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Use of P Wave Configuration During Atrial Tachycardia to Predict Site of Origin

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Objectives. **This study sought to construct an algorithm to differentiate left atrial from right atrial tachycardia foci on the basis of surface electrocardiograms (ECGs).**

Background. **Atrial tachycardia is an uncommon form of supraventricular tachycardia, often resistant to drug therapy.**

Methods. **A total of 31 consecutive patients with atrial tachycardia due to either abnormal automaticity or triggered rhythm underwent detailed atrial endocardial mapping and successful radiofrequency catheter ablation of a single atrial focus. P wave configuration was analyzed from 12-lead ECGs during tachycardia during either spontaneous or pharmacologically induced atrioventricular block. P waves inscribed above the isoelectric line (TP interval) were classified as positive, below as negative, above and below (or conversely, below and above) as biphasic and flat P waves as isoelectric (0). In 17 patients the tachycardia was located in the right atrium: crista terminalis (n = 4); right atrial appendage (n = 4); lateral wall (n = 4); posteroinferior right atrium (n = 3); tricuspid annulus (n = 1); and near the coronary sinus (n = 1). In 14 patients, atrial tachycardia was located in the left atrium: at the entrance of the right (n = 6) or left (n = 4) superior pulmonary veins; left inferior pulmonary vein (n = 1); inferior left atrium (n = 1); base of left atrial appendage (n = 1); and high lateral left atrium (n = 1).**

Results. **There were no differences in P wave vectors between sites at the right atrial lateral wall versus the right atrial appendage or between sites at the entrance of right versus left superior pulmonary veins. However, analysis of P wave configuration showed that leads aVL and V_1 were most helpful in distinguishing right atrial from left atrial foci. The sensitivity and specificity of using a positive or biphasic P wave in lead aVL to predict a right atrial focus was 88% and 79%, respectively. The sensitivity and specificity of a positive P wave in lead V_1 in predicting a left atrial focus was 93% and 88%, respectively.**

Conclusions. **1) Analyses of surface P wave configuration proved to be reasonably good in differentiating right atrial from left atrial tachycardia foci. 2) Leads II, III and aVF were helpful in providing clues for differentiating superior from inferior foci.**

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Atrial tachycardia is an uncommon form of supraventricular tachycardia and is caused by multiple mechanisms. The arrhythmia may be due to abnormal automaticity from one or more atrial foci (1), triggered rhythm (2) or atrial reentry (3). In the present study we included only those patients with abnormal automaticity or triggered rhythms.

Advances in percutaneous radiofrequency catheter ablation have made this technique a new and effective treatment option for patients with atrial tachycardia. A number of studies (4–7) have demonstrated excellent success, with low risk associated with catheter ablative therapy in patients with failed drug therapy. Precise mapping of the earliest atrial activation site is critical for determining the site for ablation and thus the success of the procedure. Prior studies (8,9) examined the P wave configuration in 12-lead electrocardiogram (ECG) recordings using atrial pacing. However, to our knowledge no prior studies have examined the P wave configuration associated with clinical episodes of automatic atrial tachycardia. Prior atrial pacing studies have emphasized the difficulty in using the surface P waves in separating right from left atrial pacing sites. The present study therefore sought to analyze P wave configuration in patients with atrial tachycardia who underwent successful catheter ablation. Our goal was to differentiate ectopic foci originating in left or right atrium on the basis of surface ECG findings.

Methods

Patients. The protocol was approved by the Committee on Human Research, and written informed consent was obtained from each patient or his or her parents. We excluded all patients with reentrant atrial tachycardias because the site of successful ablation in a slow conducting zone might be distant from the exit site. Our experience using catheter ablation for

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Table 1. Clinical Characteristics of 31 Study Patients

Pt No.	Age (yr)/Gender	Ectopic Focus	Activation Time (ms)	Structural Heart Disease
1	8/F	RA appendage	−50	None
2	5/F	RA appendage	−35	None
3	74/F	RA appendage	−60	Mild idiopathic cardiomyopathy
4	15/M	Mouth of RA appendage	−35	None
5	52/F	High lateral RA	−25	None
6	64/F	High anterolateral RA	−14	Cardiomyopathy
7	36/F	High lateral aspect of RA	−38	None
8	18/F	Inferoposterior lateral	−71	None
9	83/F	Anterolateral RA	−20	None
10	30/F	Posterolateral RA	−20	Cardiomyopathy
11	43/F	Midlateral RA	−45	Aortic and mitral valve replacement, LA dilation
12	14/M	Midanterior RA	−45	Tachycardia-related cardiomyopathy
13	32/F	Low posterior RA	−45	None
14	68/F	Low posterior RA	−60	None
15	55/M	Right posterior wall, near IVC	−110	Left ventricular hypertrophy, slightly enlarged LA
16	39/F	Near inferior tricuspid annulus	−32	Tricuspid insufficiency, enlarged RA
17	45/F	Coronary sinus os	0	None
18	64/M	Right superior PV	−32	Hypertensive disease
19	55/M	Right superior PV	−56	Ischemic cardiomyopathy (EF 40%)
20	45/F	Right superior PV	−49	None
21	41/F	Right superior PV	−40	None
22	30/M	Right superior PV	−20	None
23	35/M	Right superior PV	−34	None
24	43/F	Left superior PV	−51	None
25	32/F	Left superior PV	−20	None
26	26/M	Left superior PV	−30	Tachycardia-related cardiomyopathy
27	38/M	Left superior PV	−50	Tachycardia-related cardiomyopathy
28	50/M	Low left LA	−37	None
29	14/M	Base of LA appendage	−40	Tachycardia-related cardiomyopathy
30	24/M	Left inferior PV	−59	None
31	25/M	High lateral LA	−110	Pulmonary stenosis, s/p Glenn procedure

Activation time = time interval between atrial activation at successful site and earliest P wave recorded from 12-lead surface electrocardiogram; EF = ejection fraction; F = female; IVC = inferior vena cava; LA = left atrium; M = male; Pt = patient; PV = pulmonary vein; RA = right atrium; s/p = status post.

both reentrant and automatic atrial tachycardia has been described elsewhere (7). Thirty-one consecutive patients with automatic or triggered atrial tachycardia from January 1991 to June 1994 were considered for inclusion in the study (13 male, 18 female; age range 5 to 83 years, mean [±SD] 39 ± 20) (Table 1). Five other patients were excluded from the study either because of unsuccessful ablation (three patients) or because P wave configuration during tachycardia could not be defined (two patients). When necessary, carotid sinus message or adenosine was used to create atrioventricular (AV) block and to allow for P wave analysis.

