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ELECTROPHYSIOLOGIC STUDIES

Sustained Intraatrial Reentrant Tachycardia: Clinical, Electrocardiographic and Electrophysiologic Characteristics and Long-Term Follow-Up

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Although intraatrial reentry has been traditionally listed as a mechanism for supraventricular tachycardia, few reports describing the clinical features of this arrhythmia exist. Nineteen patients with a clinical history of sustained supraventricular tachycardia were diagnosed as having intraatrial reentrant tachycardia. Seventeen (89%) patients of the 19 had underlying structural heart disease and 17 had echocardiographic evidence of atrial enlargement; the mean left ventricular ejection fraction was 51 ± 16%. A history of concomitant atrial fibrillation or flutter was present in 13 patients (68%). The mean atrial cycle length during tachycardia was 326 ± 57 ms (range 260 to 460). Fourteen patients had 1:1 atrioventricular (AV) conduction during tachycardia, of whom 50% had an RP′/RR′ ratio >0.5.

Intravenous adenosine (dose range 37.5 to 150 μg/kg) and verapamil (dose range 5 to 10 mg) had no effect on atrial tachycardia cycle length in 13 of 14 and 9 of 9 patients, respectively, despite induction of second degree AV block. Type 1a antiarrhythmic drugs achieved long-term suppression of intraatrial reentrant tachycardia in only 6 patients, whereas amiodarone (326 ± 145 mg/day) was successful in 11 patients during a 32 ± 20 month follow-up period. The remaining two patients and one patient who later developed amiodarone toxicity either progressed to (n = 1) or had (n = 2) catheter-induced high grade AV block and were treated with long-term ventricular pacing.

It is concluded that intraatrial reentrant tachycardia is often associated with structural heart disease, particularly of types that cause atrial abnormalities, but left ventricular dysfunction is not a requisite finding. Other arrhythmias are frequently observed in these patients. This arrhythmia responds poorly to type 1a antiarrhythmic drugs, but is effectively treated with amiodarone. Catheter ablation of the AV junction offers a therapeutic option for patients who are refractory to medical therapy.

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Within the last decade, there have been significant advances in understanding the mechanisms and pharmacologic treatment of the common forms of supraventricular tachycardia. However, experience with less common arrhythmias, particularly tachycardias due to intraatrial reentry, has been limited. Intraatrial reentry has been considered as the probable mechanism for tachycardia that has a regular atrial rhythm, is initiated and terminated by programmed stimulation and has an atrial activation sequence that differs from that of normal sinus rhythm (1). Unlike the more common forms of supraventricular tachycardia, this arrhythmia does not require atrioventricular (AV) node participation in the reentrant circuit and can persist despite induction of high grade AV node block (2). The primary electrocardiographic (ECG) feature that has been used to differentiate intraatrial reentrant tachycardia from atrial flutter is its slower rate (3).

There have been only sporadic clinical reports (1,4–6) describing patients with intraatrial reentrant tachycardia, and the electrophysiologic characteristics and results of long-term therapy of this arrhythmia are poorly characterized. In the present study, we evaluated 19 patients with sustained intraatrial reentrant tachycardia. Our purpose is to report salient clinical, ECG and electrophysiologic characteristics of these patients and review their response to antiarrhythmic therapy.

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Table 1. Clinical Features of 19 Patients With Intraatrial Reentrant Tachycardia

Pt. No.	Age (yr)/ Gender	Symptom Duration (mo)	Prior AAD Trials	Cardiac Disease	Atrial Enlargement	LVEF	Concomitant Arrhythmias
1	31/M	3	D	Congenital	Yes	60	AFib, AFlut, VT
2	41/M	9	D, A, Pr, P	CAD	—	75	AFlut
3	53/F	1	D, P, Q	Valvular	Yes	47	AFib, AFlut
4	74/M	3	P	CAD	—	—	VT
5	47/M	7	V, Q, Pr	CM	No	55	AFib, AFlut
6	76/M	3	D, V, Q, Mx	CM	Yes	23	AFib, AFlut, AVNR, VT
7	26/M	27	V, P, T, Q	Valvular	No	52	VT
8	62/F	12	D, Q	Valvular	Yes	35	AFib, AFlut
9	16/M	120	D, P, Q	Congenital	Yes	71	SB
10	46/M	<1	D, P	None	Yes	60	AFib, VT
11	19/M	27	D, Dp	Congenital	Yes	73	AFib, VT
12	62/F	4	D, Dz, Pi, Pr, Q	None	No	60	AFlut
13	71/F	<1	D, Pr	CAD	Yes	40	AFlut
14	33/M	1	D, Q	CM	Yes	12	VT
15	68/F	1	D, V, Pr	CAD	No	33	AFib
16	38/M	1	—	Valvular	Yes	48	None
17	79/F	1	—	CM	Yes	52	AFib, AFlut
18	72/M	1	Pr	CAD	Yes	30	None
19	76/F	12	D, V, P, Pr	Valvular	Yes	60	AFlut

A = atenolol; AAD = antiarrhythmic drugs; AFib = atrial fibrillation; AFlut = atrial flutter; AVNR = atrioventricular node reentry; CAD = coronary artery disease; CM = cardiomyopathy; D = digoxin; Dp = diphenylhydantoin; Dz = diltiazem; F = female; LVEF = left ventricular ejection fraction; M = male; Mx = mexiletine; P = propranolol; Pi = pindolol; Pr = procainamide; Pt. = patient; Q = quinidine; SB = sinus bradycardia; T = timolol, V = verapamil; VT = ventricular tachycardia.

