

Role of Catheter-Induced Mechanical Trauma in Localization of Target Sites of Radiofrequency Ablation in Automatic Atrial Tachycardia

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Objectives. We compared the efficacy of two different mapping techniques in identifying the ablation site for atrial tachycardia. Moreover, we evaluated the additive positive predictive value of mechanical interruption of atrial tachycardia to reduce the number of ineffective radiofrequency applications.

Background. Radiofrequency catheter ablation has been suggested as a highly effective technique to treat drug-resistant atrial tachycardia. However, irrespective of the mapping technique utilized, success was most often achieved with a large number of radiofrequency applications.

Methods. Forty-five patients with atrial tachycardia underwent radiofrequency catheter ablation. Mapping techniques included identification of earliest atrial activation and pace-mapping concordant sequence.

Results. Atrial tachycardia was successfully treated in 42 (93.3%) of 45 patients with a mean of 3.9 radiofrequency pulses/patient. An interval between the onset of the intracavitary atrial deflection and the onset of the P wave during atrial tachycardia

(AP interval) ≥ 30 ms ($p < 0.001$) and pace-mapping concordant sequence ($p = 0.01$) were all significant predictors of outcome. An AP interval ≥ 30 ms and a pace-mapping concordant sequence were highly sensitive (92.8%, 95% confidence interval [CI] 80.5% to 98.5%; 85.7%, 95% CI 71.5% to 94.6%, respectively) but less specific (47.8%, 95% CI 37.9% to 58.2%; 36.8%, 95% CI 27.6% to 47.2%, respectively) in identifying the site of ablation. By using atrial tachycardia mechanical interruption combined with the AP interval > 30 ms or the pace-mapping concordant sequence, we obtained a specificity of 76.5% (95% CI 66.4% to 84.0%) and 73.5% (95% CI 63.2% to 81.4%), respectively, and a positive predictive value of 49.2% and 44.6%, respectively.

Conclusions. An AP interval ≥ 30 ms and a pace-mapping concordant sequence were reliable mapping features for predicting the outcome of the ablation procedure. Mechanical interruption of atrial tachycardia improved the specificity and positive predictive value of these two mapping techniques.

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Automatic atrial tachycardia is an uncommon form of supraventricular arrhythmia. This form of tachycardia is frequently associated with progressive cardiac dilation but is potentially reversible with control of the arrhythmia (1,2). Because medical treatment is of limited efficacy (3-6), and surgery, although effective, has intrinsic limitations (2,7-11), radiofrequency ablation of primary atrial tachycardia has been suggested as a highly effective technique with a low incidence of side effects. Because the radiofrequency-induced lesion is of limited size, identification of the exact site of the arrhythmogenic focus is crucial. In several studies showing a high success rate of radiofrequency ablation in interrupting the arrhythmia (80% to 100%), this was achieved with a large number of radiofrequency applications (12-16). When radiofrequency is

applied to the atrial wall, a transmural necrosis is produced with the formation of a well-organized fibrotic scar (17), and a high number of such scars may represent a new arrhythmogenic substrate. Improving the mapping technique may further increase the success rate and reduce the number of ineffective radiofrequency applications. The aim of this study was to assess and compare the efficacies of two different mapping techniques (earliest atrial activation, activation sequence during atrial pacing) in precisely identifying the site of the anatomic substrate and to correlate them with the probability of radiofrequency ablation success. Further, we evaluated the additive positive predictive value of mechanical interruption of atrial tachycardia in order to reduce the number of ineffective radiofrequency applications.

Methods

Between December 1991 and January 1995, 45 patients referred to our center for ablation of automatic atrial tachycardia refractory to antiarrhythmic medical treatment underwent intracardiac electrophysiologic study and radiofrequency

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catheter ablation (25 male, 20 female; mean [\pm SD] age 29.4 ± 11.4 years, range 7 to 64). As for the underlying cardiac condition, 22 patients were free of any organic cardiac disease, 17 presented with dilated cardiomyopathy, 3 had valvular and 3 congenital heart disease (one "ostium secundum" atrial septal defect, one corrected transposition of the great vessels one aneurysm of interatrial septum). The diagnosis of atrial tachycardia had been made 3 months to 18 years before the admission, and all patients had been treated with two to seven different drugs (mean 3.8 ± 2.7).

Electrocardiographic and electrophysiologic patterns.