Electrophysiologic testing. Antiarrhythmic drugs were terminated for 5 half-lives before study and ablation. The procedure was performed in the postabsorptive state with mild intravenous sedation using fentanyl and midazolam. Quadripolar, hexapolar or decapolar catheters (6F or 7F) were placed into the high right atrium, low septal right atrium (for recording a His bundle electrogram), right ventricular apex and coronary sinus. A transseptal puncture was performed using a Brockenbrough needle and Mullin sheath (10,11), or a patent foramen ovale was crossed for patients requiring left atrial recordings. Surface ECGs and intracardiac electrograms (filtered from 30 to 250 Hz) were recorded on either an electrostatic paper recorder (paper speed 100 mm/s) or a computer-based digital amplifier/recorder system with optical disk storage (ART Inc.).

Single and double programmed extrastimuli were introduced in patients whose arrhythmia was not present at the time of electrophysiologic study. Stimuli were delivered after an 8 beat drive at two cycle lengths at two atrial sites and atrial decremental burst pacing to the cycle length at which two-to-one capture occurred. Isoproterenol was given (0.5 μg/min or 0.025 μg/kg body weight per min in children) to patients whose tachycardia was not sustained. The dose was increased every 5 min until heart rate increased by 40%, or tachycardia became sustained either spontaneously or with pacing.

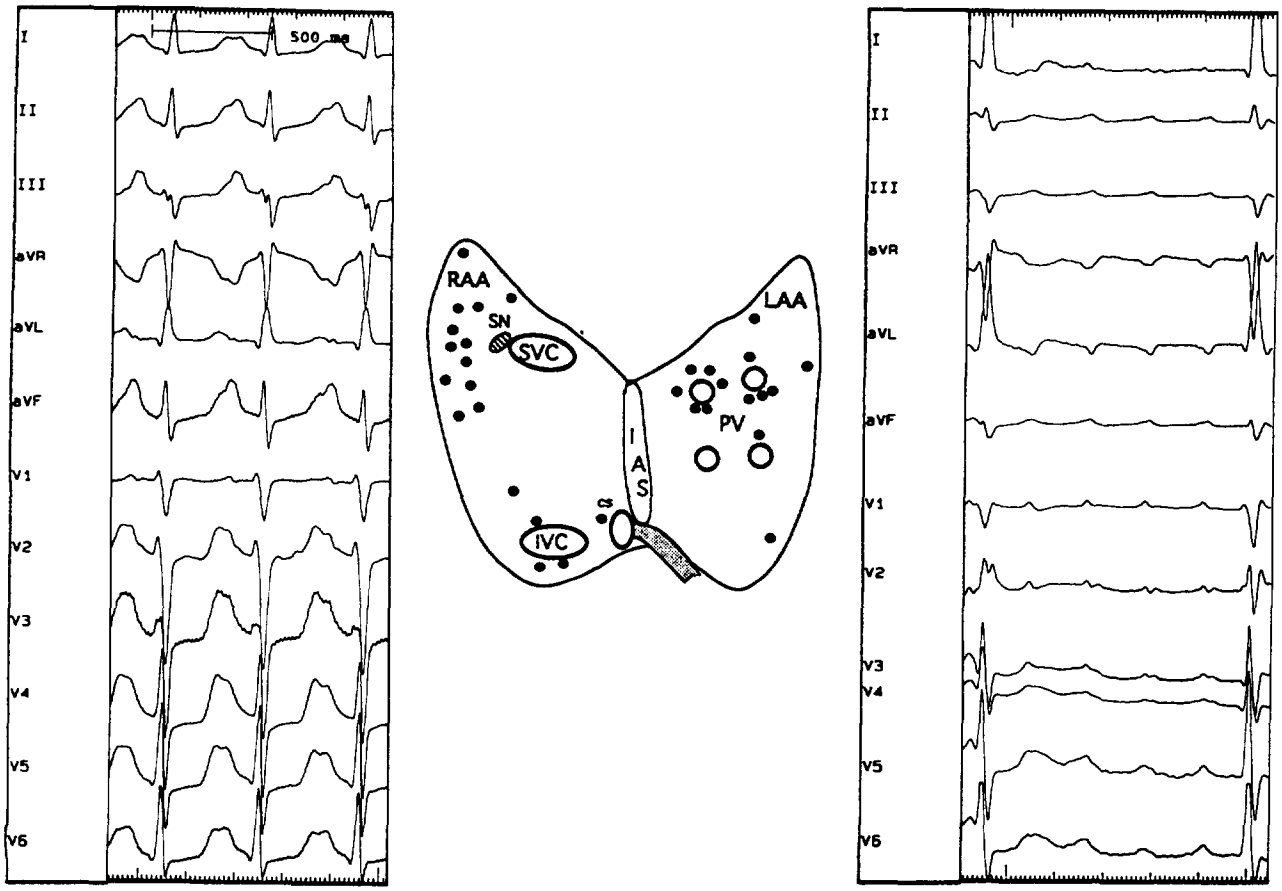


Figure 1. Schema showing atrial tachycardia sites and representative 12-lead electrocardiograms from Patient 5 with a right atrial appendage focus **(left)** and Patient 19 with a right superior pulmonary vein focus. CS = coronary sinus; IAS = interatrial septum; IVC = inferior vena cava; LAA = left atrial appendage; PV = pulmonary veins; RAA = right atrial appendage; SN = sinus node; SVC = superior vena cava.