Methods

Study patients. Nineteen patients who had a history of recurrent episodes of symptomatic tachycardia that met criteria for intraatrial reentry were evaluated at the University of Virginia Hospital between September 1982 and September 1987. In this study, the criteria used for definition of the tachycardia were similar to those proposed by Josephson and Seides (3) and included: 1) an atrial cycle length >250 ms and <600 ms in the absence of antiarrhythmic medication; 2) a P wave morphology or an atrial activation sequence, or both, distinctly different from that documented during sinus rhythm; 3) persistence of the atrial arrhythmia despite spontaneous or pharmacologically induced high grade AV node block; and 4) initiation or termination, or both, with appropriately timed atrial extrastimuli or rapid atrial pacing. All patients had presented with one or more clinical episodes of tachycardia, with a duration of ≥1 h that required intervention for termination. Ongoing tachycardia was defined as tachycardia that was continuous from the initial symptomatic presentation until termination at the time of invasive evaluation. All studies were performed for clinical indications, and all patients gave informed consent before invasive testing.

Cardiac evaluation included cardiac catheterization with coronary angiography in 8 patients and two-dimensional echocardiography in 17 patients. Atrial enlargement was defined according to standard echocardiographic criteria as a left atrial dimension >4 cm in the parasternal plane. Left ventricular ejection fraction was calculated from biplane cineangiograms according to standard methods (8 patients) or by a visual estimate from two-dimensional echocardiograms by two observers when an angiogram was not available (10 patients).

Electrophysiologic study. Studies were performed with the patient in a fasting nonsedated state after written informed consent had been obtained. Antiarrhythmic drugs were discontinued ≥48 h before the study. Patients underwent evaluation with a single (6 patients) or multiple (13 patients) 6F quadripolar electrode catheters according to standard electrophysiologic procedures, as previously described (7). During an episode of sustained tachycardia, bipolar atrial electrograms were recorded at a paper speed of 200 mm/s. The atrial catheter was positioned at four to six sites around the tricuspid valve ring, at high, mid and low sites on the anterior, lateral and septal walls, in the left atrium if a probe-patent foramen ovale was present and in the coronary sinus in selected cases. Relative atrial activation times were measured from the earliest onset of the P wave on the surface ECG to the first rapid deflection of the atrial electrogram across the baseline. The earliest site of atrial activation was defined as the earliest presystolic atrial activity that occurred in the latter half of the atrial cycle. Tachycardia cycle length was determined by measuring the AA′ interval from intracardiac electrograms acquired during ongoing clinical tachycardia over 30 s. In cases where additional episodes of atrial tachycardia with different rates occurred in the clinical setting, their cycle length was

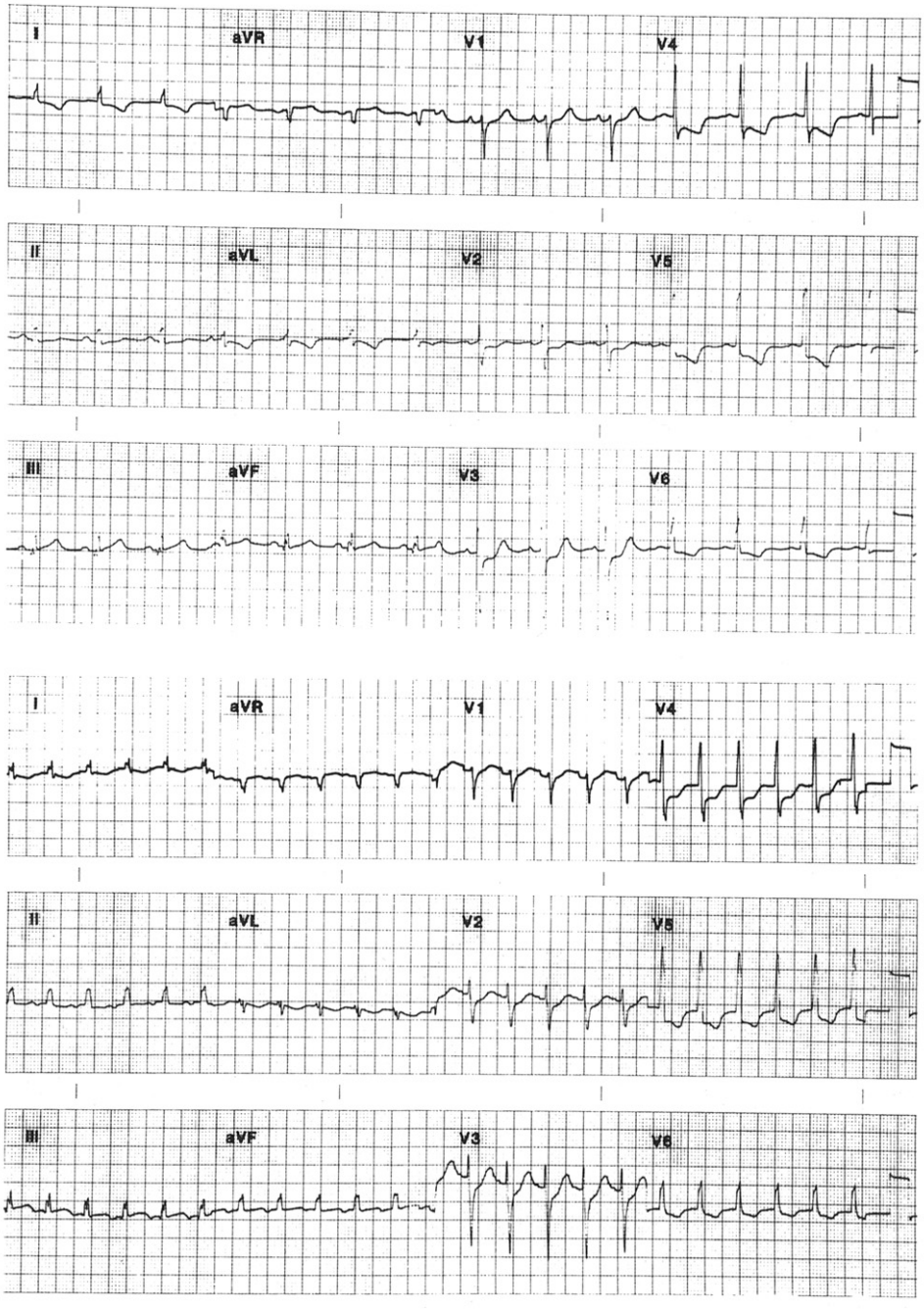

Figure 1. Patient 19. Twelve lead surface electrocardiograms during normal sinus rhythm (**upper three panels**) and intraatrial reentrant tachycardia (**lower three panels**).

approximated by measurement of the PP intervals on a surface ECG at a paper speed of 25 mm/s.

