

Clinical and Electrophysiologic Characteristics of Left Septal Atrial Tachycardia

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OBJECTIVES	It was the purpose of this study to define the electrophysiologic (EP) identity of left septal atrial tachycardia (AT).
BACKGROUND	The clinical and EP characteristics of this particular type of arrhythmia have not been fully described.
METHODS	A total of 120 patients with AT underwent invasive EP evaluation. Five patients (two men and three women; mean age 49 ± 15 years) with left septal AT were identified. Mapping of the right and left atrium was performed using conventional electrode catheters (five patients) and a three-dimensional electroanatomic mapping system (three patients) followed by radiofrequency (RF) ablation at the earliest site of local endocardial activation.
RESULTS	Five tachycardias with a mean cycle length of 320 ± 94 ms were mapped, and the earliest endocardial electrogram occurred 22 ± 10 ms before the onset of the surface P-wave. Three left septal ATs were found to be originating from the left inferoposterior atrial septum and two from the left midseptum. During tachycardia, positive (three patients), biphasic negative-positive deflection (one patient), or isoelectric (one patient) P waves were recorded in lead V_1 . The inferior leads demonstrated a positive or biphasic P-wave morphology in four of five patients (80%). Four patients were given both adenosine and verapamil during AT. In three of four patients, verapamil successfully terminated AT after adenosine had failed. Adenosine successfully terminated AT in one of four patients. Successful RF ablation was performed in all patients (mean 2.2 ± 1.7 RF applications) without affecting atrioventricular conduction properties. No recurrence of AT was observed after a mean follow-up of 14 ± 8 months.
CONCLUSIONS	Left septal AT ablation is safe and effective. There was no consistent P-wave morphology associated with this particular type of AT. This arrhythmia appears to be resistant to adenosine and moderately responsive to calcium antagonists. (J Am Coll Cardiol 2002;40:1133-9) © 2002 by the American College of Cardiology Foundation

The clinical and electrophysiologic (EP) characteristics of most supraventricular tachycardias (SVTs) have been well-studied (1,2). Only 7% of all SVTs have been ascribed to atrial tachycardia (AT), making this tachycardia the least common form of SVT (1-3). The success rate of radiofrequency (RF) ablation for the treatment of AT ranges between 60% and 95% depending on the arrhythmia mechanism, site of origin, and the mapping methods used to localize the arrhythmia (3-7). The ability of adenosine to terminate AT varies between 50% and 80% depending on the AT site of origin (4-8). Recently, the EP identity of right septal AT has been reported by Chen et al. (8). These authors described a high sensitivity of right septal AT to adenosine. Moreover, they showed a positive P-wave morphology in lead V_1 to be highly predictive of right postero-septal AT. To our knowledge no study has yet defined the clinical and EP characteristics of left septal AT. In this study we report on the different electrocardiographic (ECG) manifestations, location of arrhythmia origin, response to adenosine and calcium antagonists, and the results of RF ablation of left septal AT.

METHODS

Patients. Between March 1998 and February 2000, 120 patients with focal AT were evaluated in our laboratory. Five of the 120 patients (4%) in whom mapping of both atria revealed left septal AT were included in this study. The study population consisted of two men and three women with a mean age of 49 ± 15 years (range, 35 to 76 years). Atrial tachycardia was paroxysmal in four patients and incessant in one. The mean duration of symptoms was 4.6 years (range, 2 to 10 years). Coronary artery disease and sick sinus syndrome were present in one patient; one patient had mild mitral valve regurgitation, while no structural heart disease was present in the remaining three patients. All patients had previously documented narrow QRS complex tachycardia. All tachycardias were refractory to a mean of 2.6 ± 1.6 antiarrhythmic drugs (range, 1 to 5). Three patients failed treatment with beta-blockers while none were treated with a calcium antagonist.

EP study protocol. Antiarrhythmic drugs were withheld at least five half-lives before EP study. All patients gave witnessed written informed consent. Electrode catheters were positioned under biplane fluoroscopic guidance. Four multipolar catheters were inserted percutaneously into the right femoral vein. A 7F duodecapolar catheter (2-mm interelectrode spacing; Daig Corporation, Minnetonka,

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Abbreviations and Acronyms

A-A-V	= atrial-atrial-ventricular pattern
A-H	= atrial-His interval
AT	= atrial tachycardia
AV	= atrioventricular
CS	= coronary sinus
ECG	= electrocardiogram/electrocardiographic
EP	= electrophysiologic
RF	= radiofrequency
SVT	= supraventricular tachycardia
VA	= ventriculoatrial

Minnesota) was placed along the crista terminalis, and a 6F octapolar catheter (2-mm interelectrode spacing; EP Technologies, Sunnyvale, California) was used to record His-bundle potentials. A 6F quadripolar catheter (2-5-2-mm interelectrode spacing; Daig Corporation) was placed at the right ventricular apex. In two patients, a 7F quadripolar asymmetrical thermocoupled ablation catheter (4-mm tip electrode; EP Technologies) was used for atrial mapping and ablation. In the other three patients, a nonthermocoupled 7F quadripolar catheter (Biosense Webster, Diamond Bar, California) was used for the same purpose. Coronary sinus (CS) recordings were performed using a 6F decapolar catheter (2-5-2-mm interelectrode spacing; Daig Corporation), which was inserted through the right internal jugular vein.