Automatic atrial tachycardia was diagnosed using the following established criteria (12): 1) prolonged or incessant episodes of atrial tachycardia frequently showing "warm-up" at initiation and "cool down" at termination; 2) abnormal P wave axis and configuration during tachycardia and atrial activation sequence suggesting a nonsinus origin; and 3) inability to reliably initiate or terminate tachycardia with atrial programmed stimulation. Tachycardia initiation usually required isoproterenol or atrial overdrive pacing, or both, suggesting the possibility of a triggered rhythm. The possibility of AV reentry by persistence of the tachycardia was excluded by spontaneous or pharmacologically induced AV block. Atrioventricular node reentry was believed to be unlikely because of P wave configuration during tachycardia, presence of episodic AV block, lack of dual AV node function curves as well as the relation of the P wave to the QRS complex during tachycardia. Finally, a discrete atrial focus was successfully ablated for all patients included in the study. The technique used for intracardiac mapping and radiofrequency ablation has been described in detail elsewhere (7).

P wave analyses. Surface 12-lead ECG recordings were obtained for all patients. Spontaneous or pharmacologically induced AV block allowed analyses of P wave configuration independent of the T wave. P waves were classified into four types: 1) *positive* = P waves showing deflections above the isoelectric line; 2) *negative* = P waves inscribed below the isoelectric line; 3) *biphasic* = P waves showing both positive and negative components; and 4) *isoelectric* = flat P waves. The height or depth of the P wave was measured from the P wave peak or nadir to the isoelectric line (TP interval). For purposes of this study, positive P wave deflections between 0 and 1 mm were recorded as (+), those between 1 and 2 mm as 2+. A similar scheme was used for negative P wave deflections. Biphasic P waves were recorded as (±) and isoelectric as (0). Only those P waves clearly isolated from the preceding T waves were used for the analyses. Each patient's P wave configuration during tachycardia was compared with that during sinus rhythm.

Statistical methods. A chi-square test was used to evaluate the ability of each lead to localize ectopic focus to either the left or right atrium. A p value <0.05 was considered significant. Sensitivity, specificity and positive and negative predictive values were calculated for ECG leads $p < 0.05$.

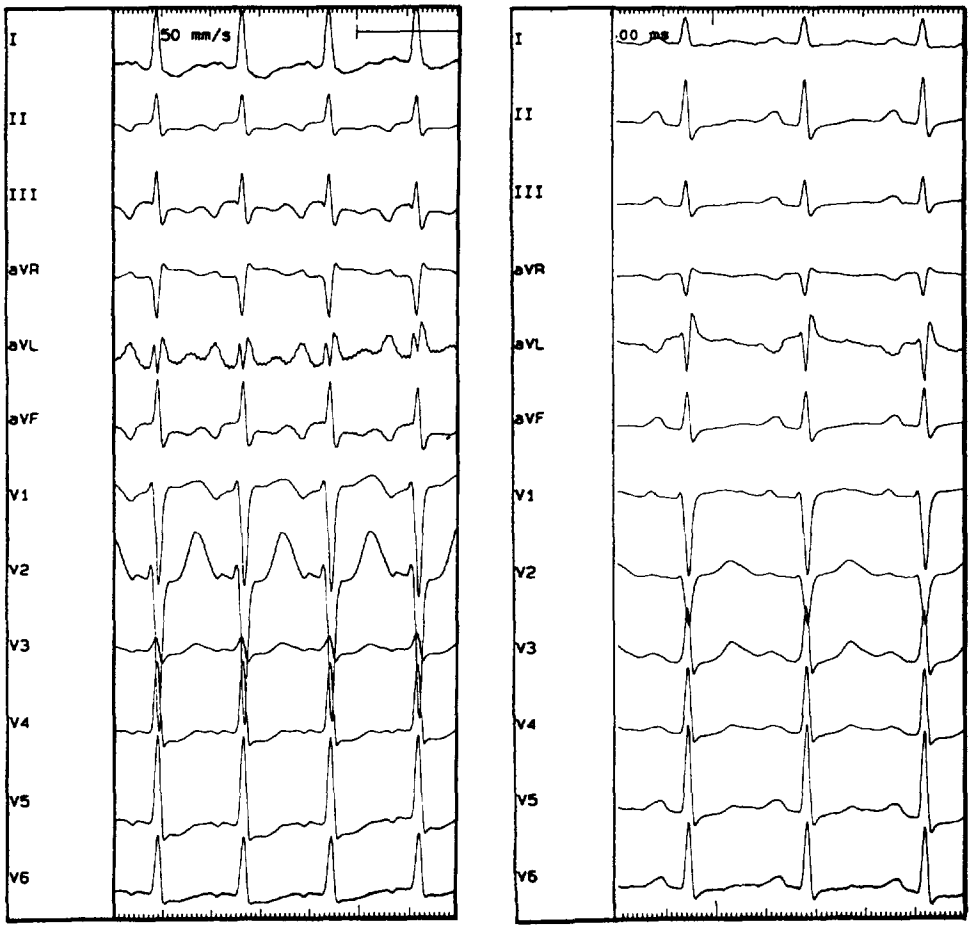


Figure 2. Twelve-lead electrocardiograms from a patient with two different ectopic foci. Right atrial focus (low posterior right atrium **[left]**) showed positive P waves in lead aVL and negative P waves in lead V_1. Left atrial focus (left superior pulmonary vein **[right]**) showed negative P waves in lead aVL and positive P waves in lead V_1.

Results

Patients. A total of 31 atrial foci in 30 patients were successfully ablated. Seventeen patients had right and 14 left atrial tachycardia. Four patients had ectopic foci near the crista terminalis (Fig. 1). Four ectopic foci were located near the right atrial appendage; four were found in the lateral margin of the right atrium and three at the posteroinferior region of right atrium. One patient had a focus near the tricuspid annulus that was located at the mouth of the coronary sinus. Ten of 14 patients with left atrial tachycardia had foci near either the right (6 patients) or left (4 patients) superior pulmonary veins (Fig. 1). One patient had a focus located at the low left atrial wall; one had the focus mapped to the left inferior pulmonary vein; one had the focus located at the high lateral wall; and one had the focus near the base of left atrial appendage. Patients 13 and 25 are the same person because this patient had two distinct foci causing atrial tachycardia. One focus was in the right atrium and the second one in the left atrium. Thus, she was included twice in the study (Fig. 2).