Atrial tachycardia associated with enhanced automaticity (8,18-24) was diagnosed initially by standard surface electrocardiogram and/or Holter monitoring. In all patients the following criteria were met: 1) the P wave axis and configuration were different during tachycardia as compared to sinus rhythm; 2) the PR interval was influenced by rate of tachycardia; 3) an atrioventricular (AV) block could be induced occasionally by vagal maneuvers or by rapid injection of adenosine (6 to 12 mg) (2 patients) or verapamil (5 to 10 mg) (43 patients); 4) the presence of warming-up and cooling-down phenomena.

The electrophysiologic patterns used for the diagnosis of automatic atrial tachycardia were: 1) failure in consistently starting or terminating the atrial tachycardia by an atrial extrastimulus; 2) induction of atrial tachycardia during a programmed atrial stimulation with no relation to intraatrial or atrioventricular delay; 3) warming-up and cooling-down phenomena; 4) an atrial activation sequence different from that observed during sinus rhythm, with an AH interval correlated directly to the tachycardia cycle so that the faster the rate the longer the interval; 5) failure of atrial pacing in initiating, entraining or terminating tachycardia, although overdrive suppression might be observed; 6) the onset of AV block that does not interfere with the tachycardia cycle.

Mapping techniques. All patients underwent electrophysiologic study and catheter ablation in a single session, in the fasting state, after discontinuation of pharmacologic therapy for at least 5 half-lives of drugs. Children under the age of 10 received general anesthesia with fentanyl (0.1 to 0.3 mg) and propofol (2 mg/kg followed by 2 to 8 mg/kg per h, intravenously); in older patients no sedation was used (11). All patients received sodium heparin (100 UI/kg up to 5,000 UI intravenously) as a bolus, followed by a continuous infusion of 20 UI/kg per h, with monitoring of activated clotting time. In some patients, to convert an intermittent atrial tachycardia into a stable rhythm, the electrophysiologic study was carried out under isoproterenol infusion (0.01 to 0.03 μ g/kg per min).

At least five quadrupolar or hexapolar standard 6F catheters with interelectrode spacing of 2 or 5 mm were inserted through the left or right femoral vein and the left subclavian vein. The electrodes were positioned at the upper right atrium, middle right atrium, atrioventricular junction, right ventricular apex and coronary sinus. Mapping and endocardial ablation of the substrate were carried out using two steerable 7F quadrupolar catheters with a 4-mm tip and an interelectrode spacing

of 2 mm. Right atrial sites were reached through the inferior or superior vena cava, and those in the left atrium were reached through a transaortic, transmitral retrograde approach or, in only two cases, through a patent foramen ovale.

Leads I, III, V₁, and the intracardiac electrograms were simultaneously recorded by a Midas (PPG, Hellige) recorder in a bipolar fashion at a paper speed of 100 or 200 mm/s and filtered between 30 and 500 Hz. Electrical stimulation was delivered by a programmable stimulator (Medtronic, Model 5238) with pulse duration of 2 ms and an amplitude twice the diastolic threshold.

Catheter positions were examined under monoplane fluoroscopy using the following projections: anteroposterior, left anterior oblique (45°) and right anterior oblique (45°).

To identify the ablation site for atrial tachycardia, we used two different mapping techniques: 1) earliest atrial activation, and 2) pace mapping.

Earliest atrial activation. This technique consists of a point-to-point exploration of the atrium, calculating the interval between the beginning of the intracavitary atrial deflection, recorded by the distal pair of the exploring catheter electrodes, and the beginning of the P wave on the surface electrocardiogram (AP interval) during atrial tachycardia. The site characterized by the longest AP interval, with an AP interval ≥ 30 ms, was considered as the ideal atrial ablation site (Fig. 1). To locate right-sided ablation sites, two steerable catheters were used. Once the first catheter identified a site of early atrial activation, it was left in place and used as a reference, while the second catheter was moved until an earlier site of atrial activation was recorded. Catheters were alternatively moved until an atrial activation earlier than the one recorded by the reference catheter could not be found. For left-sided atrial foci we performed point-to-point exploration of the atrium with only one catheter, looking for the longest AP interval.

Pace mapping. In this technique, once any early atrial activation site was located, we stimulated the atrial wall from the distal pair of exploring catheter electrodes to a rate higher than the tachycardia's and compared the morphology of the P wave and the sequence of endocavitary atrial activation during atrial pacing with that of atrial tachycardia (Fig. 2). For the study of the paced activation sequence, we used as reference sites the high right atrium, midright atrium, right low septal atrium and atrium in coronary sinus. The relative time intervals were recorded, and the map of the paced activation sequence was compared with the map of the spontaneous activation sequence during tachycardia. The ablation catheter was then positioned where the pacing more closely reproduced the spontaneous activation sequence and the inter- and intraatrial conduction intervals with a maximal error range of 5 ms.

