Focal Atrial Tachycardia
Arising From the Mitral Annulus
Electrocardiographic and Electrophysiologic Characterization

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OBJECTIVES
The study was done to characterize the electrocardiographic and electrophysiologic features of focal atrial tachycardia originating at the mitral annulus (MA).

BACKGROUND
Though the majority of left atrial tachycardias originate around the ostia of the pulmonary veins, only isolated reports have described focal tachycardia originating from the MA.

METHODS
Seven patients of a consecutive series of 172 patients undergoing radiofrequency ablation for focal atrial tachycardia are reported. Electrophysiologic study involved catheters positioned along the coronary sinus (CS), crista terminalis (CT), His bundle, and a mapping/ablation catheter.

RESULTS
All seven patients had tachycardia foci originating from the superior region of the MA in close proximity to the left fibrous trigone and mitral-aortic continuity. These foci demonstrated a characteristic P-wave morphology and endocardial activation pattern. The P-wave morphology in the precordial leads typically showed a biphasic pattern, with an inverted component followed by an upright component. The P-wave was consistently of low amplitude in the limb leads. Earliest endocardial activity occurred at the His bundle region in all seven patients. In general, CS activation was proximal to distal, and mid-CT activation was earlier than high or low CT. Ablation was successful at the superior aspect of the MA in all patients.

CONCLUSIONS
The MA is an unusual but important site of origin for focal atrial tachycardia, with a propensity to be localized to the superior aspect. It can be suspected as a potential anatomic site of tachycardia origin from analysis of P-wave morphology and the atrial endocardial activation sequence map. Using mapping targeted to anatomic structures achieved a high success rate for ablation. (J Am Coll Cardiol 2003;41:2212–9) © 2003 by the American College of Cardiology Foundation

Atrial tachycardia (AT) is a well-recognized but relatively uncommon cause of supraventricular tachycardia, and curative radiofrequency ablation (RFA) of this arrhythmia has been extensively described (1). Intriguingly, the advent of RFA for focal AT has led to the recognition that these foci are not randomly distributed but rather demonstrate a characteristic anatomic distribution (2). In the right atrium (RA), foci tend to occur along the long axis of the crista terminalis (CT), in the para-Hisian region, and around the ostium of the coronary sinus (CS) (3). Recently ATs originating from around the tricuspid annulus have been characterized (4). However, there are only isolated reports of ATs occurring from the mitral annulus (MA) (5). In this study, we characterize the electrocardiographic (ECG) and electrophysiologic features of focal AT originating from the MA in a consecutive series of patients undergoing RFA.

METHODS

Study population. The study population included seven patients of a consecutive series of 172 patients undergoing RFA for focal AT between July 1996 and July 2002 at the Royal Melbourne Hospital. All patients had clinically documented sustained AT for which they were having RFA. Focal AT is “characterized by radial spread of activation and endocardial activation not covering the whole cycle. Ablation of the focus of origin interrupts the tachycardia” (1).

Patient preparation. All patients underwent electrophysiologic study following the provision of informed written consent. The study was approved by the Melbourne Health Research Ethics Committee. Patients were studied in the fasted awake state with minimal use of sedation. All antiarrhythmic drugs were ceased a minimum of five half-lives before the procedure. All antiarrhythmic drugs were ceased a minimum of five half-lives before the procedure. No patients were taking amiodarone.

Catheter positioning. Catheter positioning and the approach used in our laboratory for RFA of AT have been previously published (4). In brief, catheters were positioned at the following locations: 1) CS positioned with the proximal bipole at the CS os; 2) CT positioning assisted by intracardiac echocardiography (9 MHz); 3) His bundle; and 4) mapping and RFA catheter. Atrial tachycardia was...
diagnosed using standard electrophysiologic criteria (1). Mapping of the earliest site of endocardial activity relative to surface P-wave was performed with a 4-mm tip mapping and RFA catheter. When a left-sided origin was suspected (and in the absence of a patent foramen ovale), transeptal puncture was performed. Following left atrium (LA) access, intravenous (IV) heparin maintained the ACT at >250 s.

Bipolar intracardiac electrograms filtered between 30 and 500 Hz were recorded and stored digitally on a computerized system simultaneously with 12-lead surface ECG. Offline analysis was performed using on-screen digital callipers at 150 mm/s speed.