The three-dimensional electroanatomic mapping system used in this study has been described elsewhere (9,10). In short, it consists of an electromagnetic location pad positioned under the patient's back, a CARTO processor (Biosense Webster), a Silicon Graphics workstation, and two electromagnetic sensor-equipped catheters. One catheter was used as a reference and was placed on the back of the patient, while the second catheter was used as a rove for identification of landmarks, mapping, and ablation.

The EP study included assessment of sinus node function, atrial and atrioventricular (AV) conduction properties. Atrial overdrive pacing and right atrial extrastimuli were used to induce AT. Atrial overdrive pacing was initiated at a drive cycle length of 600 ms for 15 beats with decrements of 20 to 250 ms unless tachycardia was initiated. Single, double, and triple atrial extrastimuli were used if the above mentioned protocol was not successful in inducing AT. If both maneuvers failed to induce AT or if the AT was nonsustained (i.e., lasting for <30 s with spontaneous termination), intravenous isoproterenol was administered (1 to 6 $\mu\text{g}/\text{min}$), and repeat pacing was performed to induce the tachycardia. Atrial tachycardia was defined as a sudden onset of rapid atrial rhythm with discrete P waves at rates from 130 to 240 beats/min with a clearly defined isoelectric baseline between P waves in all leads (11).

Two or more of the following criteria were used to confirm the diagnosis of AT (12-17): 1) tachycardia initiation independent of a critical atrial-His (A-H) interval; 2)

demonstration of AV conduction block during tachycardia; 3) if ventriculoatrial (VA) conduction was present, ability to perform overdrive pacing via the right ventricle at a cycle length 10 to 40 ms shorter than the cycle length of AT and with the electrogram sequence immediately after the last ventricular paced beat showing an "atrial-atrial-ventricular" (A-A-V) pattern; 4) demonstration of AV dissociation with ventricular pacing; 5) delivery of a single atrial-paced beat during AT producing a variable VA interval of the return cycle; and 6) delivery of a single ventricular-paced beat during AT advancing the His-bundle potential without affecting atrial activation.

RF catheter ablation. The right atrium was mapped after confirming the diagnosis of AT using the above mentioned criteria. Conventional activation mapping was performed in two patients, and combined conventional and three-dimensional electroanatomic mapping was carried out in the remaining three patients. The earliest local endocardial activation relative to the onset of the P-wave on the surface ECG was targeted for ablation. Radiofrequency energy was delivered using an EPT 1000 generator (EP Technologies) that allowed continuous observation of changes in energy, temperature, and impedance. Radiofrequency energy was applied in a power range from 10 to 50 W or using a power-titrated temperature approach ($>50^{\circ}\text{C}$). In case the earliest atrial activation site was proven to be located at the right atrial septum, and to precede the onset of surface P-wave by ≤ 15 ms, left atrial mapping was performed before right atrial RF delivery. When RF application led to AT termination within 10 to 15 s, energy delivery was resumed for a total of 60 s. If the RF ablation application failed to terminate the tachycardia, mapping was resumed to find an earlier site. Immediately and 30 to 40 min after successful RF ablation, burst pacing and programmed atrial stimulation with and without isoproterenol was conducted in order to confirm that the arrhythmia was no longer inducible. After ablation each patient was monitored on telemetry for 24 h before discharge.

P-wave analysis. Atrial tachycardia P-wave analysis was performed during the EP study. A 12-lead surface ECG was recorded at a paper speed of 50 and 100 mm/s. If the P-wave could not be visualized during tachycardia due to QRST overlap of the preceding heart beat, single premature ventricular-paced beats were introduced to allow for better visualization of the P-wave. If this maneuver failed to isolate the P-wave, adenosine was given, and P-wave visualization was achieved if the AT did not terminate. The P-wave morphology was visually evaluated and based on consensus between two observers.

Administration of verapamil and adenosine. In order to assess the sensitivity of the tachycardia to verapamil and adenosine, both drugs were administered as an intravenous bolus during sustained AT in four of five patients. Verapamil was administered at a dosage between 5 and 10 mg over 30 s. Adenosine was administered at a dosage of 6 mg,

Table 1. Patient Demographics and Left Septal AT Characteristics

Patient	Age	Gender	LA (mm)	SHD	Verapamil-Sensitive	Adenosine-Sensitive	Criteria to Prove AT*	Localization of AT	AT Cycle Length (ms)	Inducibility of AT	EEA to P-Wave (ms)
1	35	F	34	—	yes	no	1, 2	IPS	465	Spontaneous	10
2	46	M	40	MVR	yes	no	1, 3, 5	IPS	270	AOD	30
3	44	M	36	SSS, CAD	n/a	n/a	3, 4	IPS	240	Isoproterenol	20
4	76	F	38	—	yes	no	1, 2, 3	MS	260	3 ES	15
5	45	F	32	—	n/a	yes	2, 6	MS	366	AOD	28

*See text for explanation of criteria.