Pharmacologic testing. Adenosine (37.5 to 150 μg/kg) was administered as a rapid bolus injection to 13 patients according to a protocol previously reported (2). If AV block was not achieved at the initial dose, the dose was increased incrementally until transient AV block was observed. Additional intravenous acute pharmacologic testing was performed with verapamil (2.5 to 10 mg; nine patients), propranolol (0.1 mg/kg, one patient) and procainamide (500 to 1,500 mg, five patients).

Long-term therapy and follow-up. All patients underwent therapeutic trials with a type 1a antiarrhythmic agent (quinidine, procainamide or disopyramide) alone or in combination with digoxin or a beta-adrenergic blocker, or both. Therapy was discontinued if the patient experienced intolerable side effects or if episodes of tachycardia recurred clinically despite therapeutic antiarrhythmic drug levels. If therapy with these agents was unsuccessful, patients were then treated with amiodarone, 200 to 400 mg/day after a 4 to 7 day loading period of 800 mg/day. Two patients underwent catheter ablation of the His bundle after failure of amio-

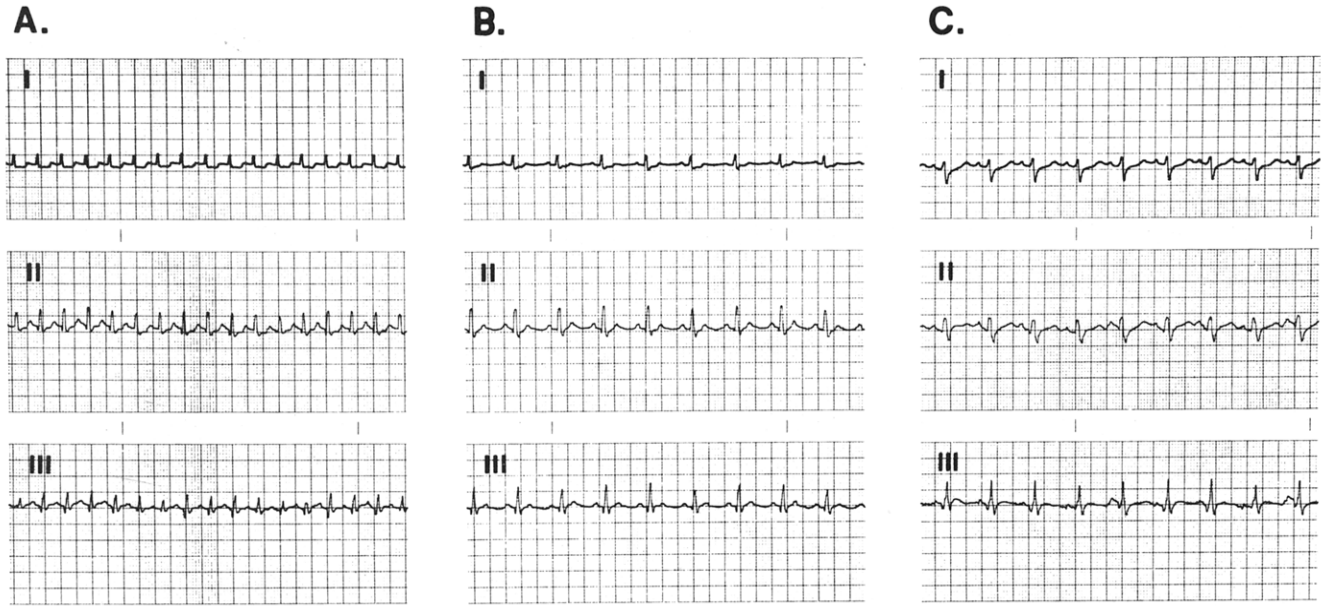

Figure 2. Patient 18. Surface electrocardiographic leads I, II and III during sustained intraatrial reentrant tachycardia (cycle length 290 ms) with 1:1 atrioventricular (AV) conduction (**panel A**) and after an intravenous injection of 10 mg of verapamil (**panel B**). This tracing has a superficial appearance of normal sinus rhythm, but careful inspection reveals persisting atrial tachycardia with 2:1 AV block. After intravenous administration of 1.0 g of procainamide (**panel C**), the patient's rhythm converted spontaneously to sinus rhythm and demonstrated a change in P wave configuration and axis (best seen in lead III), consistent with a change in intraatrial activation sequence.

darone therapy to control their arrhythmia. All patients were followed up after their initial evaluation at regular intervals to assess continuing safety and efficacy of therapy. Patient follow-up data were obtained through personal office visits or telephone contact with the referring physician, or both.

Results

Clinical and ECG data (Table 1). The mean age of the 19 patients was 52 ± 21 years. There was a wide range of duration of antecedent symptoms (2 weeks to 10 years). All patients were symptomatic during tachycardia and had received a mean of 2.5 ± 1.5 prior antiarrhythmic drug trials without success. Structural heart disease was a common finding, being observed in 17 (89%) of the 19 patients. Of the five patients with valvular heart disease, four had mitral and one had aortic disease. The three patients with congenital heart disease had tetralogy of Fallot (two patients) and an atrial and ventricular septal defect (one patient). Left atrial enlargement was common. Left ventricular ejection fraction at rest was depressed (<60%) in 11 of 18 patients (mean 51 ± 16%), but only 5 patients demonstrated severe impairment of systolic function with an ejection fraction <40%.

Fourteen patients had a history of other electrocardiographically discrete atrial arrhythmias in addition to intraatrial reentry. Four patients demonstrated episodes of spontaneous sustained monomorphic ventricular tachycardia (lasting >30 s or requiring cardioversion), and three patients had nonsustained ventricular tachycardia. Other arrhythmias observed included AV node reentrant tachycardia and intermittent sinus arrest with junctional bradycardia. Only two patients had intraatrial reentrant tachycardia as their only documented rhythm abnormality.