**Mapping of AT.** Anatomic localization of the atrial focus was performed during tachycardia or atrial ectopy by analysis of: 1) surface ECG P-wave morphology; 2) RA activation sequence during tachycardia (3,4); 3) paced activation sequence mapping (6); and 4) point mapping to locate site of earliest endocardial activation relative to surface P-wave onset. In all patients, before performing transeptal puncture, the RA was systematically mapped with the RFA catheter to include the following regions not covered by the standard multipolar catheters: high, mid, and low lateral RA; tricuspid annulus; high, mid, and low septum, particularly the region of Bachman’s bundle; the superior vena cava, and the region of the triangle of Koch. Right atrial mapping was particularly focused around the region of earliest endocardial activation. Transeptal puncture was performed when earliest activation was recorded in a septal or His bundle location, allowing the possibility that earlier activity would be present on the left atrial side.

**P-WAVE MORPHOLOGY.** Surface 12-lead ECG P-wave morphology was assessed as previously described (7). The P waves were described based on the deviation from baseline during the T-P interval as being: 1) positive(+); 2) negative(−); 3) biphasic: if there were both positive and negative (+− or −+) deflections from baseline; and 4) isoelectric: arbitrarily defined when there was no P-wave deviation from baseline of >0.05 mV.

**RIGHT ATRIAL ENDOCARDIAL ACTIVATION SEQUENCE.** Activation timing was measured from onset of the P-wave in lead II of the surface ECG (arbitrarily assigned a time of 0 ms) to each of the intracardiac bipoles of the catheters listed previously.

**DEFINITIONS.** An MA location for the origin of the tachycardia was defined by:

1. The catheter tip demonstrating the characteristic annular location and motion when viewed in right and left anterior oblique fluoroscopic views at the site of successful RFA. In an annular location, the catheter motion was generally synchronized to the motion of the CS catheter in both fluoroscopic projections.
2. Atrial to ventricular ratio <1 with a ventricular electrogram >0.5 mV at the site of successful RFA.
3. Successful elimination of tachycardia and ectopy by RFA at this site.

**RFA and outcome.** Radiofrequency ablation was performed with continuous temperature feedback control of power output to achieve a target temperature of 60°C for a maximum power of 50 W, at the site of earliest endocardial activity. An irrigated tip catheter was not used. Procedural success was defined by the absence of tachycardia or ectopy 30 min following RFA despite the use of programmed stimulation to three atrial extrastimuli and burst atrial pacing down to a cycle length at which 2:1 atrial capture occurred (with and without isoproterenol).

**RESULTS**

**Patient characteristics.** Of 172 patients who underwent RFA of 182 ATs, 7 (3.8%) were determined to have an AT originating from the MA. Baseline characteristics of these patients are presented in Table 1. Six of seven patients were women (mean age 50 ± 16 years; range 24 to 73 years). One

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age/Gender</th>
<th>Structural Heart Disease</th>
<th>LA Size (mm)</th>
<th>Clinical Presentation</th>
<th>Duration of Symptoms</th>
<th>No. of Antiarrhythmic Drugs Failed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55/F</td>
<td>nil</td>
<td>32</td>
<td>Paroxysmal AT</td>
<td>42 months</td>
<td>2</td>
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<tr>
<td>2</td>
<td>63/F</td>
<td>nil</td>
<td>38</td>
<td>AT with LBBB</td>
<td>4 months</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>24/F</td>
<td>nil</td>
<td>35</td>
<td>Paroxysmal AT</td>
<td>9 months</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>73/M</td>
<td>nil</td>
<td>35</td>
<td>Paroxysmal AT</td>
<td>7 months</td>
<td>2</td>
</tr>
<tr>
<td>5</td>
<td>45/F</td>
<td>nil</td>
<td>45</td>
<td>Paroxysmal AT</td>
<td>9 months</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
<td>41/F</td>
<td>Severe MR/MVP</td>
<td>31</td>
<td>Paroxysmal AT</td>
<td>6 months</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>53/F</td>
<td>nil</td>
<td>36</td>
<td>Paroxysmal AT</td>
<td>12 months</td>
<td>2</td>
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</tbody>
</table>

AT = atrial tachycardia; LA = left atrial; LBBB = left bundle branch block; MR = mitral regurgitation; MVP = mitral valve prolapse.
patient had severe mitral regurgitation, with the remainder having no structural heart disease.