Before radiofrequency delivery, attempts to induce mechanical block were made intentionally by applying a slight pressure with the tip of the mapping catheter on those endocardial sites identified as ideal ablation sites by the two mapping techniques described above (Fig. 3). Pressure was applied through the plunger steering mechanism of the ablation catheter while avoiding inappropriate "whiplash" move-

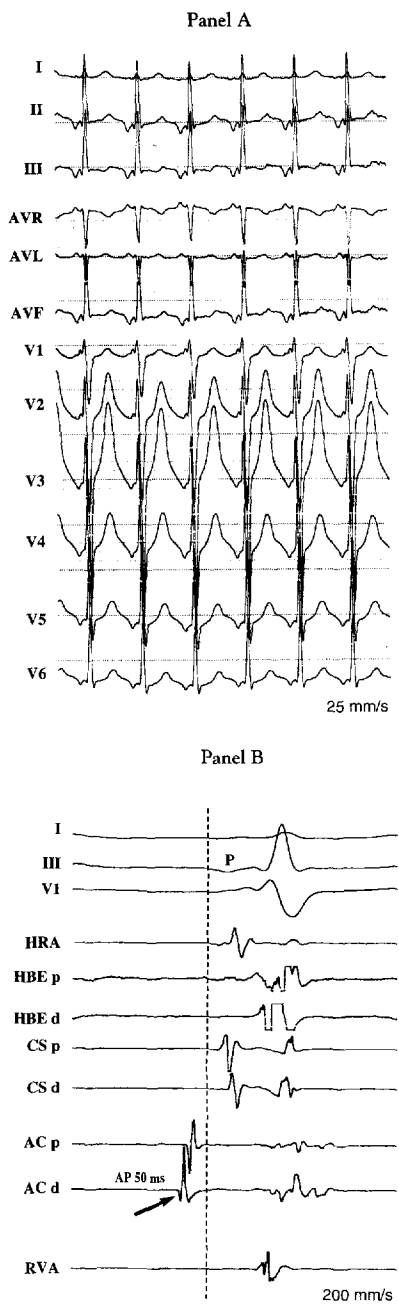


Figure 1. Patient 16. **Panel A**, Surface electrocardiographic recordings during ectopic atrial tachycardia. **Panel B**, Surface and intracardiac electrograms recorded during mapping of a left-sided ectopic atrial focus. The distal electrode pair of the mapping/ablation catheter is located at the site where successful ablation was achieved. Local atrial activation (arrow) precedes the onset of the P wave (dashed line) by 50 ms. AC = ablation catheter; CS = coronary sinus; d = distal electrode pair; HBE = His bundle; HRA = high right atrium; p = proximal electrode pair; RVA = right ventricular apex.

ments and lateral torsion. If catheter dislodgement or any change in atrial potential occurred, pressure was stopped.

For each of the ablation sites considered ideal where radiofrequency was delivered, we analyzed the AP interval, the activation sequence during atrial tachycardia and atrial pacing and the