A normal P wave axis (0° to +90°) was observed in 16 patients during sinus rhythm and in 10 patients during intraatrial reentrant tachycardia. In all patients (except one in whom a sinus mechanism was never identified), the P wave axis or configuration was clearly different during the two separate rhythms (Fig. 1). The mean P wave axis shift (positive or negative) was 87 ± 61° (range 15 to 165). Although some patients always manifested second degree AV block during tachycardia, 14 of the 19 patients had 1:1 AV conduction during atrial tachycardia at some point during their clinical course (Fig. 2). The RP′/RR′ ratio during episodes of tachycardia with 1:1 AV conduction was variable, ranging from 0.2 to 0.7, and was >0.5 in 7 of the 13 patients with discernible P waves during tachycardia.

Electrophysiologic data (Table 2). The mean tachycardia cycle length observed in the absence of drug therapy at electrophysiologic study was 326 ± 57 ms (range 260 to 460). All tachycardias showed minor beat to beat variability in cycle length (5 to 15 ms). Two patients demonstrated a fixed pattern of cycle length oscillation during episodes of tachycardia. Eight patients had only one morphologic intraatrial tachycardia documented, but 11 patients demonstrated ei-

Table 2. Electrocardiographic and Electrophysiologic Characteristics of 19 Patients

Pt. No.	IART CL (ms)	CL Oscillation	P Wave Axis Shift NSR to IART	Maximal Observed AV Conduction	RP'/RR' During 1:1 Conduction	Earliest Site of Atrial Activation
1	260	No	+155	1:1	0.54	NA
2	270, 370	No	+30	1:1	0.43	Mid lat RA
3	315, 400	No	+180	1:1	0.40	Low lat RA
4	275, 310	No	+90	1:1	*	High lat RA
5	305	No	−150	1:1	0.51	Mid LA
6	340, 350, 395	No	−60	1:1	0.41	NA
7	320, 360, 460	Yes	−15	1:1	0.22	Mid lat RA
8	300	No	−30	2:1	—	High lat RA
9	260	No	−120	2:1	—	NA
10	300	Yes	+30	1:1	0.33	NA
11	280, 315	No	−60	1:1	0.57	NA
12	380, 460	No	−120	1:1	0.70	Low post RA
13	280, 300	No	+15	2:1	—	Mid lat RA
14	280	No	−150	2:1	—	Low ant RA
15	280, 345, 460	No	−165	1:1	0.65	Low sept RA
16	380, 440	No	+15	1:1	0.58	Low lat RA
17	350	No	NA†	2:1	—	Low sept RA
18	290, 325	No	−30	1:1	0.50	Low lat RA
19	430, 380, 305	No	+150	1:1	0.40	Mid lat RA

*Onset of P wave not discernible in the available rhythm strip during intraatrial recurrent tachycardia (IART) and 1:1 atrioventricular (AV) conduction; †Axis of P wave during intraatrial reentrant tachycardia indeterminate. ant = anterior; CL = cycle length; LA = left atrium; lat = lateral; NA = not available; NSR = normal sinus rhythm; post = posterior; RA = right atrium; sept = septal; other abbreviations as in Table 1.

ther two or three tachycardias with different cycle length, P wave configuration or atrial activation sequences (Fig. 3).

Eleven patients manifested ongoing atrial tachycardia at the time of invasive evaluation. In seven of these patients, initiation of the tachycardia by programmed stimulation was not demonstrated after its successful termination because attempts at reinitiation either yielded atrial fibrillation or atrial flutter or the clinician's judgment was against attempting reinitiation of the tachycardia. Tachycardia in the remaining patients could be reproducibly initiated with single, double or triple atrial premature stimuli (four, three and two patients, respectively) or with rapid atrial pacing (three patients) (Fig. 4 and 5). In the four patients whose tachycardia was initiated by single atrial extrastimuli, a critical intraatrial conduction delay was not measurable coincident with tachycardia onset. Tachycardia in all patients could be terminated by rapid atrial pacing, although addition of a type 1a drug to facilitate paced termination was required in five patients. Single or double atrial extrastimuli were not effective in terminating the arrhythmia in any patient.

Mapping of multiple right atrial and coronary sinus sites was performed in 14 patients to determine the relative atrial activation sequence (Fig. 6). Presystolic atrial activity was recorded in the right atrium in 13 of the 14 patients. Only one patient demonstrated atrial presystolic activity from a left atrial location.

Acute drug testing. During tachycardia, intravenous adenosine was administered to 14 patients in incremental doses until a transient period of second or third degree AV block was observed (mean 99 ± 38 μg/kg, range 37.5 to 150). Despite the induction of high grade AV block in all patients, no tachycardia was terminated and there was no effect on atrial cycle length in 13 patients. In one patient atrial tachycardia converted to atrial flutter after adenosine administration and, hence, the effect of the drug on intraatrial reentrant tachycardia cycle length could not be assessed. Intravenous verapamil (5 to 10 mg) was administered to nine patients during sustained atrial tachycardia; it did not terminate tachycardia or alter atrial cycle length in any patient despite increasing the degree of AV block (Fig. 7).

Long-term therapy and follow-up (Table 3). Patients underwent 2.5 ± 1.5 unsuccessful therapeutic trials with various drugs including digoxin, verapamil, a beta-blocker and type 1a antiarrhythmic agents. Drugs that increased AV node refractoriness often resulted in immediate symptomatic improvement as a result of a decrease in ventricular response during the arrhythmia. However, these drugs did not prevent recurrence of intraatrial reentry or alter the tachycardia cycle length, and stable long-term rate control was not achieved.