AOD = atrial overdrive pacing; AT = atrial tachycardia; CAD = coronary artery disease; EEA = earliest endocardial activation; ES = extrastimulus; IPS = inferoposterior septum; LA = left atrium; MS = midseptum; MVR = mitral valve repair; n/a = not applied; SHD = structural heart disease; SSS = sick sinus syndrome.

12 mg, and 18 mg over 2 to 3 s. The minimal dose required for termination was documented.

Data analysis. Multiple comparisons were made with analysis of variance. Data were expressed as mean ± SD. A value of $p < 0.05$ was considered statistically significant; p value was not corrected for multiple comparisons.

RESULTS

EP study. One of the five study patients presented with spontaneous sustained AT. Atrial overdrive pacing at a cycle length ranging between 200 and 300 ms initiated AT in two patients. Triple atrial extrastimuli were needed to induce AT in one patient, and intravenous isoproterenol at 2 $\mu\text{g}/\text{min}$ was given to induce AT in the remaining patient. The mean AT cycle length was 320 ± 94 ms (range, 240 to 465 ms) (Table 1).

Different P-wave configuration during tachycardia compared with sinus rhythm was observed in all patients. In Patient 1, tachycardia initiation was independent of a critical A-H interval (criteria #1), and AV Wenckebach block during tachycardia (criteria #2) supported the diagnosis of AT. Patient 2 also showed criteria #1 and had an A-A-V pattern (criteria #3) after ventricular pacing during tachycardia. In addition, delivery of a single atrial-paced beat during AT produced a variable VA interval of the return cycle during tachycardia (criteria #5). Patient 3 also showed criteria #3 and had AV dissociation at multiple ventricular-paced cycle lengths (criteria #4). Patient 4 had criteria #1, #2, and #3. In Patient 5 we demonstrated, in addition to criteria #2, that delivery of a single ventricular-paced beat during AT advanced the His-bundle potential without affecting atrial activation (criteria #6).

P-wave configuration. The P-wave duration during tachycardia (82 ± 25 ms; range, 56 to 109 ms) was shorter than the P-wave duration during sinus rhythm (115 ± 25 ms; range, 90 to 134 ms) ($p < 0.05$). The different observed P-wave morphologies are shown in Table 2. In lead V_1 a positive P-wave (Fig. 1A) was found in three patients, while isoelectric (Fig. 1B) or biphasic (Fig. 1C) P waves (negative-positive) were present in two patients. The rest of the precordial leads showed a positive P-wave in three patients and an isoelectric P-wave in the remaining two patients. In lead I the P-wave was positive in three patients, isoelectric in one patient, and biphasic (positive-negative) in one

patient. Lead II showed a positive P-wave morphology in three patients, an isoelectric P-wave in one patient, and a biphasic P-wave (positive-negative) in one patient. In lead III the P-wave morphology was positive in one patient, negative in two patients, and biphasic (positive-negative) in two patients. Lead aVL showed a positive P-wave in one patient, an isoelectric P-wave in one patient, and a biphasic P-wave (negative-positive) in the remaining three patients. While a short P-wave duration suggested a left septal site, there were no specific morphology characteristics associated with a left septal AT. Figures 1 to 3 represent surface 12-lead ECG from three study patients.

Response to verapamil and adenosine. Verapamil was successful in terminating AT in all three patients in whom the drug was administered. In the same group of patients, adenosine with a maximum applied bolus of 18 mg failed to terminate the tachycardia. In one patient verapamil was not given, but 6 mg of adenosine was successful in converting the patient back to sinus rhythm. Neither verapamil nor adenosine was applied in the remaining patient (Table 1).

AT localization and catheter ablation. Mapping of the right atrium showed that the earliest endocardial activation originated from the septum in all patients. In two patients RF application at the earliest site of right septal activation (20 ms and 25 ms before the onset of the surface P-wave) failed to terminate the tachycardia. Hence, the left atrium was mapped in these two patients, and the earliest site of activation was found to be midseptal in one and inferoposteroseptal in another patient (10 and 15 ms before the onset of surface P-wave, respectively). In the remaining three patients, the left atrium was mapped before right atrial RF delivery because the earliest site of endocardial activation located at the right atrial septum was <15 ms before the onset of surface P-wave. Mapping of the left atrium revealed

Table 2. Surface P-Wave Morphology During Tachycardia

Patient	Location of AT	V_1	I	II	III	aVL
1	IPS	+	+	+	+/-	+/-
2	IPS	+	0	0	-	0
3	IPS	0	+/-	+	+	+
4	MS	+/-	+	+	-	+/-
5	MS	+	+	+/-	+/-	+/-

+ = positive; - = negative; I = isoelectric; +/- = biphasic; AT = atrial tachycardia; IPS = inferoposterior septum; MS = midseptum.