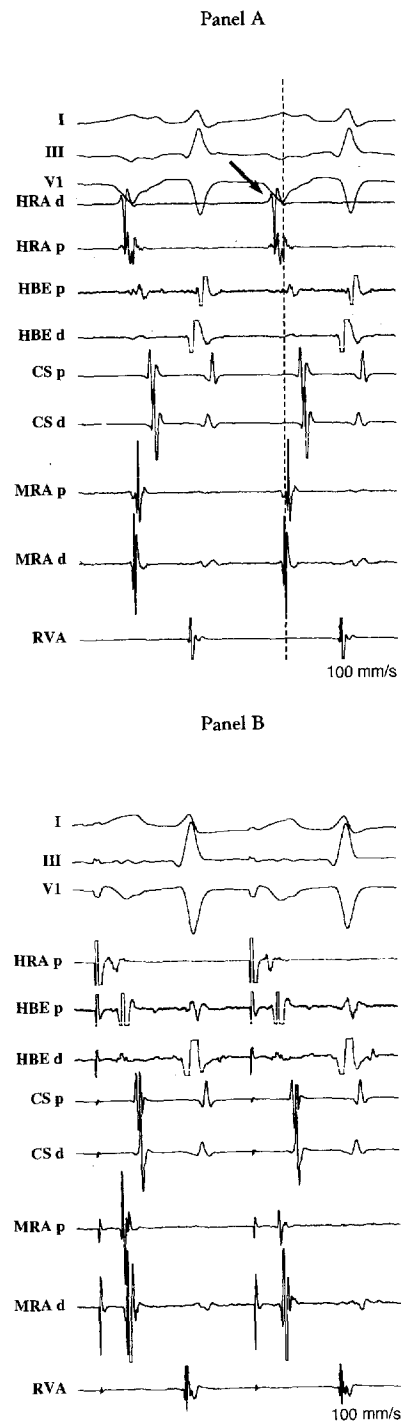


Figure 2. Patient 14. Surface electrocardiographic and intracardiac electrographic recordings during spontaneous tachycardia (**Panel A**) and during atrial pacing from the successful site of ablation (**Panel B**), showing the same intracardiac sequence. During atrial tachycardia, local atrial activation (arrow) precedes the onset of the P wave (dashed line) by 30 ms. MRA = middle right atrium; other abbreviations as in Figure 1.

atrial tachycardia mechanical interruption during catheter placement. The characteristics of the ablation sites were analyzed independently by three of the authors (C.P., A.D.S., G.S.). In

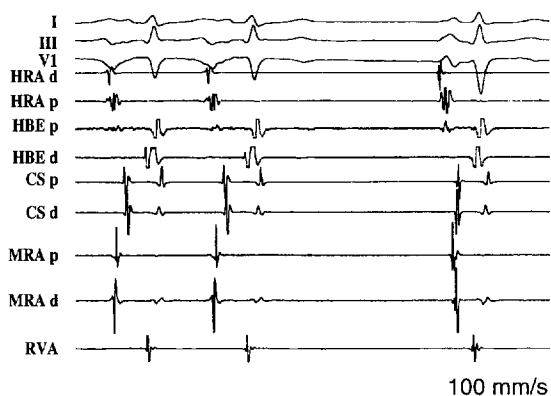


Figure 3. Patient 14. Surface electrocardiographic and intracardiac electrographic recordings at the site where the pace-mapping was concordant. When a slight pressure was applied with the ablation catheter on the endocardial site, the interruption of the tachycardia was observed. Abbreviations as in Figure 1.

patients successfully ablated, both failure and success sites were considered. In patients in whom catheter ablation failed, the first four sites where radiofrequency was applied were analyzed.

Ablation procedure. Radiofrequency energy was delivered as a continuous, unmodulated sine wave at 500 kHz (RFG 3C Radionics, Inc.) between the distal electrode of the ablation catheter and a large skin electrode on the posterior chest. Each radiofrequency application was monitored by the following parameters: current (mA), power (W), voltage (V), duration (s) and impedance (ohms). The radiofrequency current was applied in a power range from 15 to 25 W. If atrial tachycardia did not terminate during the first 8 s of energy application, radiofrequency discharge was discontinued, and further attempts to localize an effective target site were made. Simple decrease or increase of the tachycardia cycle (25) or the appearance of a single sinus beat was considered a predictor of success, and radiofrequency was continued for additional 10 s. In the event of an impedance rise, the ablation catheter was removed, and the distal electrode was wiped clean of blood clots. If atrial tachycardia was terminated during the first 10 s, the energy was applied for a total of 30 to 60 s. In the last 41 patients, a second 15-s radiofrequency pulse was delivered at successful target sites to minimize the possibility of recurrence of atrial tachycardia (15,26). In the last 13 patients, we used temperature monitoring to avoid a rise in impedance.

Postablation and follow-up evaluation. All patients underwent control electrophysiologic study under basal conditions and during isoproterenol infusion, 60 min after the ablation procedure. Before hospital discharge (usually 24 h after the ablation procedure), an electrocardiogram (ECG), a chest radiograph and an echocardiogram were obtained. During the follow-up period all patients underwent an electrocardiogram every 3 months and a 24-h Holter monitoring and an echocardiogram every 6 months.

Statistical analysis. All the variables are expressed as mean value \pm SD. Comparison between successful and unsuccessful sites was made by Student or chi-square tests (28). A

probability value <0.05 was considered statistically significant. For each of the mapping techniques we determined specificity, sensitivity and positive predictive value. The 95% confidence intervals for sensitivity and specificity were calculated.