The final discharge drug regimen was chosen on clinical grounds without repeat programmed stimulation, and patients were followed up for symptomatic clinical recurrence. In 11 patients one or more type 1a antiarrhythmic agents were unsuccessful clinically, and these patients were eventually treated with amiodarone at a dose of 326 ± 145 mg/day

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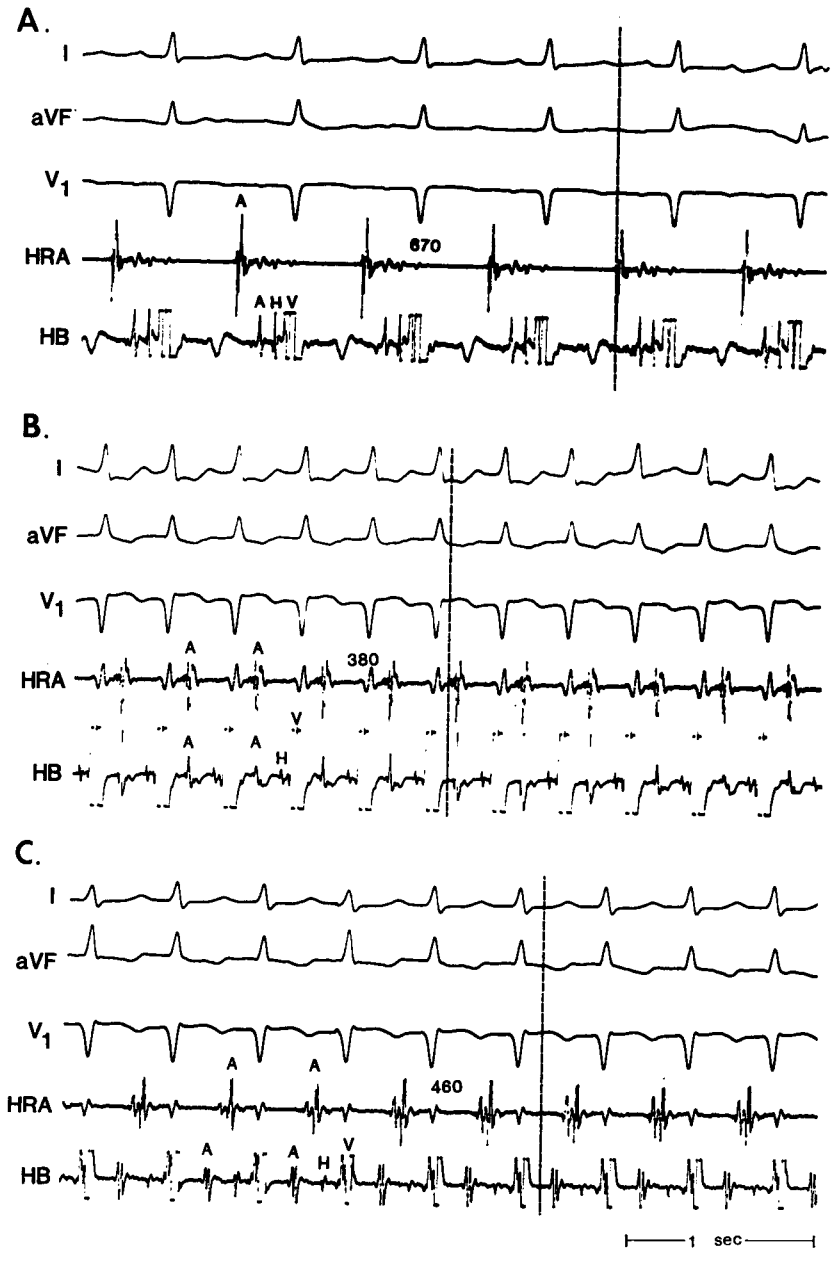


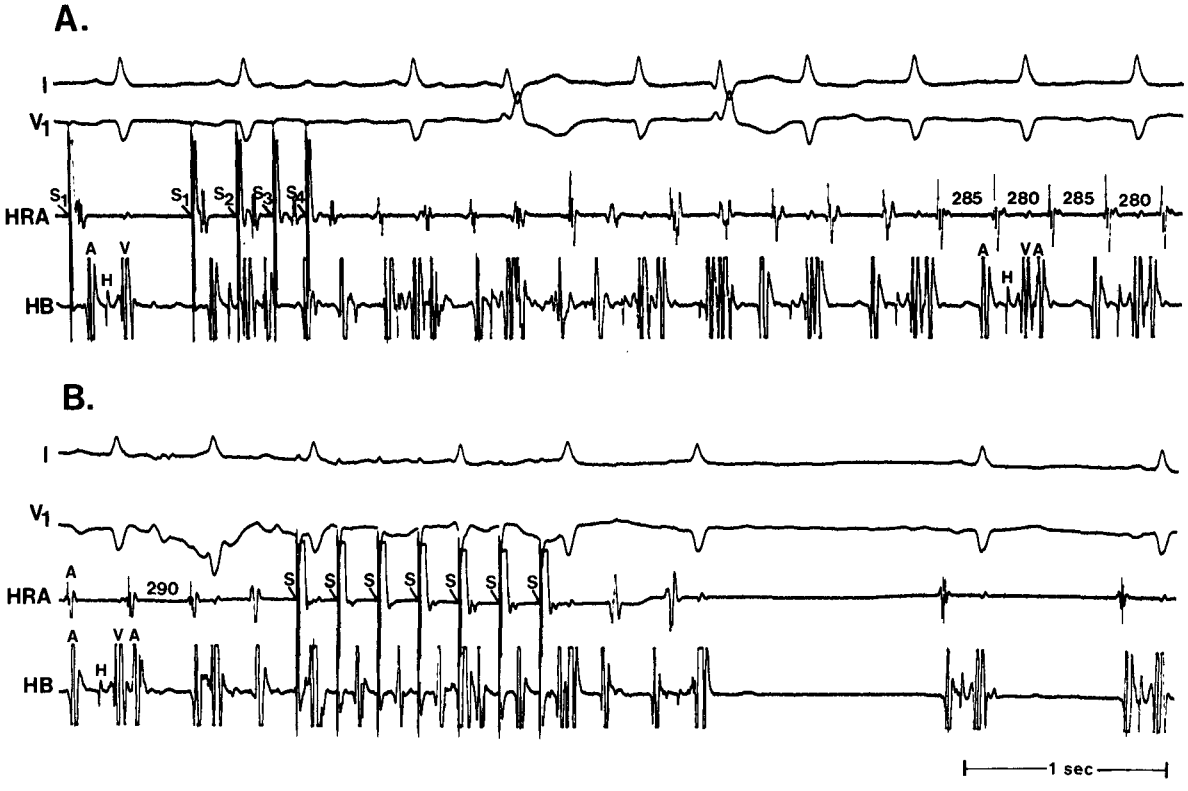
Figure 3. Surface electrocardiographic (ECG) leads I, aVF and V_1 and intracardiac electrograms from the high right atrium (HRA) and the region of the His bundle (HB). The **vertical line** represents the onset of the surface ECG P wave. **Panel A** shows normal sinus rhythm and a normal atrial activation sequence with the high right atrial electrogram onset coincident with the P wave onset. **Panel B** demonstrates intraatrial reentrant tachycardia with a cycle length of 380 ms. The atrial activation sequence is different from that seen during sinus rhythm. **Panel C** demonstrates another intraatrial reentrant tachycardia in the same patient, with a 460 ms cycle length and another unique atrial activation sequence. Electrograms have been retouched.