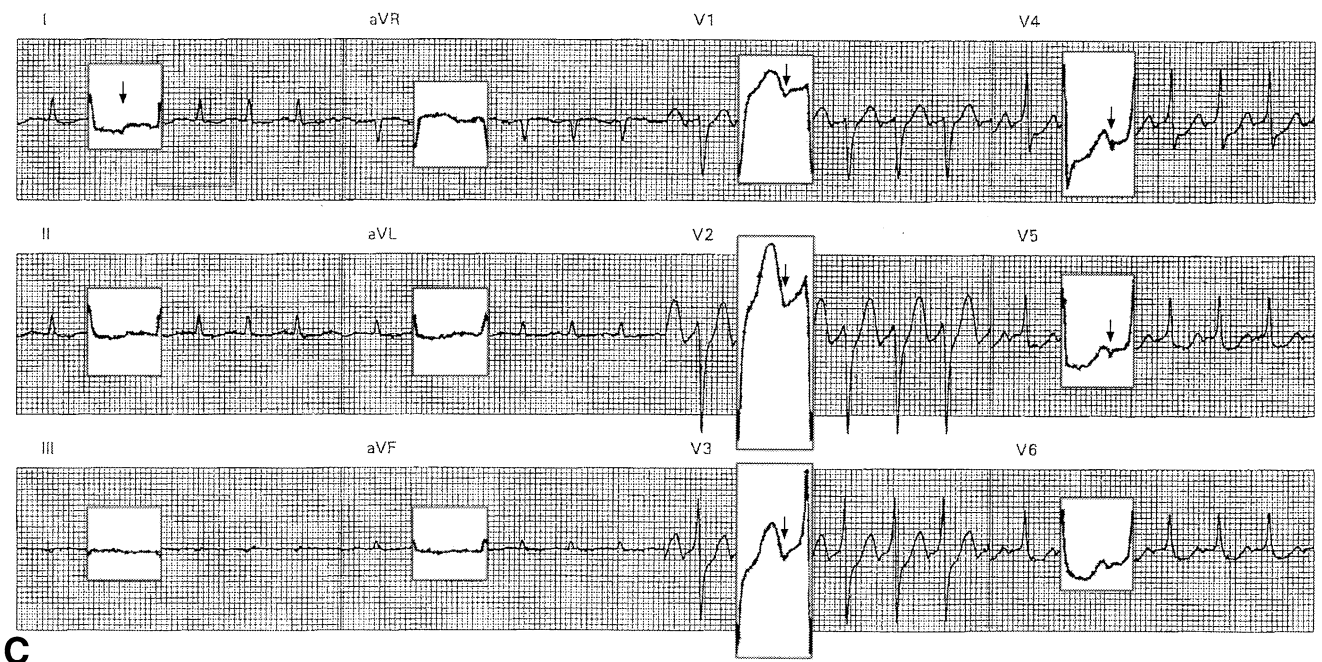
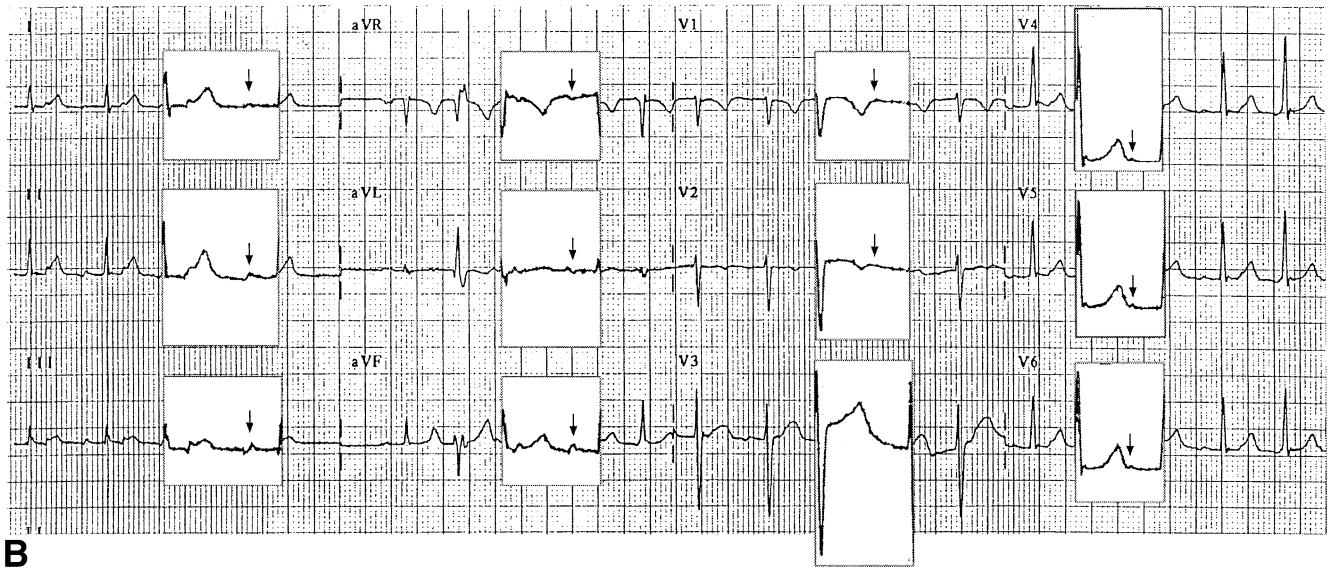