P wave configuration. The P wave polarity and amplitude for all patients are summarized in Table 2. There was a significant correlation between a negative or isoelectric P wave in lead aVL and left atrial foci ($p < 0.001$) and between a positive P wave in lead V_1 and right atrial foci ($p < 0.0005$). The rest of the surface leads did not show significant correlation between P wave configuration and location of ectopic foci in either the right or left atrium. Of 17 patients with right ectopic foci, 14 showed a positive P wave deflection in lead aVL, 2 (Patients 6 and 11) showed a negative deflection, and only 1 (Patient 8) showed a biphasic deflection. Conversely, of 14 patients with left ectopic foci, 10 displayed negative P waves in lead aVL (range −1 to −2 mm), 3 showed positive P waves (Patients 20, 21 and 23), and 1 showed isoelectric P waves. The three patients with positive P waves in lead aVL all had foci near the right superior pulmonary vein.

Lead V_1 also showed significant differences in P wave polarity between right and left atrial foci. Of 13 patients with a left ectopic focus, 12 showed positive P waves (range 1 to 2 mm) in lead V_1, and 1 (Patient 29) had biphasic P waves. Conversely, of 17 patients with right ectopic foci, 15 showed P waves with either totally negative (7 patients) or biphasic configurations (8 patients), and the remaining 2 patients had positive P waves. Leads aVL and V_1 from representative ectopic foci in the present study are shown in Figure 3. In lead I, an isoelectric or negative P wave deflection was associated with a left atrial focus but this finding occurred in only six patients with left atrial tachycardia. Hence, this finding proved to be very specific but insensitive for locating left atrial foci. A

Table 2. P Wave Configuration During Tachycardia

Pt No.	Ectopic Focus	Electrocardiographic Lead									
		I	II	III	aVR	aVL	aVF	V_1	SV_1	V_2	V_3–V_6
RA Focus											
1	RA appendage	2	2	1	−2	1	1	−2	(±)	−2	2
2	RA appendage	1	2	1	−2	1	1	−1	−1	−1	2
3	RA appendage	1	2	1	−2	1	1	(±)	(±)	(±)	2
4	Mouth of appendage	2	2	1	−2	1	2	(±)	(±)	2	1
5	High lateral RA	2	2	(±)	−2	2	2	(±)	(±)	1	1
6	High anterolateral RA	1	1	1	−1	−1	1	1	1	1	1
7	High lateral RA	1	1	1	−1	1	1	2	1	2	1
8	Lateral wall RA	(∓)	1	1	(±)	(∓)	−1	−1	1	−1	1
9	Anterolateral RA	1	1	(±)	−1	1	(±)	−2	(±)	(±)	1
10	Right posterolateral	2	2	1	−2	1	1	−1	(±)	−1	1
11	Midlateral RA	1	2	2	1	−1	(±)	(±)	(±)	1	2
12	Midanterior RA	2	2	2	−2	1	2	(±)	(±)	(±)	*
13	Low posterior RA	1	−2	−2	1	2	−2	−1	1	1	0
14	Low posterior RA	2	2	2	−2	1	2	(±)	−1	1	2
15	Right posterior wall, near IVC	1	−2	−2	1	1	−1	−1	−1	−1	−1
16	Posterior RA, near TA	1	−2	1	0	1	−1	(∓)	1	0	0
17	Coronary sinus	1	−1	1	−1	1	−1	(∓)	(±)	1	1
LA focus											
18	Right superior PV	0	2	2	−1	−1	2	2	(±)	2	2
19	Right superior PV	0	2	1	−1	−1	2	2	1	1	1
20	Right superior PV	2	1	0	−1	1	1	1	(±)	1	1
21	Right superior PV	1	1	1	−1	1	1	1	(±)	2	2
22	Right superior PV	2	3	3	−2	−1	2	1	(±)	2	2
23	Right superior PV	1	2	1	−1	1	2	2	(±)	2	2
24	Left superior PV	1	2	2	(±)	−1	2	2	1	3	2
25	Left superior PV	0	2	2	−1	−2	1	1	1	2	1
26	Left superior PV	1	2	2	(±)	−1	2	2	(±)	2	2
27	Left superior PV	0	2	2	−1	−1	2	2	(±)	2	2
28	Low left atrial	−1	−1	1	1	0	−1	1	(±)	0	−1
29	Base of LA appendage	(∓)	2	3	−2	−2	3	(±)	−1	(±)	2
30	Left inferior PV	−1	2	2	−1	−1	2	2	1	2	1
31	High lateral LA	−1	0	0	0	−1	1	1	0	1	1

*P wave varied in the precordial leads: biphasic (±, ∓) in leads V_3–V_5, positive in lead V_6. SV_1 = sinus V_1; TA = tricuspid annulus; other abbreviations as in Table 1.

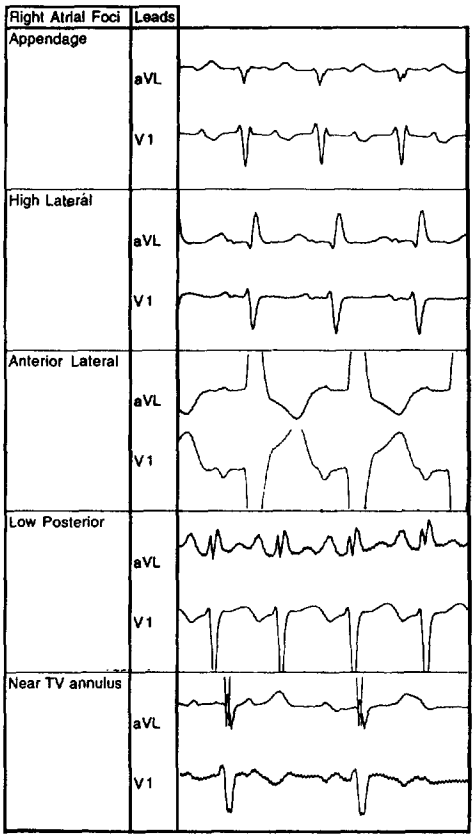
negative P wave in lead I associated with a left atrial focus has been described elsewhere (11).