**Tachycardia characteristics.** In three patients, tachycardia was induced by atrial programmed extrastimuli (PES) alone and in one with PES during isoprenaline infusion. In one patient, AT could be induced with PES, but it also occurred spontaneously. In two patients, AT or ectopy from the tachycardia focus occurred spontaneously. The tachycardia characteristics are listed in Table 2.

**P-WAVE MORPHOLOGY.** The P-wave morphology on surface ECG for each patient is presented in Table 3. The P waves were consistently low amplitude in limb leads. In inferior leads, P waves were either isoelectric or upright. However, when upright they were always low amplitude and in all cases of lower amplitude than the sinus P-wave (mean P-wave amplitude in lead II in sinus rhythm 140 μV versus 50 μV in tachycardia, p = 0.001). In lead I, the P-wave was isoelectric in six patients and slightly inverted in one patient. In no cases was an upright P-wave observed in leads I or aVL. The precordial leads had a characteristic biphasic pattern in all seven patients, with an inverted component followed by an upright or isoelectric component. In lead V1 the upright component was dominant but rapidly became low amplitude or isoelectric from lead V2 onwards. Thus, from lead V2 onwards, the initial inverted component was dominant. Representative 12-lead ECGs from three patients are shown in Figure 1.

**Table 2.** Tachycardia Characteristics at Electrophysiology Study and Outcome of Radiofrequency Ablation Procedure

<table>
<thead>
<tr>
<th>Pr. No.</th>
<th>Mode of Tachycardia Initiation/Termination</th>
<th>TCL (ms)</th>
<th>Location on MA</th>
<th>A:V Ratio</th>
<th>Procedural Outcome and Follow-up*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Programmed extrastimuli</td>
<td>365</td>
<td>12:00</td>
<td>1.5:1</td>
<td>successful, no recurrence</td>
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<tr>
<td>2</td>
<td>Programmed extrastimuli + isoprenaline</td>
<td>383</td>
<td>11:00</td>
<td>1:2</td>
<td>successful, no recurrence</td>
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<tr>
<td>3</td>
<td>Nil*</td>
<td>Ectopy only</td>
<td>11:00</td>
<td>1:3</td>
<td>successful, no recurrence</td>
</tr>
<tr>
<td>4</td>
<td>Programmed extrastimuli</td>
<td>291</td>
<td>12:00</td>
<td>1:3:5</td>
<td>successful, no recurrence</td>
</tr>
<tr>
<td>5</td>
<td>Programmed extrastimuli + spontaneous</td>
<td>382</td>
<td>12:00</td>
<td>1:4</td>
<td>successful, no recurrence</td>
</tr>
<tr>
<td>6</td>
<td>Programmed extrastimuli</td>
<td>404</td>
<td>12:00</td>
<td>1:3:5</td>
<td>recurrence at 5 months</td>
</tr>
<tr>
<td>7</td>
<td>Spontaneous</td>
<td></td>
<td>12:00</td>
<td>1:3</td>
<td>successful, no recurrence</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td>365 ± 43</td>
<td></td>
<td>1.28 ± 0.9</td>
<td></td>
</tr>
</tbody>
</table>

*Tachycardia not induced.

TCL = tachycardia cycle length; MA = mitral annulus; A:V Ratio = ratio of atrial to ventricular electrogram amplitude recorded on ablation catheter from site of successful ablation.

**Table 3.** The P-Wave Morphology During Tachycardia

<table>
<thead>
<tr>
<th>Pr. No.</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>aVL</th>
<th>aVR</th>
<th>aVF</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
<th>V5</th>
<th>V6</th>
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<tbody>
<tr>
<td>1</td>
<td>iso</td>
<td>iso</td>
<td>iso</td>
<td>iso</td>
<td>iso</td>
<td>iso</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
</tr>
<tr>
<td>2</td>
<td>iso</td>
<td>pos</td>
<td>pos</td>
<td>iso</td>
<td>iso</td>
<td>iso</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
</tr>
<tr>
<td>3</td>
<td>neg</td>
<td>pos</td>
<td>pos</td>
<td>iso</td>
<td>iso</td>
<td>iso</td>
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<td>-/+</td>
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<td>-/+</td>
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<td>-/+</td>
<td>-/iso</td>
<td>-/iso</td>
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<td>-/iso</td>
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<tr>
<td>5</td>
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<td>neg</td>
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<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
<td>-/+</td>
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<td>-/+</td>
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</tbody>
</table>

ECG = electrocardiographic; iso = isoelectric; neg = negative; pos = positive; -/+ = biphasic (negative then positive).