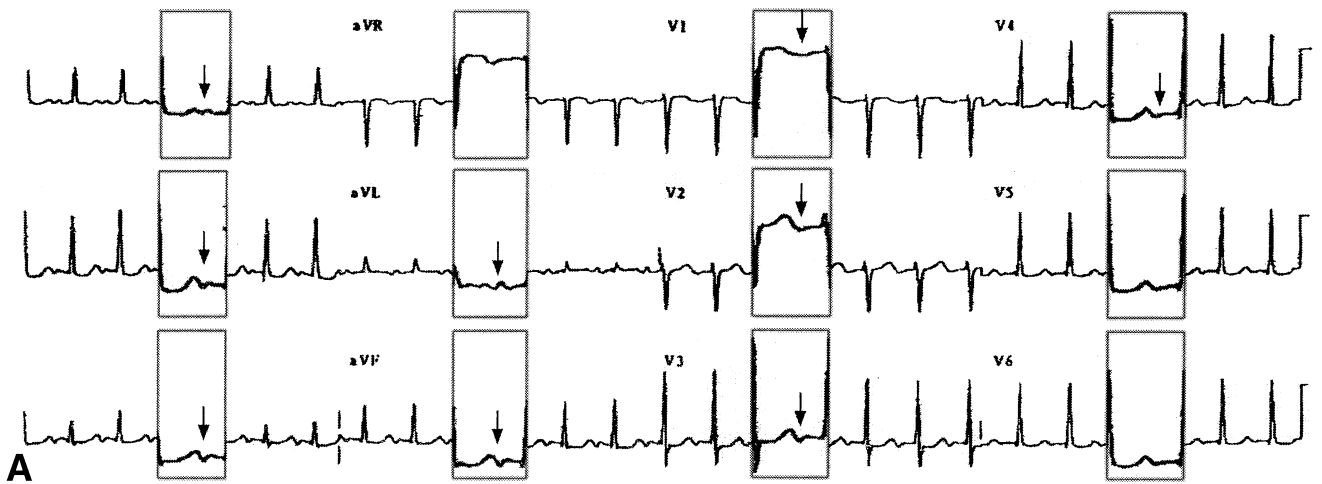


