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JOURNAL OF CARDIOVASCULAR ELECTROPHYSIOLOGY, Volume 12, No. 5, May 2001

Incidence and Clinical Significance of Inducible Atrial Tachycardia in Patients with Atrioventricular Nodal Reentrant Tachycardia

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Significance of Atrial Tachycardia. ***Introduction:*** **The purpose of this prospective study was to determine the prevalence and clinical significance of inducible atrial tachycardia in patients undergoing slow pathway ablation for AV nodal reentrant tachycardia who did not have clinically documented episodes of atrial tachycardia.**

Methods and Results: **Twenty-seven (15%) of 176 consecutive patients who underwent slow pathway ablation for AV nodal reentrant tachycardia were found to have inducible atrial tachycardia with a mean cycle length of 351 ± 95 msec. The atrial tachycardia was sustained in 7 (26%) of 27 patients and was isoproterenol dependent in 20 patients (74%). The atrial tachycardia was not ablated or treated with medications, and the patients were followed for 9.7 ± 5.8 months. Six (22%) of the 27 patients experienced recurrent palpitations during follow-up. In one patient each, the palpitations were found to be due to sustained atrial tachycardia, nonsustained atrial tachycardia, recurrence of AV nodal reentrant tachycardia, paroxysmal atrial fibrillation, sinus tachycardia, and frequent atrial premature depolarizations. Thus, only 2 (7%) of 27 patients with inducible atrial tachycardia later developed symptoms attributable to atrial tachycardia.**

Conclusion: **Atrial tachycardia may be induced by atrial pacing in 15% of patients with AV nodal reentrant tachycardia. Because the vast majority of patients do not experience symptomatic atrial tachycardia during follow-up, treatment for atrial tachycardia should be deferred and limited to the occasional patient who later develops symptomatic atrial tachycardia.** *(J Cardiovasc Electrophysiol, Vol. 12, pp. 507-510, May 2001)*

atrial tachycardia, paroxysmal supraventricular tachycardia, slow pathway

Introduction

During electrophysiologic testing in patients with AV nodal reentrant tachycardia, atrial tachycardia sometimes is induced by atrial pacing. In patients without clinically documented episodes of atrial tachycardia, the clinical significance of inducible atrial tachycardia may be uncertain. Therefore, it often is unclear whether treatment of the atrial tachycardia is necessary. The purpose of this prospective study was to determine the prevalence and clinical significance of atrial tachycardia induced in patients undergoing radiofrequency ablation for AV nodal reentrant tachycardia.

Supported in part by the Don Nouse Arrhythmia Research Fund. Dr. Sticherling was supported in part by the German Research Foundation.

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Manuscript received 18 December 2000; Accepted for publication 2 February 2001.

Methods

Characteristics of the Study Population

The subjects of this study were 176 consecutive patients (130 women [74%] and 46 men; mean age 48 ± 17 years) referred to the University of Michigan Medical Center for radiofrequency ablation of paroxysmal supraventricular tachycardia found to be caused by AV nodal reentrant tachycardia. None of these patients had prior ECG recordings suggestive of atrial tachycardia. Twenty-six patients (15%) had underlying heart disease (coronary artery disease 15, valvular heart disease 7, dilated cardiomyopathy 3, and hypertrophic cardiomyopathy 1). Mean left ventricular ejection fraction was 0.58 ± 0.05, and mean left atrial diameter was 36 ± 7 mm.