(range 100 to 600) after an appropriate loading period. Six patients were treated chronically with a type Ia agent alone or in combination with digoxin or a beta-blocker. The remaining two patients had high grade AV block that developed spontaneously or was induced with transcatheter electrical ablation of the His bundle, and were treated with permanent ventricular pacing.

During a follow-up period of 32 ± 20 months (range 3 to 65), all but two patients had successful suppression of their intraatrial reentrant tachycardia. One patient stopped taking procainamide and continues to have recurrent symptomatic atrial tachycardia despite therapy with digoxin and pindolol. The second patient had a favorable initial response to amiodarone (400 mg/day), but developed amiodarone pulmonary toxicity; she has since undergone successful transcatheter electrical His bundle ablation. Despite good control of their atrial arrhythmias, nine patients died during the follow-up period. Causes of death included congestive heart failure (two patients), pulmonary embolism (one patient), intractable ventricular tachycardia (one patient) and noncardiac disease (five patients).

Discussion

Although intraatrial reentry has been accepted as one of several mechanisms potentially responsible for recurrent supraventricular tachycardia, few reports (1,4–6,8) describing the clinical and the electrophysiologic characteristics of patients with this arrhythmia are available. The observations contained in the present study document that supraventricular tachycardia due to intraatrial reentry is a distinct syndrome and that an understanding of the mechanism of



this arrhythmia, as previously defined, is necessary for optimal clinical management of these patients.

Clinical features. The ECG features of the tachycardias in the patients in this series are distinctive and usually allow the arrhythmias to be differentiated from the other more common forms of paroxysmal supraventricular tachycardia.

Figure 4. Patient 13. Surface electrocardiographic leads I and V_1 and intracardiac electrograms from the high right atrium (HRA) and the region of the His bundle (HB). **Panel A** demonstrates initiation of sustained intraatrial reentrant tachycardia with triple atrial extrastimuli. Note the minor degree of cycle length variability. **Panel B** demonstrates termination of the atrial tachycardia by a burst of rapid atrial pacing (S). Electrograms have been retouched.

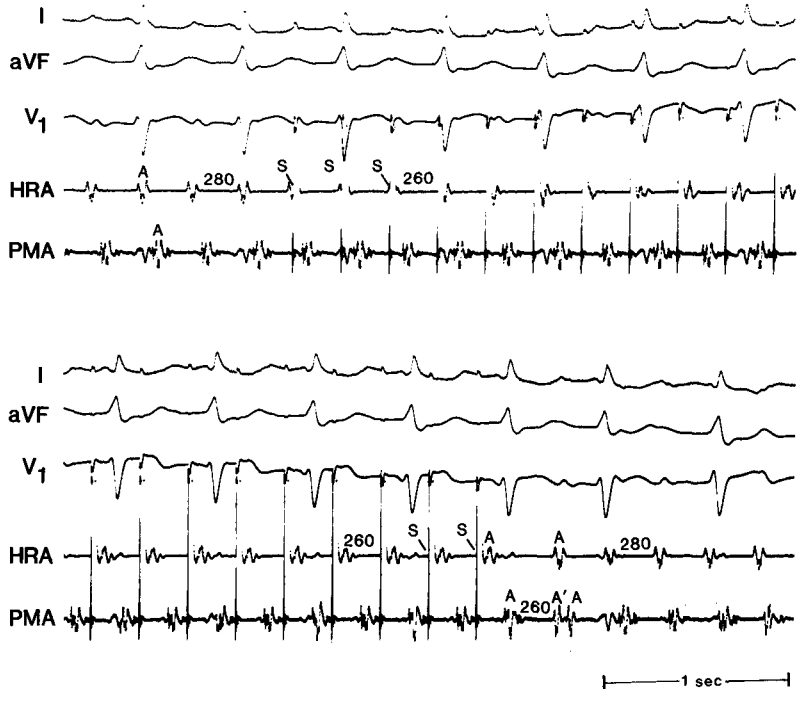


Figure 5. Surface electrocardiographic leads I, aVF and V_1 and intracardiac electrograms from the high right atrium (HRA) and the low posteromedial right atrium (PMA) during intraatrial tachycardia with a 280 ms cycle length. The **upper panel** demonstrates onset of rapid atrial pacing (S) at a cycle length of 260 ms from the high right atrial catheter. The continuation in the **lower panel** shows cessation of the paced drive with fractionated local electrical activity (A'A) measured from the low posteromedial right atrial electrode. The return cycle length of the low posteromedial right atrial electrogram (260 ms) is identical to the pacing cycle length, thus demonstrating one criterion for entrainment.