Figure 1. (A) Shows a 12-lead electrocardiogram (ECG) for Patient #4 with tachycardia originating from the left midseptal area. The P-wave in the insert is enlarged to show slightly positive P-wave in lead V₁, slightly positive in I, biphasic in II, III, aVL, and aVF. (B) Shows a 12-lead ECG for Patient #3 with tachycardia originating from the left inferoposteroseptal area. The P-wave in the insert is enlarged to show isoelectric P waves in lead V₁, positive waves in I, II, III, and aVL. (C) Shows a 12-lead ECG for Patient #4 with tachycardia originating from the left midseptal area. The P-wave in the insert is enlarged to show biphasic P waves across the precordium, slightly positive waves in I, II, aVF, and isoelectric P waves in the remainder.

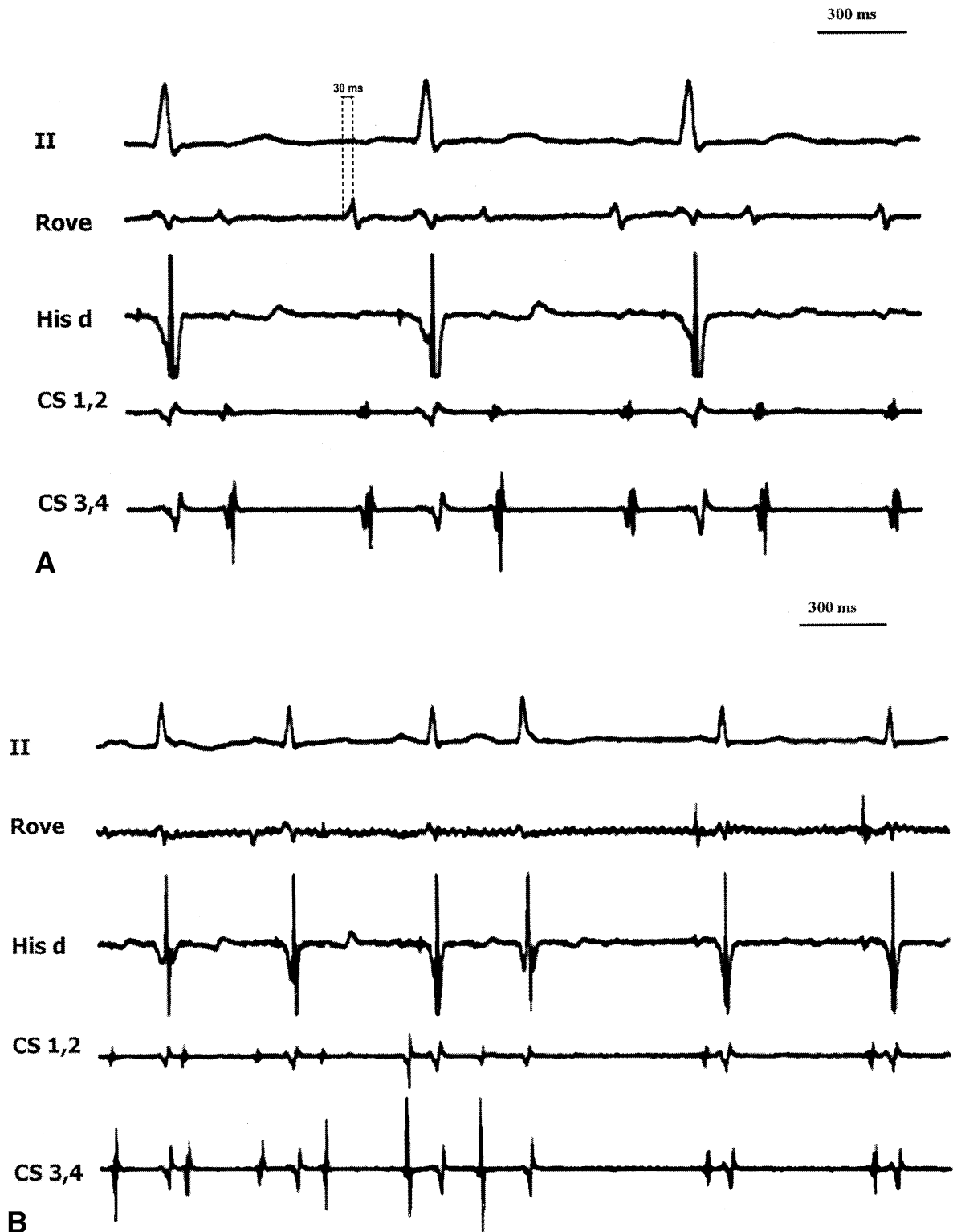


Figure 2. (A) Shown are simultaneous recordings from surface lead II, Rove catheter records from the left septum, His bundle recording, and distal coronary sinus (CS)_{1,2} and proximal CS_{3,4} recordings. The earliest activation site in the left septum was 30 ms before the onset of the surface P-wave. (B) Application of radiofrequency energy to the earliest left septal site during tachycardia results in acceleration before return of sinus rhythm.

the earliest site of activation to be midseptal in one patient and inferoseptal in two patients. Radiofrequency energy was delivered at the earliest site of left atrial activation in all patients during left septal AT (Figs. 2 and 3). The time

interval between local left septal activation and the onset of the surface P-wave was 20 ± 8 ms (range, 10 to 30 ms). A mean of 2.2 ± 1.7 RF energy applications (range, 1 to 5) was required to terminate left septal AT. In three patients