Electrophysiologic Testing

In all patients, antiarrhythmic drug therapy was discontinued at least five half-lives before the procedure. After obtaining informed consent and with the patients in a fasting state, three quadripolar electrode catheters were inserted into a femoral vein and positioned in the high right atrium, His-bundle position, and right ventricular apex. Several ECG leads and the intracardiac electrograms were displayed

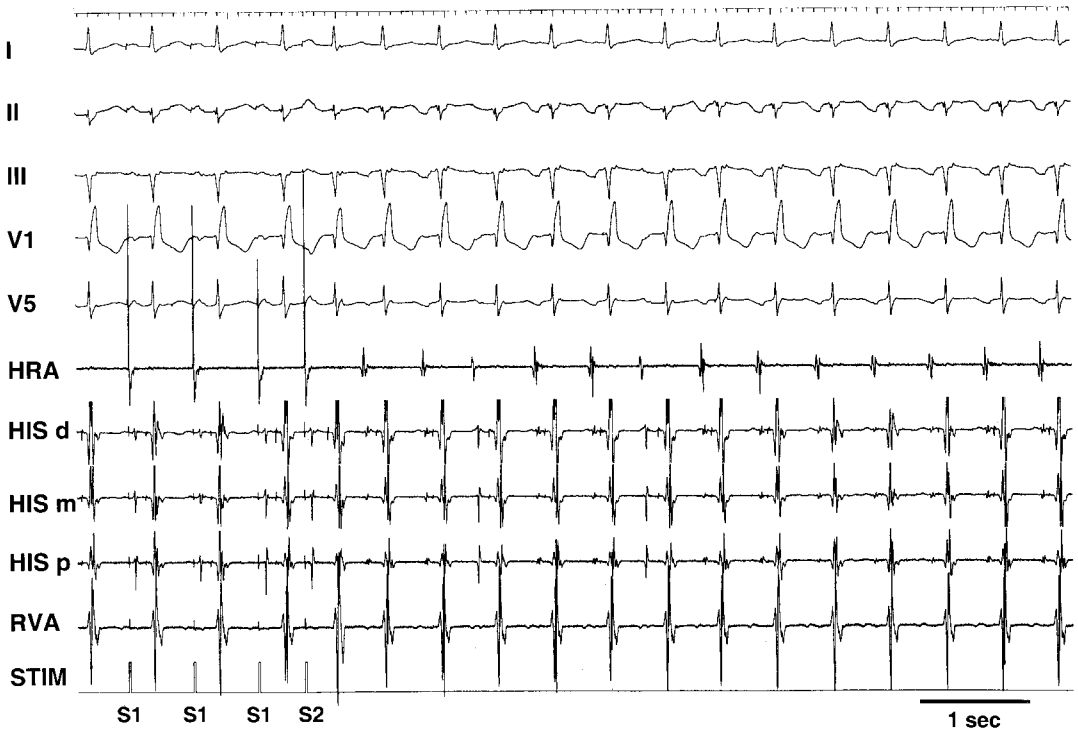

Figure 1. *Example of sustained atrial tachycardia (cycle length 540 msec, duration 40 sec) induced by programmed atrial stimulation with a single extrastimulus. Displayed are leads I, II, III, V_1, and V_5, an electrogram recorded at the high right atrium (HRA), the distal (d), medial (m), and proximal (p) His-bundle electrograms (HIS), right ventricular electrogram (RVA), and stimulus marker (STIM).*

on an oscilloscope and recorded on optical disk (EPMed-Systems, Mount Arlington, NJ, USA). Pacing was performed at twice the pacing threshold using a programmable stimulator (EPMedSystems).

Study Protocol

During the electrophysiologic procedure, incremental atrial and ventricular pacing and atrial and ventricular programmed stimulation with up to three extrastimuli were performed to induce tachycardia. If these maneuvers did not result in arrhythmia induction, they were repeated during infusion of isoproterenol 1 to 4 μg/min. AV nodal reentrant tachycardia was induced in all patients. After slow pathway ablation, which was successful in all patients, the same pacing maneuvers were used to confirm successful elimination of the AV nodal reentrant tachycardia.