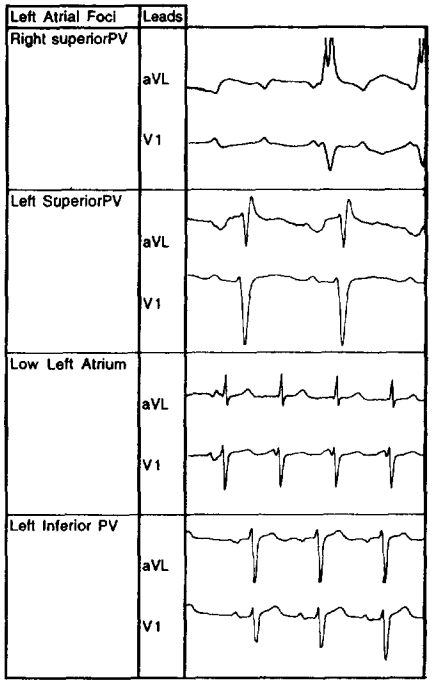
Sensitivity, specificity and predictive accuracy. Table 3 shows the calculated sensitivity, specificity and predictive accuracy of selected leads. Using the criterion that a positive P wave in lead V_1 indicates a left atrial focus results in a sensitivity of 92.9%, specificity of 88.2%, positive predictive accuracy of 86.7% and negative predictive accuracy of 93.8%. One patient (Patient 29) with left atrial tachycardia did not display the P wave patterns previously described.

The criterion that a positive or biphasic P wave in lead aVL indicates a right atrial focus (15 of 17 patients) was associated with a sensitivity of 88.2%, specificity of 78.6%, positive predictive accuracy of 83.3% and a negative predictive accuracy of 84.6%. The two exceptions occurred in patients with foci in the right atrial lateral wall. A positive P wave in lead aVL was also found in patients with foci in the right superior pulmonary vein (Patients 20, 21 and 23). A feature found to be helpful in distinguishing high anterolateral foci (cranial portion of the crista terminalis) from those in the right superior pulmonary vein was a change in P wave configuration in lead V_1 during sinus rhythm versus tachycardia. Patients with foci in the right superior pulmonary vein showed a change in P wave configuration from biphasic in sinus rhythm to a totally positive P wave during tachycardia (Fig. 4). This change was not observed in patients with right atrial anterolateral foci or other right atrial foci. P waves during tachycardia for the four patients (Patients 5, 6, 7 and 9) with a focus near the crista terminalis were compared with those during sinus rhythm. Only one patient (Patient 6) showed similar P wave configuration during sinus rhythm and tachycardia (Fig. 5). We found an overall predictive accuracy of 93.5% by using the combined criteria (Fig. 6) for predicting right atrial foci (positive or biphasic P wave in lead aVL) and an observed change in P wave configuration in lead V_1 characteristic of a right superior pulmonary venous focus. False prediction occurred in two patients (Patients 6 and 11).

Superior versus inferior foci. For patients with either right or left atrial foci, leads II, III and aVF helped to distinguish superior from inferior foci. For the right atrium, seven patients

Figure 3. Representative electrocardiographic tracings from various foci. Only leads aVL and V_1 are shown. **Top,** Representative right atrial foci: appendage (Patient 4), high lateral (Patient 5), anterolateral (Patient 9), low posterior (Patient 13), near tricuspid valve (TV) annulus (Patient 16 with 2:1 atrioventricular [AV] block) and near coronary sinus os (Patient 17). **Bottom,** Representative left atrial foci: right superior pulmonary vein (PV) (Patient 19), left superior pulmonary vein (Patient 25), low left atrium (Patient 28) and left inferior pulmonary vein (Patient 30).





had positive P waves in leads II, III and aVF that were associated with atrial appendage (4 patients) and right superior lateral foci (3 patients). In four patients (Patients 13, 15, 16 and 17) with right atrial foci, the P wave was negative in leads II, III and aVF, and this was correlated with foci in the region of the inferior vena cava or coronary sinus ostium or near the posteroseptal space. One exception proved to be Patient 14, who had an inferior focus in the right atrium (lateral to inferior vena cava) but showed positive P waves in leads II, III and aVF. Similarly, for patients with left atrial foci, a positive P wave in leads II and III (12 patients) was associated with foci around the superior pulmonary veins or left atrial appendage. A negative P wave in leads II and III was seen in one patient (Patient 28) with an inferior left lateral focus. In one patient (Patient 31) with isoelectric P waves in the inferior leads, the focus was at the high lateral wall of left atrium. A negative P wave in lead I was recorded in two patients (Patients 27 and 28) and was associated with a low lateral left atrial focus.

Discussion

Predictive accuracy of ECG criterion. To our knowledge, the present study is the first attempt to correlate P wave configuration with specific atrial foci using successful catheter ablation as the reference standard for location of the atrial focus. In the present study we found P wave configuration in leads aVL and V_1 to be most helpful in discriminating right atrial from left atrial foci. Use of the criterion of a positive P wave in lead V_1 for predicting left atrial foci was associated with a sensitivity of 92.9%, specificity of 88.2%, positive predictive accuracy of 86.7% and a negative predictive accuracy of 93.8%. For the same criteria, lead V_1 had a 90.3% overall predictive accuracy with only three false predictions. Conversely, a sensitivity of 88.2%, specificity of 78.6%, positive predictive accuracy of 83.3%, and negative predictive accuracy of 84.6% resulted when the criteria of positive or biphasic deflection in lead aVL was used for predicting right atrial foci. This criterion had a 83.9% overall predictive accuracy with five false predictions. However, three of the five patients with a false prediction had positive P waves in lead aVL, and foci were all localized to the right superior pulmonary vein. Furthermore, all three patients showed a change in P wave configuration from biphasic during sinus rhythm to a totally positive P wave during tachycardia. This change was not observed in any of the other patients with right atrial foci. When this observation was incorporated along with the criteria for lead aVL, we were able to make an accurate prediction in 93.5% with only two false predictions. Thus, we tailored our algorithm accordingly to give the best overall predictive accuracy based on the results in our patients (Fig. 6). The combination of leads aVL and V_1 was preferred over lead V_1 alone because it resulted in a higher overall predictive accuracy (93.5%).