**ATRIAL ENDOCARDIAL ACTIVATION SEQUENCE MAPPING.** Earliest endocardial activation on the standard catheters (His bundle, CT, and CS) occurred at the His bundle region in 7/7 patients. The mean activation times to P-wave onset were as follows: His bundle -23 ± 11 ms; proximal CS 0 ± 16 ms; distal CS 18 ± 7 ms; high CT 8 ± 11 ms; mid-CT -5 ± 20 ms; and low CT 9 ± 30 ms; p < 0.002. The characteristic atrial endocardial activation sequence is demonstrated in Figures 2A and 2B. In general, for MA tachycardias in this location, CS activation was earlier proximally than distally, and mid-CT activation was earlier than high or low CT activation.

**Radiofrequency catheter ablation.** Mapping for the earliest endocardial activity was performed during tachycardia or with atrial ectopy. Following transeptal puncture, sequential mapping was performed at the pulmonary veins (PVs), left atrial appendage, posterior wall, and MA, with attention to differentiating the left atrial septal from the annular location. In all cases the earliest endocardial atrial activation was located at the superior aspect of the MA (between 11:00 and 12:00) as shown in Figures 3 and 4.

At this site on the MA, endocardial atrial activation preceded the onset of the P-wave by a mean of 43 ± 17 ms. In all cases successful ablation of the AT was achieved. The median number of RFA applications was 8.6. The mean ratio of atrial to ventricular electrogram at the successful ablation site was 1.28 ± 0.8. Mean fluoroscopy time was 37 ± 19 min. No procedural complications occurred. During a
mean follow-up of 17 ± 10 months, there was one recurrence. This occurred five months following ablation, and the patient is currently controlled on medical therapy, having declined repeat intervention.

**DISCUSSION**

This study presents the clinical, electrocardiographic, and electrophysiologic findings in a consecutive series of patients with AT originating at the MA. Although recent studies have demonstrated a distribution of AT foci around the tricuspid annulus, only isolated cases of AT originating from the MA have been reported (5). In recent years, identification of a characteristic distribution of ATs has facilitated mapping targeted to anatomic structures. Using this targeted mapping technique has yielded a high ablation success rate without routine requirement for sophisticated three-dimensional mapping techniques (3,4). In the current study, ablation success was achieved in 100% with late recurrence in 1/7 patients. An appreciation of the MA as an important site of origin of left atrial ATs should further assist mapping targeted to anatomy. In particular, we observed a clustering of foci around the superior aspect of the MA in immediate proximity to the region of the left fibrous trigone. Though the reason for this clustering is unclear, it does allow characterization of the ECG morphology and endocardial activation patterns for foci originating in this region.

**P-wave morphology.** The surface ECG P-wave morphology can provide only a general guide to AT localization. Nevertheless, the morphology was remarkably consistent in patients with MA tachycardia in this series with a biphasic (negative followed by positive) appearance in the precordial leads and a low amplitude appearance in the limb leads.

A prior study observed that a positive P-wave in lead V1 and a negative P-wave in lead aVL was relatively specific for a left atrial focus (7). In a recent study (8) of the paced P-wave morphology from the four PVs in 30 patients, the morphology in lead V1 was always upright. The current study demonstrates that for an LA origin at the superior MA there may be a prominent initial inverted deflection in lead V1 prior to the upright deflection. This initial deflection may be due to the relatively anterior position of the MA compared with the PVs, the downward deflection representing initial forces directed posteriorly and away from lead V1. This observation may therefore aid in differentiating a superior MA tachycardia from a PV location.

In the study by Tang et al. (7), the sensitivity and specificity of a positive P-wave in lead aVL for prediction of a right atrial focus was 88% and 79%, respectively. However, it is well recognized that a positive P-wave in lead aVL is also consistent with a right PV origin (8). In the current study, the P waves in lead aVL and lead I were frequently of very low amplitude and were classified as isoelectric. Whereas this finding is not helpful in differentiating a left atrial from a right atrial origin, it is suggestive of a focus close to the septum. Thus, lateral right atrial ATs demonstrate upright P waves in leads I and aVL (4) and left PV foci tend to be inverted in these leads (although they may also be isoelectric in lead I) (8).