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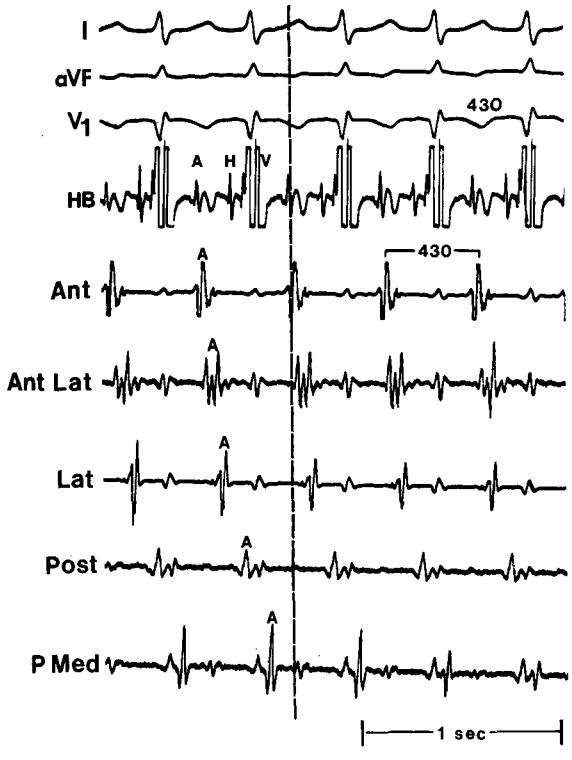

Figure 6. Catheter map of selected right atrial sites during sustained intraatrial reentrant tachycardia. **Top to bottom**, the tracings represent surface electrocardiographic (ECG) leads I, aVF and V_1 and right atrial electrograms from the region of the His bundle (HB), anterior (Ant), anterolateral (Ant Lat), lateral (Lat), posterior (Post) and posteromedial (P Med) segments. The **vertical line** represents the onset of the P wave on the surface ECG. Electrograms have been retouched.

The most helpful finding for localizing the arrhythmia origin was the documentation of second degree AV block during sustained tachycardia. This finding either occurred spontaneously or could be induced by pharmacologic agents or physiologic maneuvers during the tachycardia. Other ECG findings were less helpful; RP′ intervals were variable and no characteristic P wave configuration pertained to all patients. Several patients manifested more than one P wave configuration during different episodes of tachycardia.

In many cases, the recognition of sustained tachycardia with AV block led to an initial clinical diagnosis of "paroxysmal atrial tachycardia with block" due to digoxin toxicity. Although, historically, atrial tachycardia with block has often been thought to be a specific marker for digitalis toxicity (9,10), the arrhythmias in this series were not clinically improved after withdrawal of digoxin and could usually be reinitiated by stimulation in the total absence of cardiac glycosides. Despite the perception that digitalis toxicity accounts for the majority of cases of atrial tachycardia with block (11), intraatrial reentry is more prevalent in our referral center.

Other clinical features served to differentiate intraatrial reentrant tachycardia from other forms of paroxysmal supraventricular tachycardia. Our patients frequently had clinically significant structural heart disease, and 17 of the 19 patients had concomitant arrhythmias associated with conduction abnormalities within atrial or ventricular myocardium. The high mortality rate in this group of patients during the follow-up period underscores their poor overall cardiac and medical condition. As in patients with recurrent sustained ventricular arrhythmias in the setting of structural abnormalities of the ventricular myocardium, it appears that our patients frequently had the potential for several different morphologically distinct atrial arrhythmias as a result of large areas of electrically abnormal atrial tissue.

Previous studies. Previous reports (1,5,6,8) of patients with intraatrial reentrant tachycardia have been limited by small numbers of patients, with limited or no follow-up data reported. Coumel et al. (4) reported 20 cases of intraatrial reentrant tachycardia, but the majority of their patients had no apparent documentation of this arrhythmia outside the setting of invasive electrophysiologic testing; the patients were young and had no structural heart disease. The clinical and electrophysiologic characteristics of the patients in our series are similar to those observed in patients with related rhythm disorders. Like patients with clinical sustained atrial flutter, patients with intraatrial reentrant tachycardia had a high prevalence of underlying structural heart disease, and their tachycardia could be initiated and terminated by appropriate atrial extrastimuli or overdrive pacing (12,13). The majority of these patients had tachycardia refractory to type 1 antiarrhythmic drugs, but similar to reports (14,15) of patients with drug-resistant atrial flutter or fibrillation, they responded well to amiodarone therapy. A series of patients with another variety of reentrant tachycardia, sinoatrial reentrant tachycardia (16), had clinical and electrophysiologic characteristics very similar to those seen in our series.

Mechanisms. The observation that spontaneous or pharmacologically induced AV block during ongoing tachycardia failed to alter the cycle length or terminate the rhythm suggests that the AV node was not a requisite limb of the reentrant circuit. The configuration of the P waves and of the atrial activation sequences observed during tachycardia was distinct from that seen during sinus rhythm, thus excluding the diagnosis of sinoatrial reentrant tachycardia (16). As such, the location of these rhythms appears to be within the atrium.

Reentry as the mechanism for the tachycardias in the present series of patients may be inferred by their initiation and termination by programmed stimulation (12,17–19). Although criteria for entrainment were not demonstrated, these are often dependent on pacing from a critical site (20,21). In the present study, pacing was usually performed from the high right atrium only. Also, the low amplitude of P waves (as opposed to QRS complexes) during tachycardia precluded reliable application of the "progressive fusion" criterion during attempts to demonstrate transient entrainment. However, termination of the tachycardias by rapid