It is equally instructive to review the apparent discrepant results. Because the left atrium is a posterior midline structure (Fig. 7), its activation would be expected to produce a P wave

Table 3. Sensitivity, Specificity and Predictive Accuracy of Leads aVL and V_1

Criteria	Predicted Atrial Focus	Prediction Results	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Positive or biphasic P waves in lead AVL	RA	TP, 15/17; TN, 11/14	88.2	78.6	83.3	84.6
Positive P waves in lead V_1	LA	TP, 13/14; TN, 15/17	92.9	88.2	86.7	93.8

NPV = negative predictive accuracy (number of correct predictions/number of patients without the given criteria); PPV = positive predictive accuracy (number of correct predictions/number of patients with the given criteria); Sensitivity = true positive/sample size (number of patients with sites in predicted atrium); Specificity = true negative/sample size (number of patients without sites in predicted atrium); TN = true negative (number of patients without electrocardiographic criteria who did not have tachycardia localized to the predicted atrial site); TP = true positive (number of patients with accurate prediction of atrial site); other abbreviations as in Table 1.

vector that is positive for lead V_1. Patient 29 who had a left atrial focus, showed a negative P wave deflection in lead aVL but had a biphasic P wave in lead V_1 instead of the expected positive deflection. The tachycardia focus in this patient was located at the anterobasal portion of the left atrial appendage. Conceivably, the relatively anterior location of this particular focus might explain the discrepant P wave configuration in lead V_1. Three patients (Patients 20, 21 and 23) with foci located at the insertion of the right superior pulmonary vein showed a positive P wave in lead aVL instead of the expected negative deflection. Because of the anatomic proximity, patients with right superior pulmonary venous foci might be expected to show a P wave configuration similar to that for foci arising from the cranial region of the crista terminalis. We found that comparing P wave configuration in lead V_1 during sinus rhythm with that during tachycardia is helpful in distinguishing the two sites when lead aVL fails to differentiate the two. In these cases, patients with an atrial focus near the right superior pulmonary vein often show a change from a biphasic P wave in sinus rhythm to a positive P wave in lead V_1 during tachycardia (Fig. 4). This change was observed in Patients 20, 21 and 23. Findings in Patient 17 are instructive in the sense that this patient showed negative P waves in leads II, III, aVF and V_6. A negative P wave in V_6 is thought by some to be pathognomonic of a left atrial focus (11,12); however, this type of configuration is the rule in activation of the atrium from the region of the mouth of the coronary sinus. For example, this type of P wave pattern is almost always observed for patients with the permanent form of junctional reentrant tachycardia (13). The most likely explanation is that a markedly superior P wave axis may project negative to the V_6 axis. Patients 6 and 11 both had right atrial foci with a negative P wave in lead aVL

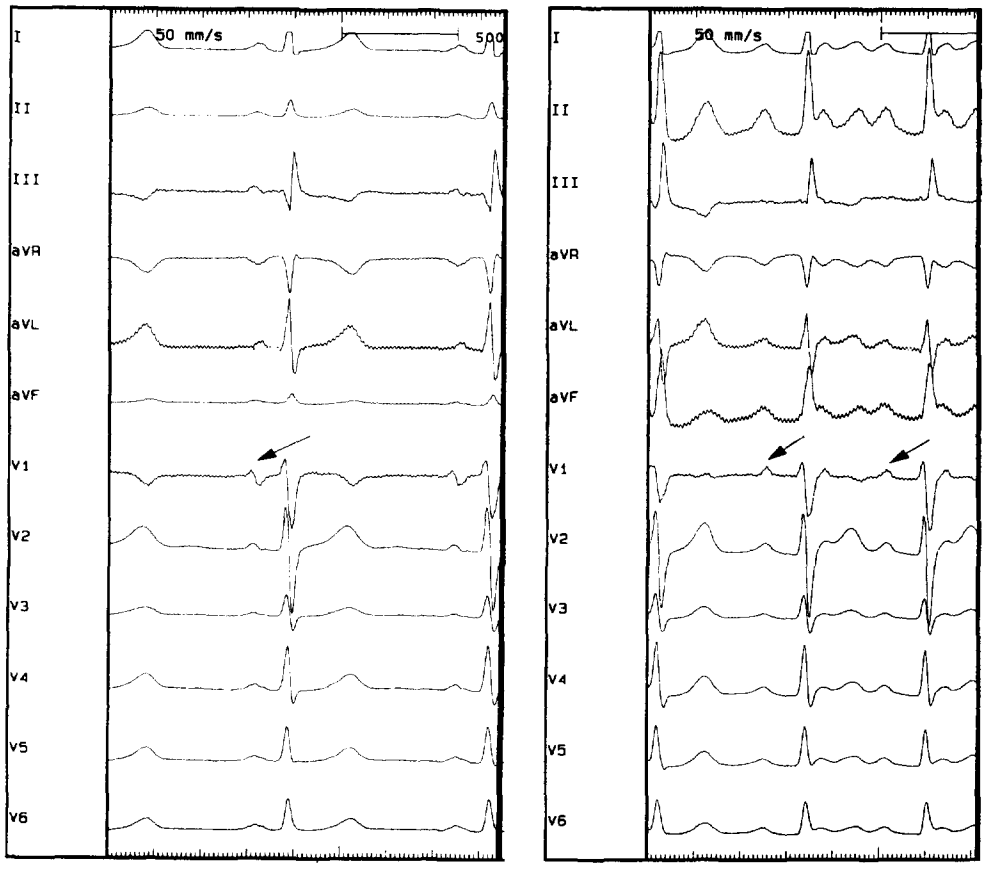


Figure 4. Twelve-lead electrocardiogram from Patient 20 in transition from sinus rhythm **(left)** to tachycardia **(right)**. Patient 20 had a focus near the right superior pulmonary vein. During sinus rhythm, the P wave is biphasic in lead V_1 and positive in lead aVL. During tachycardia, the P wave remains positive in lead aVL, but lead V_1 becomes totally positive **(arrows)**, suggesting a left atrial origin.

