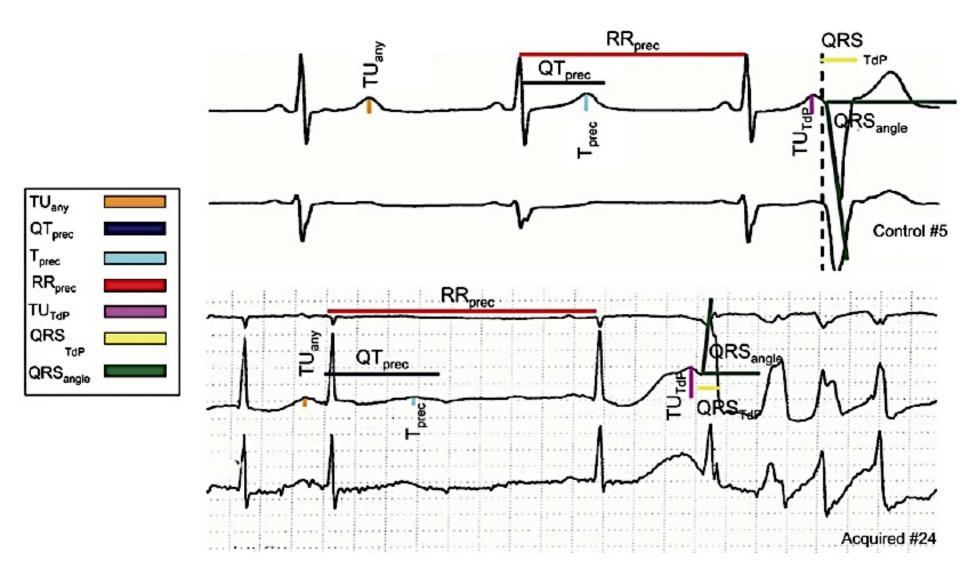


Plausible explanation for her syncopes

- Intermittent AV block
- AF
- TdP
- Vaso vagal
- Loss of balance



Multiple early afterdepolarizations (EADs) from progressively more negative transmembrane potential



what's wrong with me?