Because short episodes of inducible atrial tachycardia are often likely to be a nonspecific response to atrial stimulation, for the purposes of the present study, inducible atrial tachycardia was defined as a monomorphic atrial tachycardia that had cycle length <600 msec and duration >5 seconds, and was reproducibly inducible (Fig. 1). The following criteria were used to diagnose atrial tachycardia: (1) induction by an atrial depolarization that blocked in the AV node; (2) induction independent of a critical atrial-His interval; (3) eccentric atrial activation; (4) a variable VA relationship during tachycardia; (5) failure to terminate the tachycardia by ventricular pacing without advancing the atrial electrograms; (6) a "V-A-A" response upon cessation of pacing after entrainment with ventricular pacing[1]; and (7) when the tachycardia was nonsustained, consistent spontaneous termination in the absence of AV block. The atrial tachycardia was considered sustained if its duration was >30 seconds.

None of 27 patients who had inducible atrial tachycardia initially were treated for the tachycardia with either radiofrequency catheter ablation or antiarrhythmic drug therapy. However, 6 (22%) of these patients were treated with verapamil, atenolol, or flecainide for hypertension, inappropriate sinus tachycardia, or paroxysmal atrial fibrillation.

The patients were seen in an outpatient clinic 4 months after the electrophysiologic procedure and were told to contact one of the authors in the event of a recurrence of palpitations. In the event of recurrent symptoms, the patient was provided with a continuous-loop event monitor to document the rhythm at the time of symptoms. Mean duration of follow-up was 9.7 ± 5.8 months (range 1.1 to 19).

Statistical Analysis

Continuous variables are expressed as mean ± 1 SD. Comparisons were performed with Student's *t*-test or the Fisher exact test. $P < 0.05$ was considered significant.

Results

Prevalence of Induced Atrial Tachycardia

Among the 176 patients, 27 patients (15%) had inducible atrial tachycardia. Age, gender, ejection fraction, left atrial diameter, and prevalence of structural heart disease did not differ significantly between patients with and patients without inducible atrial tachycardia (Table 1).

TABLE 1
Clinical Characteristics of Patients With and Without Inducible Atrial Tachycardia

	Inducible Atrial Tachycardia	No Inducible Atrial Tachycardia	P Value
No. of patients	27 (15%)	149 (85%)	
Age (years)	49 ± 15	48 ± 18	0.9
Ejection fraction	0.59 ± 0.03	0.58 ± 0.06	0.3
Left atrial diameter (mm)	36 ± 5	36 ± 7	0.8
Female gender	21 (78%)	109 (73%)	0.4
Heart disease	4/27 (15%)	22/149 (15%)	0.6

Continuous values are expressed as mean ± SD.

Characteristics of Induced Atrial Tachycardia

Among the 27 patients with inducible atrial tachycardia, the tachycardia was nonsustained in 20 patients (74%) and was inducible only during isoproterenol infusion in 20 patients (74%). Mean tachycardia cycle length was 350 ± 117 msec for the sustained episodes and 352 ± 89 msec for the nonsustained episodes (P = 1.0). The sustained episodes had a mean duration of 107 ± 96 seconds, and the nonsustained episodes had a mean duration of 10 ± 8 seconds (P < 0.001). The sustained episodes were terminated by atrial overdrive pacing in 2 of 7 patients and terminated spontaneously after 30 seconds in the remaining patients.

Atrial tachycardia was induced by right atrial overdrive pacing in 8 patients, right ventricular overdrive pacing in 2, programmed atrial stimulation with a single extrastimulus in 11, programmed atrial stimulation with two extrastimuli in 5, and programmed ventricular stimulation with a single extrastimulus in 1.

Recurrent Palpitations

Six (22%) of the 27 patients with inducible atrial tachycardia had recurrent symptoms of palpitations after successful ablation of AV nodal reentrant tachycardia. The palpitations occurred at 108 ± 123 days of follow-up (range 14 to 350) and were described as being similar to the palpitations experienced before the ablation procedure by 1 patient and as different from the palpitations experienced before the ablation procedure by 5. Four patients had palpitations lasting >30 seconds, and 2 patients had episodes lasting <30 seconds.