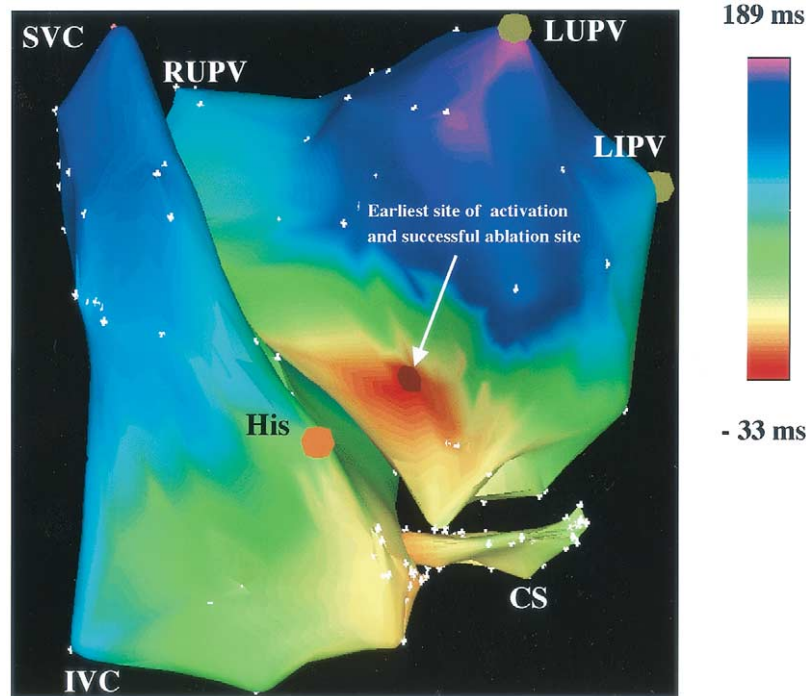


Figure 3. Left anterior oblique of three-dimensional anatomic map for Patient #1. The His bundle recording site (His) superior vena cava (SVC), inferior vena cava (IVC), coronary sinus (CS), and pulmonary veins are noted. The atrial map shows earliest left septal activation with passive spread to both left and right atrium. LIPV = left inferior pulmonary vein; LUPV = left upper pulmonary vein; RUPV = right upper pulmonary vein.

the tachycardia terminated after 2 to 3 s of a single RF application. Electrophysiologic study after successful ablation showed no significant changes in AV nodal properties. No AT recurrence was observed after a mean follow-up time of 14 ± 8 months (range, 8 to 29 months).

DISCUSSION

Main finding. This is the first report to define the ECG and EP characteristics of left septal AT. We found that the P-wave duration during tachycardia was significantly shorter than during sinus rhythm, and this phenomenon was believed to be related to a rapid biatrial spread from a focal septal origin of activation. In contrast, the morphologic features of the P waves were quite variable, and no characteristic pattern was discernible. The tachycardia was associated with a relatively short interval between the earliest endocardial activation and the onset of the surface P-wave and was found to be verapamil-responsive and adenosine-resistant for most of the patients tested.

Comparison with prior studies. Previous studies have reported on catheter mapping and ablation of AT (4,6,7). With atrial free wall sites of AT origin, the time from earliest endocardial activation to onset of the P-wave has been shown to vary between 20 to 60 ms (4,6-8). Chen et al. (8) reported that the interval between the earliest endocardial activation and the onset of the P-wave was 36 ± 6 ms in a series of 27 patients with ATs originating from the right and left free walls. We cannot explain the discrepancy between studies. Of note was the relatively short P-wave

duration during tachycardia. We propose that activation interval for our patients with left septal AT can be explained by the fact that activation originating from a septal focus has ready access to both the right and left atrium over the previously reported intra-atrial connections such as Bachman's bundle, the region of the fossa ovalis, and the CS (18-20). Sun et al. (19) demonstrated discordance in activation time between the right and left atrial septum in dogs during pacing at the right and left atrial septum. They showed that left septal activation was completed in a significantly shorter period of time compared with right septal activation. These data are in line with the present and previously published results, where left septal ATs demonstrated a shorter P-wave duration compared with the P-wave in sinus rhythm.

We could not find a characteristic P-wave morphology for patients with left septal AT. This finding is in contrast with prior studies, showing good sensitivity and specificity in the use of the 12-lead ECG in the separation of right and left atrial foci (21,22). These reports, however, did not include patients with left septal foci. In contrast with our findings, Chen et al. (8) described characteristic P-wave morphology findings for patients with right septal foci. They reported a positive P-wave in lead V_1 and a negative P-wave in the inferior leads in right posteroseptal AT, a biphasic P-wave in lead V_1 and a negative P-wave in the inferior leads in right midseptal AT, and a biphasic P-wave in lead V_1 and a positive P-wave in the inferior leads in right anteroseptal AT. It is interesting to note that the P-wave

morphology of septal AT originating from the right or left atrium would demonstrate such significant differences.

The response to pharmacologic agents may allow insight into the arrhythmogenic mechanism of left septal AT. A positive response to verapamil was observed in all patients in whom the drug was administered. Patients who responded to a relatively low dose of verapamil but not to the maximal dosage of adenosine may have microreentry involving a calcium-dependent portion of the circuit. In contrast, the patient who responded to adenosine may have a triggered mechanism as reported by Chen et al. (23). The small number of patients preclude definitive statements about arrhythmia mechanism. The proximity of the successful site of ablation to the AV node together with the positive response to verapamil raises the possibility that tachycardias may have involved the AV node. The aggregate of evidence in terms of both the EP findings and lack of response to maximal doses of adenosine would strongly mitigate this possibility.

Conclusions. This study demonstrated that mapping and ablation of left septal AT using conventional and three-dimensional electroanatomic catheter mapping is safe and effective. No specific 12-lead ECG P-wave morphology appears to be associated with left septal AT. Moreover, verapamil possesses a relatively high success rate in terminating left septal AT, while this tachycardia seems to be resistant to adenosine. Due to the relatively high sensitivity of left septal AT to verapamil, microreentry could be hypothesized as the underlying mechanism of this tachycardia.