Results

Atrial tachycardia characteristics. The mean cycle of atrial tachycardia was 334.6 ± 48.1 ms (range 240 to 480). In 37 patients atrial tachycardia was conducted to the ventricle at a rate of 1:1; various degrees of AV block were noted in the remaining 8 patients (Table 1). Isoproterenol infusion was used to induce and sustain tachycardia in 11 of 45 patients. In patient 17 the electrophysiologic study showed the coexistence of atrioventricular nodal reentry tachycardia. This patient underwent, during the same procedure, ablation of the atrial ectopic focus and of the slow pathway of the nodal reentry circuit.

Ablative procedure results. We obtained successful ablation of atrial tachycardia in 42 (93.3%) of 45 patients. The procedure was ineffective in eliminating atrial tachycardia in 3 patients (6.7%): In these patients the atrial focus was located in the high posterior roof of the right atrium near the sinus node, right posteroseptal and left inferoposterior region, respectively. In two of these patients a partial control of the arrhythmia was obtained by pharmacologic therapy; in the third patient with dilated cardiomyopathy (ejection fraction 18%), the AV junction was ablated, and a permanent VVIR pacemaker was implanted. The mean duration of ablation was 165.2 ± 48.5 min with a mean fluoroscopy time of 36.4 ± 19.3 min. The mean number of radiofrequency pulses needed to interrupt the atrial tachycardia was 3.9 ± 3.4 (range 1 to 16). The time between the onset of radiofrequency delivery and the termination of atrial tachycardia ranged between 1.2 and 4.6 s (mean of 2.4 ± 2.1) (Fig. 4, Table 1).

Electrophysiologic characteristics of ablation sites. Table 2 shows the characteristics of ablation sites. The AP interval ≥ 30 ms ($p < 0.001$) and the concordance of the P wave and of the atrial activation sequence during atrial tachycardia and pace-mapping ($p = 0.01$) were both significant predictors of success. In Table 3 we compared the sensitivity, specificity and positive predictive value of these mapping techniques. All of them showed a good sensitivity to localize the ablation site, with values of 92.8% for AP interval ≥ 30 ms and 85.7% for pace-mapping. However, specificities of AP interval ≥ 30 ms and pace-mapping were 47.8% and 36.8%, respectively. The mechanical interruption of atrial tachycardia was observed in a significantly greater proportion at successful sites (76.2%) than at unsuccessful sites (28.7%) ($p < 0.001$). By using mechanical interruption of atrial tachycardia combined with AP interval ≥ 30 ms or pace-mapping, we obtained a sensitivity of 73.8% and 69.0% and a specificity of 76.5% and 73.5%, respectively. By using the two mapping techniques simultaneously with successful mechanical interruption of atrial tachycardia, we had a specificity of 93.4%, and therefore, the number of