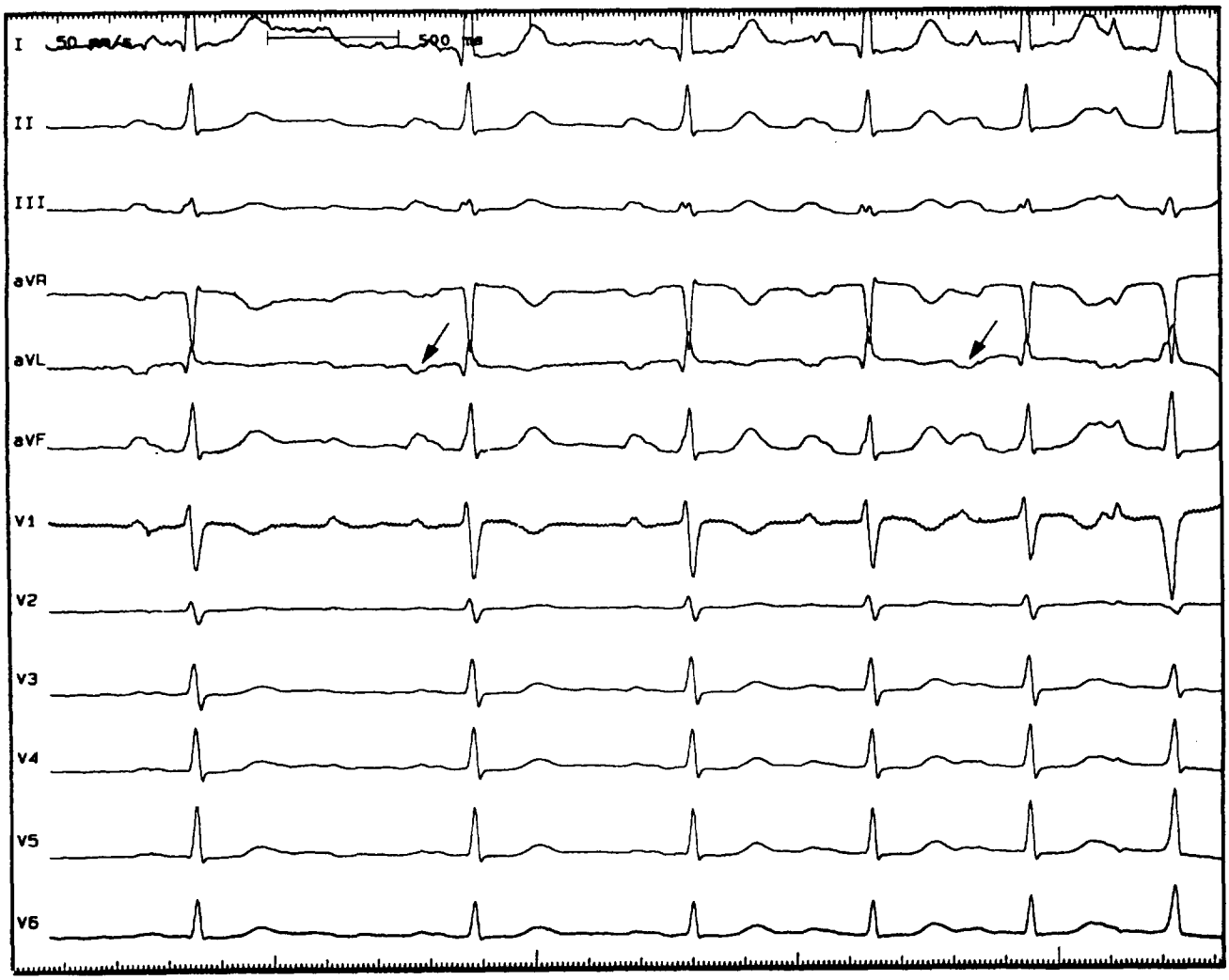


Figure 5. Twelve-lead electrocardiogram from Patient 6 in transition from sinus rhythm to tachycardia. Patient 6 had a focus in the high anterolateral right atrium and displayed a negative P wave in lead aVL during both sinus rhythm and tachycardia (**arrows**). She had structural heart disease with left atrial dilation.

and both had structural heart disease and enlarged atria. Patient 6 showed a negative P wave in lead aVL during both tachycardia and sinus rhythm (Fig. 5). This finding suggested that structural abnormalities may lead to variation in P wave configuration. Patient 11 had marked dilation in the left atrium secondary to valvular heart disease. Again, this finding could explain why this patient's P wave deviates from the criteria.

We also found that patients with posterior right atrial foci tend to have positive P waves in lead V_1, most likely because the posterior right atrium lies just to the right of the left atrium, hence posterior right atrial foci might be expected (like left atrial foci) to be positive in lead V_1. Of note is the clinical finding that patients with AV node reentry may show a positive P wave in lead V_1 (14). None of our patients had foci in the anterior septal region. It should be emphasized that atrial activation proceeds over preferential muscle bundles that determine the atrial activation sequence and hence the P wave configuration (9). These considerations explain why the proposed algorithm, though useful as a first approach, is nevertheless far from perfect.