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REFERENCES

- Wellens HJ. Atrial tachycardia. How important is the mechanism? *Circulation* 1994;90:1576-7.
- Wu D, Amat-y-leon F, Denes P, Dhinra RC, Pietras RJ, Rosen KM. Demonstration of sustained sinus and atrial re-entry as a mechanism of paroxysmal supraventricular tachycardia. *Circulation* 1975;51:234-43.
- Lai LP, Lin JL, Chen TF, Ko WC, Lien WP. Clinical, electrophysiological characteristics, and radiofrequency catheter ablation of atrial tachycardia near the apex of Koch's triangle. *Pacing Clin Electrophysiol* 1998;21:367-74.
- Kay GN, Chong F, Epstein AE, Dailey SM, Plumb VJ. Radiofrequency ablation for treatment of primary atrial tachycardias. *J Am Coll Cardiol* 1993;21:901-9.
- Tracy CM, Swartz JF, Fletcher RD, et al. Radiofrequency catheter ablation of ectopic atrial tachycardia using paced activation sequence mapping. *J Am Coll Cardiol* 1993;21:910-7.
- Walsh EP, Saul JP, Hulse JE, et al. Transcatheter ablation of ectopic atrial tachycardia in young patients using radiofrequency current. *Circulation* 1992;86:1138-46.
- Lesh MD, Van Hare GF. Status of ablation in patients with atrial tachycardia and flutter. *Pacing Clin Electrophysiol* 1994;17:1026-33.
- Chen CC, Tai CT, Chiang CE, et al. Atrial tachycardias originating from the atrial septum: electrophysiologic characteristics and radiofrequency ablation. *J Cardiovasc Electrophysiol* 2000;11:744-9.
- Marrouche NF, Beheiry S, Tomassoni G, et al. Three-dimensional nonfluoroscopic mapping and ablation of inappropriate sinus tachycardia: procedural strategies and long-term outcome. *J Am Coll Cardiol* 2002;39:1046-54.
- Shah DC, Jais P, Haissaguerre M, et al. Three-dimensional mapping of the common atrial flutter circuit in the right atrium. *Circulation* 1997;96:3904-12.
- Saoudi N, Cosio F, Waldo A, et al. Classification of atrial flutter and regular atrial tachycardia according to electrophysiologic mechanism and anatomic bases: a statement from a joint expert group from the Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *J Cardiovasc Electrophysiol* 2001;12:852-66.
- Josephson ME, Wellens HJ. Differential diagnosis of supraventricular tachycardia. *Cardiol Clin* 1990;8:411-42.
- Knight BP, Ebinger M, Oral H, et al. Diagnostic value of tachycardia features and pacing maneuvers during paroxysmal supraventricular tachycardia. *J Am Coll Cardiol* 2000;36:574-82.
- Knight BP, Zivin A, Souza J, et al. A technique for the rapid diagnosis of atrial tachycardia in the electrophysiology laboratory. *J Am Coll Cardiol* 1999;33:775-81.
- Kadish AH, Morady F. The response of paroxysmal supraventricular tachycardia to overdrive atrial and ventricular pacing: can it help determine the tachycardia mechanism? *J Cardiovasc Electrophysiol* 1993;4:239-52.
- Leitch J, Klein GJ, Yee R, Murdock C. Invasive electrophysiologic evaluation of patients with supraventricular tachycardia. *Cardiol Clin* 1990;8:465-77.
- Ross DL, Uther JB. Diagnosis of concealed accessory pathways in supraventricular tachycardia. *Pacing Clin Electrophysiol* 1984;7:1069-85.
- Antz M, Otomo K, Arruda M, et al. Electrical conduction between the right atrium and the left atrium via the musculature of the coronary sinus. *Circulation* 1998;98:1790-5.
- Sun H, Velipasaoglu EO, Wu DE, et al. Simultaneous multisite mapping of the right and the left atrial septum in the canine intact beating heart. *Circulation* 1999;100:312-9.
- Roithinger FX, Cheng J, SippensGroenewegen A, et al. Use of electroanatomic mapping to delineate transseptal atrial conduction in humans. *Circulation* 1999;100:1791-7.
- Kalman JM, Olgin JE, Karch MR, Hamdan M, Lee RJ, Lesh MD. "Cristal tachycardias": origin of right atrial tachycardias from the crista terminalis identified by intracardiac echocardiography. *J Am Coll Cardiol* 1998;31:451-9.
- Massumi R, Tawakkol AA. Direct study of left atrial P waves. *Am J Cardiol* 1967;20:331-40.
- Chen SA, Chiang CE, Yang CJ, et al. Sustained atrial tachycardia in adult patients: electrophysiological characteristics, pharmacological response, possible mechanisms, and effects of radiofrequency ablation. *Circulation* 1994;90:1262-78.