Table 1. Clinical and Electrophysiological Characteristics of Study Group

Pt No.	Age (yr)/ Gender	Symptom Duration (yr)	Heart Disease	AT Cycle (ms)	Atrial Site	RF Pulses (no.)	Response to RF
1	41/M	3.1	No HD	305	HRA	4	Terminate at 1.9 s
2	33/M	7.4	VD	345	RAA	2	Terminate at 2.4 s
3	47/M	6.2	No HD	315	P	4	None
4	28/F	3.9	No HD	395	MRA	1	Terminate at 1.3 s
5	25/M	13.5	DCM	385	CSO	7	Terminate at 3.4 s
6	64/F	11.5	DCM	250	HRA	1	Terminate at 1.6 s
7	22/M	8.2	DCM	270	FO	2	Terminate at 1.2 s
8	17/F	9.8	DCM	335	CSO	1	Terminate at 1.5 s
9	8/F	3.5	SAD	320	RAA	9	Terminate at 4.6 s
10	24/M	12.5	DCM	260	PLA	4	None
11	27/F	0.3	No HD	350	LAA	5	Terminate at 2.2 s
12	35/M	10.2	DCM	295	HPRA	1	Terminate at 1.3 s
13	19/M	8.4	No HD	305	HRA	5	Terminate at 1.6 s
14	25/M	5.3	No HD	420	HRA	1	Terminate at 1.9 s
15	7/M	3.8	DCM	315	CSO	4	None
16	18/F	7.1	No HD	480	IPV/LA	1	Terminate at 2.4 s
17	44/F	2.4	VD	320	LAA	13	Terminate at 1.6 s
18	26/M	5.4	DCM	360	MRA	2	Terminate at 1.3 s
19	21/F	6.3	DCM	280	RAA	4	Terminate at 2.0 s
20	36/M	4.9	CCTGV	240	HPRA	9	Terminate at 2.3 s
21	29/F	5.6	No HD	370	FO	1	Terminate at 3.2 s
22	34/M	3.3	No HD	405	RAA	1	Terminate at 2.6 s
23	33/F	7.9	DCM	330	FO	10	Terminate at 1.5 s
24	61/F	0.6	VD	360	MRA	2	Terminate at 4.6 s
25	14/M	1.5	DCM	320	CSO	1	Terminate at 2.4 s
26	38/M	3.5	No HD	380	HRA	6	Terminate at 1.5 s
27	16/F	9.2	DCM	360	IPV/LA	3	Terminate at 2.3 s
28	35/M	2.7	No HD	315	P	9	Terminate at 3.6 s
29	28/F	0.4	No HD	340	MRA	1	Terminate at 1.5 s
30	24/F	2.4	No HD	320	P	4	Terminate at 4.1 s
31	31/M	5.8	No HD	350	HRA	1	Terminate at 3.2 s
32	28/F	4.7	No HD	270	SVC/RA	16	Terminate at 4.1 s
33	44/M	6.1	DCM	315	LAA	2	Terminate at 1.5 s
34	25/F	1.1	No HD	325	IPV/LA	5	Terminate at 2.1 s
35	37/F	9.4	DCM	340	FO	7	Terminate at 1.7 s
36	30/M	4.5	No HD	375	HRA	3	Terminate at 1.5 s
37	27/M	2.1	No HD	305	HRA	3	Terminate at 2.7 s
38	15/M	6.8	DCM	320	P	1	Terminate at 1.9 s
39	29/F	3.1	No HD	380	IPV/LA	2	Terminate at 4.5 s
40	33/M	2.1	No HD	270	CSO	2	Terminate at 1.8 s
41	27/M	18.0	DCM	310	HRA	7	Terminate at 3.2 s
42	33/F	5.1	No HD	330	PLA	4	Terminate at 2.7 s
43	37/M	7.5	DCM	410	HPRA	1	Terminate at 1.9 s
44	24/F	9.8	ASA	380	FO	3	Terminate at 2.1 s
45	25/M	4.0	No HD	330	CSO	3	Terminate at 3.8 s

ASA = atrial septal aneurysm; AT = atrial tachycardia; CCTGV = congenitally corrected transposition of the great vessels; CSO = coronary sinus ostium; DCM = dilated cardiomyopathy; F = female; FO = fossa ovalis; HD = structural heart disease; HPRA = high posterior right atrium; HRA = high right atrium; IPV/LA = inferior pulmonary veins-left atrium; LAA = left atrial appendage; M = male; MRA = middle right atrium; P = parasinusal (high posterior roof of the right atrium); PLA = posterior left atrium; Pt = patient; RAA = right atrial appendage; RF = radiofrequency; SAD = secundum atrial defect; SVC/RA = superior vena caval-right atrial junction; VD = valvular disease.

ineffective radiofrequency applications was reduced. The positive predictive value of each single technique was significantly improved by adding successful mechanical interruption of atrial tachycardia (35.5% vs. 49.2% for the AP interval ≥ 30 ms, 29.5% vs. 44.6% for the pace-mapping). An ablation site characterized by the simultaneous presence of an AP

interval ≥ 30 ms, concordant pace-mapping and mechanical interruption of the tachycardia at the site of the ablation catheter has a 72.7% success probability as compared with 5.3% success probability in a site without these characteristics.

Complications. In Patient 11, after right femoral artery puncture, retroperitoneal hemorrhage with acute postrenal fail-

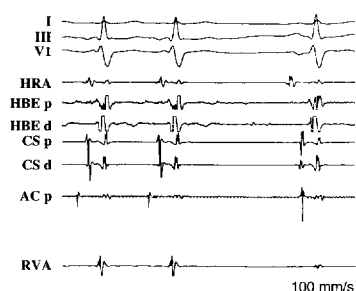


Figure 4. Patient 16. Surface electrocardiographic and intracardiac electrographic recordings during radiofrequency delivery at the ablation site shown in Figure 1.

ure was observed; this condition normalized within 36 h after the ablation procedure. This patient was on chronic anticoagulant therapy, discontinued 4 days before the procedure, and his prothrombin activity was 78% at the time of the procedure. In two patients (one with parasinusal and one with middle right atrial focus), we observed a temporary depression of the sinus node function with junctional escape rhythm. These two patients received a temporary pacemaker and remained under observation up to 6 days; recovery times for the sinus rhythm were 23 and 58 h after the ablation procedure, respectively.