ECG documentation with a continuous-loop event recorder demonstrated a probable atrial tachycardia in two patients (sustained in one and nonsustained in the other) (Fig. 2). In both cases, the documented atrial tachycardia had a cycle length similar to the induced episode (540 vs 480 msec, and 440 vs 460 msec). One patient had a recording consistent with AV nodal reentrant tachycardia (confirmed by repeat electrophysiologic procedure), 1 had inappropriate sinus tachycardia, 1 had paroxysmal atrial fibrillation, and 1 had frequent premature atrial complexes (Table 2).

Among the 27 patients with inducible atrial tachycardia, the tachycardia was sustained and inducible in the absence of isoproterenol in 4 patients. These characteristics were not predictive of recurrent palpitations after slow pathway ablation. Three of these 4 patients remained asymptomatic during follow-up, and only 1 (25%) experienced symptomatic sustained atrial tachycardia during follow-up. Among the 23 patients in whom inducible atrial tachycardia was not sustained and isoproterenol dependent, 1 patient (4%) experienced symptomatic atrial tachycardia (nonsustained) during follow-up (P = 0.3). The patient with sustained atrial tachycardia was treated with propafenone, and the patient with nonsustained atrial tachycardia was treated with atenolol. Both patients have remained asymptomatic with this therapy.

Discussion

Main Findings

The results of this study demonstrate that atrial tachycardia may be inducible in 15% of patients who undergo radiofrequency ablation of AV nodal reentrant tachycardia and who do not have prior clinical documentation of atrial tachycardia. The majority of the episodes of inducible atrial tachycardia induced in this study were nonsustained and isoproterenol dependent. Fewer than 10% of the patients in whom atrial tachycardia was induced later experienced symptoms attributable to atrial tachycardia. This observation suggests that inducible atrial tachycardia, even when monomorphic and reproducibly inducible, often may be a nonspecific finding that does not have clinical significance. Therefore, when atrial tachycardia is inducible in a patient with AV nodal reentrant tachycardia who does not have documentation of prior atrial tachycardia, therapy directed at the atrial tachycardia should be deferred and limited to the occasional patient who later develops symptomatic atrial tachycardia.

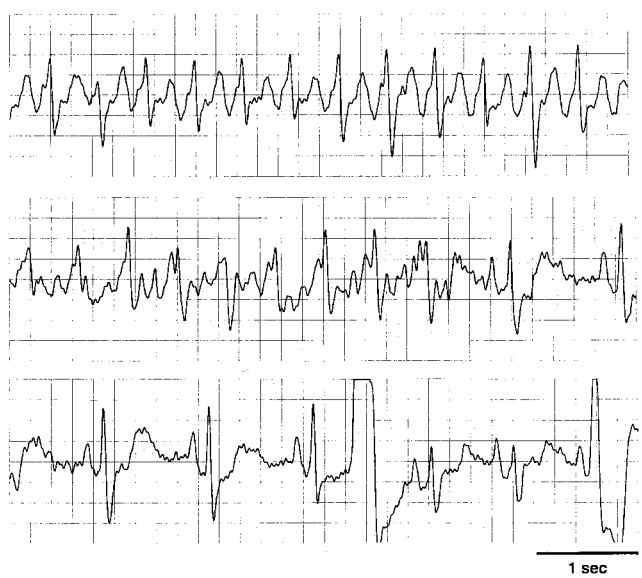

Figure 2. *Recording obtained with a continuous-loop event recorder at the time of palpitations 20 days after successful slow pathway ablation in the same patient who had the inducible atrial tachycardia shown in Figure 1. The cycle length of this tachycardia is 480 msec, and the tracing is suggestive of an atrial tachycardia. The recordings are continuous, with the top strip displaying probable atrial tachycardia, the middle strip showing spontaneous conversion to sinus rhythm, and the bottom strip showing the artifact caused by activation of the recorder by the patient.*