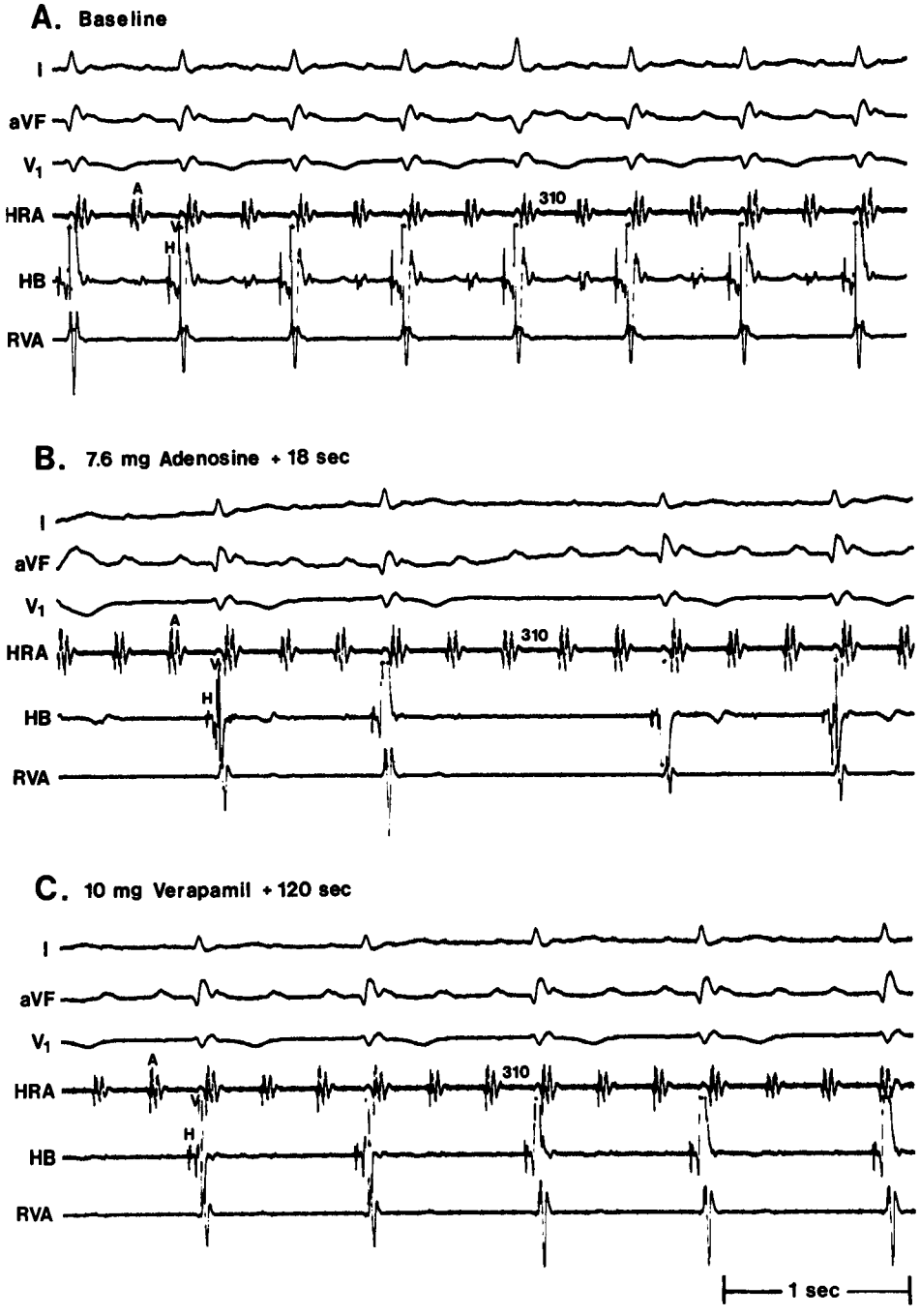

Figure 7. Patient 4. Surface electrocardiographic leads I and V_1 and intracardiac electrograms from the high right atrium (HRA), the region of the His bundle (HB) and the right ventricular apex (RVA) during an episode of sustained intraatrial reentrant tachycardia. **Panel A** shows the atrial tachycardia at baseline study, with an atrial cycle length of 310 ms. **Panel B**, 18 s after central venous injection of 7.6 mg of adenosine, the patient developed high grade atrioventricular (AV) block, but no alteration of the atrial cycle length was observed. **Panel C**, After the adenosine effect had dissipated, 10 mg of verapamil was administered intravenously. Two minutes after injection, AV block had increased from 2:1 to 3:1, but atrial cycle length remained unchanged at 310 ms.

atrial pacing implies that transient concealed entrainment likely did occur.

The response to programmed electrical stimulation does not allow one to differentiate between rhythms due to reentry and those caused by triggered activity (22). In vitro (23,24) and clinical (25,26) studies suggest that triggered activity is inhibited by verapamil or beta-adrenergic blocking agents. Because the doses of these agents used in the present study were moderate, strong conclusions cannot be made, but the insensitivity of these rhythms to these agents in all patients studied suggests that the arrhythmia mechanism was unlikely to be due to triggered automaticity.

Table 3. Long-Term Therapy in 19 Patients

	No. of Patients
Quinidine (824 ± 197 mg/day)	3
Plus digoxin	(1)
Plus digoxin/atenolol	(1)
Disopyramide (600 mg/day) plus digoxin	1
Procainamide (3,000 mg/day)	2
Amiodarone (33 ± 150 mg/day)	11*
AV block with permanent VVI pacing	2†

*One patient developed amiodarone pulmonary toxicity and subsequently underwent His bundle ablation. †One patient developed spontaneous high grade atrioventricular (AV) block, and the second underwent catheter His bundle ablation.

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Conclusion. Intraatrial reentrant tachycardia is a frequent cause of supraventricular tachycardia unresponsive to conventional therapy. Its clinical presentation may be subtle, and its mechanism is often misdiagnosed. It is usually associated with underlying structural heart disease and other ventricular and supraventricular arrhythmias. Concomitant atrial flutter or fibrillation is seen in a majority of these patients, suggesting that similar substrates are required for propagation of these arrhythmias. The diagnosis of intraatrial reentrant tachycardia may be facilitated by production of high grade AV block, but long-term therapy with agents chiefly affecting AV node conduction alone is not effective. Intraatrial reentrant tachycardia may be refractory to many antiarrhythmic drugs, but may respond to long-term therapy with low doses of oral amiodarone.

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