Follow-up. During a mean follow-up of 22.3 ± 12.1 months, three recurrences of atrial tachycardia were observed. All patients were successfully submitted to a new ablation procedure.

Discussion

The present study provides a reliable and reproducible method for precise localization of the target site for radiofrequency current ablation of automatic atrial tachycardia. The major findings were that 1) AP interval ≥ 30 ms and concordant pace-mapping were both significant predictors of success; 2) they had a high sensitivity but a low specificity; 3) mechanical interruption of atrial tachycardia improved the specificity and positive predictive value of these two mapping techniques; 4) by using the three mapping techniques, we had a 93% success rate with only 3.9 ± 3.4 radiofrequency pulses/patient.

Observations during mapping of ectopic atrial tachycardia.

The prevalence of right-sided atrial tachycardia, although in contrast with some studies (12), confirms data from the larger series (6,7,13,14,16,24,25). The AP interval ≥ 30 ms and pace-mapping concordance were significantly more frequent in successful than in failure sites, and both were significant predictors of success.

The AP interval in sites where radiofrequency was successful was greater than where radiofrequency was unsuccessful (36.75 ± 5.79 vs. 28.36 ± 5.13 ms, $p < 0.001$). The sensitivity, specificity and positive predictive value of AP interval ≥ 30 ms were 92.8%, 47.8% and 35.5%, respectively, confirming the high sensitivity and the poor specificity of this technique in predicting the success of the procedure. This may depend on the size of the distal electrode of the ablation catheter (4 mm), which was too large to record an ideal local electrogram (14). Furthermore, measurement of AP interval might be affected by the difficulty of identifying with accuracy the beginning of the P wave.

By pace-mapping concordance, the ablation site was identified successfully in 85.7% of patients, but in 63.2% of patients it was not possible to recognize failure sites by this method, with sensitivity, specificity and positive predictive value of 85.7%, 36.8% and 29.5%, respectively. Previous studies have demonstrated that the activation sequence concordance during atrial pacing and during atrial tachycardia is a predictor of success (13). In our experience this procedure has a lower positive predictive value as compared with the earliest atrial activation technique, probably as a consequence of the larger population studied. The low specificity of pace-mapping in our study was not unexpected, because we observed that by pacing we reproduced the activation sequence whenever the exploring catheter was positioned within 5 mm of the ablation site. By pacing in this area in different sites, even those not connected with the tachycardia, the same P wave configuration and the same endocavitary activation sequence observed during tachycardia could be reproduced. Accordingly, in our experience this technique appeared highly sensitive (85.7%) but less specific (36.8%). Nevertheless, its positive predictive value could vary in different laboratories according to the energy and duration of radiofrequency delivered to the ablation site. A 25-

Table 2. Results of Atrial Mapping at Successful and Unsuccessful Sites of Radiofrequency Delivery

Variable	Successful Sites (n = 42)	Unsuccessful Sites (n = 136)	p Value
AP interval (ms)	37.5 ± 6.17	28.57 ± 5.06	<0.001
AP interval ≥ 30 ms (1)	39/42 (92.8%)	71/136 (52.2%)	<0.001
Pace-mapping concordance (2)	36/42 (85.7%)	86/136 (63.2%)	0.01
Mechanical interruption (3)	32/42 (76.2%)	39/136 (28.7%)	<0.001
1 + 2	35/42 (83.3%)	54/136 (39.7%)	<0.001
1 + 3	31/42 (73.8%)	32/136 (23.5%)	<0.001
2 + 3	29/42 (69.0%)	36/136 (26.5%)	<0.001
1 + 2 + 3	24/42 (57.1%)	9/136 (6.6%)	<0.001
None	1/42 (2.4%)	18/136 (13.2%)	0.08

Data presented are mean value (\pm SD) or number (%) of patients.

Table 3. Sensitivity, Specificity and Predictive Value of Atrial Mapping in Identifying Successful Sites of Radiofrequency Delivery

Variable	Sensitivity (95% CI)	Specificity (95% CI)	Positive Predictive Value
AP interval \geq 30 ms (1)	92.8% (80.5-98.5)	47.8% (37.9-58.2)	35.5%
Pace-mapping concordance (2)	85.7% (71.5-94.6)	36.8% (27.6-47.2)	29.5%
Mechanical interruption (3)	76.2% (60.5-87.9)	71.3% (61.0-79.6)	45.1%
1 + 2	83.3% (68.6-93.0)	60.3% (49.7-69.7)	39.3%
1 + 3	73.8% (58.0-86.1)	76.5% (66.4-84.0)	49.2%
2 + 3	69.0% (52.9-82.4)	73.5% (63.2-81.4)	44.6%
1 + 2 + 3	57.1% (41.0-72.3)	93.4% (86.1-97.1)	72.7%
None	2.4% (0.1-12.6)	86.8% (78.8-92.9)	5.3%

CI = confidence interval.

to 30-W delivery for 60 s could induce a lesion sufficiently wide to minimize the lack of precision of this mapping technique.