In addition, P wave polarity in the inferior leads was helpful in distinguishing superior from inferior atrial foci for both right atrial and left atrial foci. Furthermore, we found that an isoelectric or negative P wave in lead I was highly specific for a left atrial focus (7 of 7 patients) but was very insensitive for detection of left atrial foci (7 of 14 foci). Previous studies (14) have emphasized the importance of a negative P wave in lead I, denoting a left atrial focus.

Previous studies. Previous studies used either mechanical or electrical stimulation of the atrium to analyze the relation of P wave configuration to the site of origin of atrial activation. These studies (14–18) emphasized that a negative P wave in lead I or a dome and dart configuration in lead V_1 was relatively specific for foci emanating from the left atrium. In contrast, Massumi and Tawakkol (18) emphasized the wide variability in P wave configuration in response to direct left atrial stimulation. The most extensive study of atrial rhythms

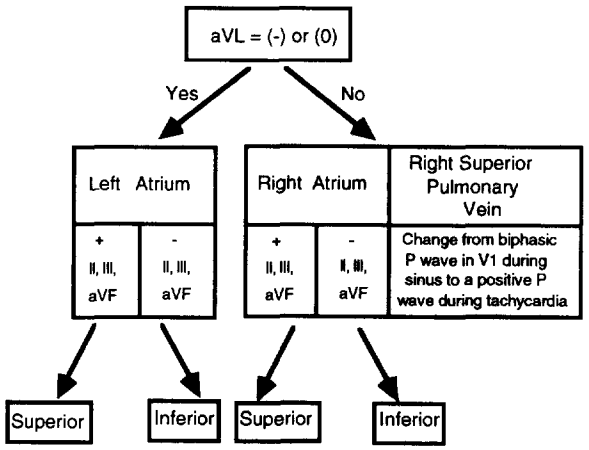


Figure 6. Proposed algorithm for predicting atrial tachycardia foci on the basis of the P wave configuration from 12-lead electrocardiographic recordings. A negative or isoelectric P wave in lead aVL usually indicates a left atrial foci. A positive or biphasic P wave in lead aVL indicates either right atrial or right superior pulmonary vein foci. If a change in lead V_1 configuration is observed from a biphasic P wave in lead V_1 during sinus rhythm to a positive lead V_1 during tachycardia (Fig. 3), then it is likely that the site is the right superior pulmonary vein. A negative P wave in leads II, III and aVF suggests an inferior atrial focus, whereas a positive P wave in leads II, III and aVF suggests a superior atrial focus.

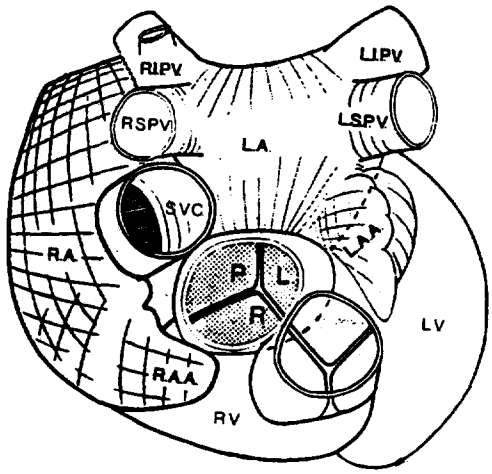


Figure 7. Superoinferior view of the human heart emphasizing the anatomic relation of the left and right atria. The left atrium is in a midline posterior position, whereas the right atrium lies more anterior and lateral. LA = left atrium; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; LV = left ventricle; RA = right atrium; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein; RV = right ventricle; other abbreviations as in Figure 1.

using pacing of 12 discrete atrial sites was published by MacLean et al. (19). They found that P wave configuration was of limited assistance in distinguishing right from left atrial pacing sites. A negative P wave in lead I, for example, was observed only during pacing near the left pulmonary veins and a positive (bifid) P wave was inscribed in lead V_1 only during pacing near the inferior pulmonary veins. MacLean et al. (19) also recorded the polarity of the P wave in lead aVL. They showed that all paced sites in the right atrium produced positive P waves in lead aVL, which agrees with our findings. In left atrial pacing, four of six sites showed either negative or isoelectric P waves in lead aVL. The two sites showing a positive P wave in lead aVL were located in the inferior portion of the left atrium; these sites were not associated with clinical tachycardias in our study. Furthermore, it is difficult to compare our results with those of previous studies because our patients had spontaneous atrial arrhythmias that occurred for the most part in patients with normal atrial anatomy. In contrast, previous studies involved atrial pacing in patients with organic cardiac disease.

Study limitations. The present study showed that analyses of the surface P wave provided good predictive power in distinguishing right from left atrial tachycardia foci. Moreover, such analyses were helpful in distinguishing superior from inferior arrhythmic sites. However, the P wave configuration was of limited value in locating specific sites within the atria. This finding was due to the relatively small sample size as well as the tendency of foci to congregate around specific areas (i.e., the superior pulmonary veins in the left atrium and the crista terminalis in the right atrium). Apparent discrepant results may be due to either distorted atrial anatomy or abnormal conduction from diseased atrial regions. For the most part, our results were obtained in patients with normal atrial anatomy. Hence, these results cannot be extrapolated to those with abnormal atrial anatomy. For these reasons, analyses of the surface ECG cannot supplant the need for careful endocardial mapping, including the use of a coronary sinus catheter.

Our study was limited to patients with either automatic or triggered atrial tachycardia; hence, these results should not be extrapolated to include patients with reentrant atrial arrhythmias in whom a critical zone of slow conduction could be some distance away from tachycardia exit (and P wave onset). In contrast to other reports, only one patient had a focus localized near the atrial septum; hence, we cannot comment on the specific configuration associated with septal foci. It should be emphasized that Waldo et al. (20) reported positive P waves in leads II, III and aVF and a flat P wave in lead aVL during pacing from the inferior right atrium 2 cm anterior to the os of the coronary sinus. A focus in this area (not observed), according to our algorithm, would falsely predict a superior location.

Finally, carotid sinus massage or adenosine was required to best show the tachycardia so that clear P waves were differentiated from antecedent T waves. We cannot exclude the possibility that these maneuvers in and of themselves distort P wave configuration.

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