**Right atrial endocardial activation sequence.** Right atrial endocardial activation in this series demonstrated earliest activity to be at the His bundle location in all seven patients (significantly earlier than at all CS sites). However, the His bundle site was only a mean of −23 ± 11 ms ahead of P-wave onset, with surrounding septal sites close to this in

![Figure 1](image-url). The tachycardia P-wave morphology of three patients is shown here. The P-wave in precordial leads is characteristically biphasic, with an inverted component followed by an upright component. The upright component is most prominent in lead V1 but diminishes with transition across the precordium. The limb leads are all of low amplitude, with a minor positive deflection in the inferior leads.
activation time and sites off the septum in the RA becoming progressively later, moving farther away from the septum. At the CT, earliest activation occurred in the mid-region, consistent with activation spreading from the LA over the septum. When the earliest sites were around the His region, we elected transeptal crossing, believing that these sites simply represented left-to-right activation. Prior studies have demonstrated that for right atrial ATs with earliest activity mapped to the septal and para-Hisian regions, a proportion will actually have a left septal origin (9). The tachycardias in our series clearly originated from the MA and not from the septum; hence, they represent a different group that might also demonstrate relatively early activity in the right para-Hisian region. It should be noted that the earliest site of RA activity for high left ATs may also occur in the region of the insertion of Bachmann’s bundle. This will no doubt depend on the nature of LA to RA connections in an individual patient. Nevertheless, in the present study the Bachmann’s bundle region was later than the His region in all seven patients.

In summary, a mitral annular (or left septal) location should be considered for AT when earliest right atrial activity is recorded in the para-Hisian region (approximately 0 to 20 ms before P-wave onset) and if the P-wave morphology demonstrates the characteristic features described above.

Atrial tachycardias at the MA. Clinical ablation studies cannot provide an explanation as to the occurrence of AT from the region of the MA, and this can only be speculative. Indeed, even the mechanism of these tachycardias is unclear and showed considerable variation in the current study. A
A possible embryologic basis for tachycardias originating around the tricuspid annulus has been postulated based on the observation by McGuire et al. (10) of cells within the annulus that display “nodal-like” properties with response to adenosine and lack of connexin43 expression (10). Similarly, Wit et al. (11) have observed that the anterior leaflet of the mitral valve contained muscle fibers in direct continuity over the fibrous annulus with the left atrial myocardium (11). These muscle fibers exhibited “nodal-like” action potentials, thereby initiating automatic impulses that could propagate into the LA. However, the junctional region at the MA was a common site of conduction block; thus, potentially also providing the substrate for initiation of reentry (11). However, why these foci particularly originate in proximity to the left fibrous trigone and the mitral-aortic junction is unclear.

Study limitations. Clinical techniques for determining arrhythmia mechanism are limited, and we have therefore not attempted to do this. All programmed extrastimulation was performed from the CS or RA without induction from the LA. Positioning of the CS catheter more distally may have provided additional clues to the site of AT origin.

Conclusions. The MA is an unusual but important site of origin for focal AT. Tachycardia originating from the MA has a propensity to be localized to the superior aspect of this structure in proximity to the mitral-aortic junction. It can be suspected as a potential anatomic site of tachycardia origin from a careful analysis of P-wave morphology and the

Figure 2 Continued. (B) Graphic representation of the mean activation times at each of the recorded endocardial sites for the seven patients. Earliest endocardial activation on these standard catheters occurred at the His bundle region in all seven patients. HBE = His bundle. Numbers listed in catheter location refer to bipolar pair.
Figure 3. Left anterior oblique and right anterior oblique projections of the catheter positions are shown with the mapping catheter at the successful ablation site on the mitral annulus in Patient #3. ABL = ablation catheter across transeptal puncture; CS = coronary sinus; CT = crista terminalis; HBE = His bundle.

Figure 4. Schematic of the mitral annulus (MA) in the left anterior oblique projection showing the location of the seven tachycardia foci at the point of successful ablation. All were positioned superiorly between 11:00 and 12:00 when viewing the MA as a clockface. AV = aortic valve; CS = coronary sinus; HBE = His bundle; MV = mitral valve; TV = tricuspid valve.
atrial endocardial activation sequence map. Using mapping targeted to anatomic structures achieved a high success rate for ablation without the need for sophisticated three-dimensional mapping systems.

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REFERENCES