Role of catheter-induced mechanical interruption of automatic atrial tachycardia. The possibility of transiently interrupting electric conduction in an accessory pathway or an ectopic focus by catheter pressure has previously been reported (12,16,26,27). The mechanical interruption of automatic atrial tachycardia in these studies was purely accidental and occurred in a percentage of patients ranging from 8.3% to 16.6%. In contrast, we intentionally made attempts to induce mechanical block before radiofrequency delivery in each patient. This may very well be the reason for the increase in rate of termination of automatic tachycardia in our series. In our study mechanical interruption was observed in 76.2% of successful ablation sites but in only 28.7% of ineffective ablation sites ($p < 0.001$) with a sensitivity, specificity and positive predictive value of 76.2%, 71.3% and 45.1%, respectively. Tachycardia mechanical interruption in our series was the best predictor of success. Moreover, this technique improved the specificity and positive predictive value of the two previous mapping techniques.

This may be the result of the thin atrial layer (1 to 2 mm) and location and size of the anatomic substrate (2 to 5 mm on either the endocardial or the epicardial side). It can be hypothesized that catheter pressure alters atrial cell properties by affecting membrane permeability of a critical mass of cells responsible for tachycardia. A previous study (29) demonstrated that catheter-induced mechanical stunning of accessory pathway conduction led to prolongation of accessory pathway refractoriness and/or slowing of conduction. Although we did not analyze the effect of catheter-induced mechanical trauma on electrophysiologic properties of automatic atrial cells, the prompt resumption of atrial tachycardia after relaxation of catheter pressure on the focus strongly supports the evidence

of a relation between catheter-induced trauma and mechanical interruption.

Limitations of the technique are 1) the traumatic stunning of the substrate may persist several hours, 2) the excessive catheter pressure might cause perforation of the atrial wall, although this event never took place in our series; 3) in some critical areas it is difficult to achieve a good contact between catheter and atrial wall to reach a sufficient pressure on the substrate; for this reason we were not able to induce mechanical interruption of atrial tachycardia in 23.8% of successful sites; 4) inappropriate "whiplash" movements and lateral torsion of the mapping catheter may dislocate the catheter from the ablation sites once mechanical interruption has been obtained. In fact, we observed mechanical interruption of atrial tachycardia in 28.7% of unsuccessful sites.

One of the unsolved problems in atrial tachycardia radiofrequency catheter ablation is the possible arrhythmogenicity of the induced lesions on failed sites (30). Though only isolated cases of postablation atrial arrhythmias are reported (14,16), this event might be related to the large number and duration of energy deliveries (12,14,15) as well as to the test lesions, that is, the short deliveries of energy used in some laboratories to verify whether radiofrequency interrupts tachycardia (12-15). Test lesions might induce transmural necrosis and the formation of a critical fibrous area, since it was shown that most of the radiofrequency effect takes place within 15 to 20 s (31,32). Therefore, we decided not to use test lesions and instead used mechanical interruption to identify the site of radiofrequency delivery. In our series, during the follow-up period of 22.3 ± 12.1 months, atrial fibrillation, flutter or tachycardia was never observed in patients successfully ablated.

Limitations of the study. Unipolar pacing or unipolar recording techniques were not used to localize the site of tachycardia origin. This technique may improve the reliability of the method, avoiding a potential problem with pace-mapping relating to capture by either the anode or the cathode during bipolar pacing.

We did not use mechanical trauma as an independent mapping technique but only to validate the so-called ideal ablation site identified by previous mapping techniques as that with the earliest atrial activation and pace-mapping. As a consequence, we do not know what would result from applying mechanical trauma to other sites.

Conclusions. The radiofrequency catheter ablation of automatic atrial tachycardia proved to be, in our experience, safe and effective. An AP interval \geq 30 ms and pace-mapping concordant sequence were reliable mapping features to predict the outcome of the ablation procedure. Mechanical interruption of atrial tachycardia improved the specificity and the positive predictive value of these two mapping techniques.

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