TABLE 2
Clinical Features of the Six Patients with Inducible Atrial Tachycardia who Experienced Palpitations after Successful Slow Pathway Ablation

Pt. No.	Age (years)/ Gender	Heart Disease	Induction Mode	Isoproterenol Dependent	CL (msec)	Sustained	Cause of Palpitations	Onset of Palpitations (Days after RFA)
1	22/F	None	RAOD	Yes	340	No	IST	36
2	37/M	None	RAOD	Yes	330	No	PAF	96
3	52/F	None	RAOD	No	290	No	AVNRT	63
4	60/M	MVR	PAS	Yes	460	No	AT	350
5	48/F	None	PAS	Yes	460	No	PAC	100
6	71/M	None	PAS	No	540	Yes	AT	14

AT = atrial tachycardia; AVNRT = AV nodal reentrant tachycardia; CL = cycle length; IST = inappropriate sinus tachycardia; MVR = mitral valve repair; PAC = premature atrial complex; PAF = paroxysmal atrial fibrillation; PAS = programmed atrial stimulation; RAOD = right atrial overdrive pacing; RFA = radiofrequency ablation.

Tachycardia Substrate

AV nodal reentrant tachycardia and accessory pathway tachycardias have a clearly defined anatomic substrate that gives rise to reentrant tachycardias and are amenable to radiofrequency catheter ablation.[2] However, in the case of atrial tachycardia, the underlying tachycardia mechanism may be reentry, abnormal automaticity, or triggered activity.[3] The atrial tachycardias induced in the present study were inducible by pacing maneuvers, consistent with either reentry or triggered activity. These types of atrial tachycardia may not require a specific anatomic substrate, which explains why they may often represent a nonspecific finding.

Previous Studies

Previous studies showed that symptomatic palpitations after slow pathway ablation for AV nodal reentrant tachycardia, or after accessory pathway ablation, occur in 32% to 36% of patients.[4,5] Atrial tachycardia was found to be the cause of recurrent palpitations in <5% of the patients who had recurrent symptoms. The low incidence of recurrent palpitations attributable to atrial tachycardia after catheter ablation of AV nodal reentrant tachycardia or of an accessory pathway in these two prior studies is consistent with the results of the present study.

In this series, 7 (26%) of the 27 patients who underwent successful slow pathway ablation were treated during follow-up with an antiarrhythmic drug for paroxysmal atrial fibrillation, atrial tachycardia, or inappropriate sinus tachycardia. This is similar to the results of a prior study in which 17% of 398 patients who underwent successful accessory pathway ablation were treated with an antiarrhythmic medication during follow-up.[6]

Limitations

Some of the patients who had inducible atrial tachycardia may have had asymptomatic recurrences of atrial tachycardia during follow-up. Therefore, the predictive value of inducible atrial tachycardia may have been underestimated. However, if episodes of atrial tachycardia during follow-up are asymptomatic, therapy is not necessary and the results of this study remain valid for clinically significant episodes of atrial tachycardia.

A second limitation is that six patients were treated with an antiarrhythmic drug for hypertension or for an arrhythmia other than atrial tachycardia. It is possible that drug therapy in these patients suppressed symptomatic atrial tachycardia that otherwise may have occurred.

Conclusion

Radiofrequency catheter ablation of atrial tachycardia has a lower success rate than catheter ablation of AV nodal reentrant tachycardia or accessory pathways,[2] a higher recurrence rate after ablation (5% to 18%),[7,8] and a higher potential for multifocality.[2] Furthermore, mapping of atrial tachycardias is tedious when the episodes of induced tachycardia are nonsustained.[9] For these reasons, and because the majority of patients with inducible atrial tachycardia after slow pathway ablation remain asymptomatic, we recommend deferring catheter ablation or drug therapy for the inducible atrial tachycardia, unless there is documentation that the atrial tachycardia is clinically significant.

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