Coexistence of Type I Atrial Flutter and Intra-Atrial Re-Entrant Tachycardia in Patients With Surgically Corrected Congenital Heart Disease

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OBJECTIVES
This study assessed the coexistence of intra-atrial re-entrant tachycardia (IART) and isthmus-dependent atrial flutter (IDAF) in patients presenting with supraventricular tachyarrhythmias after surgical correction of congenital heart disease (CHD).

BACKGROUND
In patients with CHD, atrial tachyarrhythmias may result from IART or IDAF. The frequency with which IART and IDAF coexist is not well defined.

METHODS
Both IDAF and IART were diagnosed in 16 consecutive patients using standard criteria and entrainment mapping. Seven patients had classic atrial flutter morphology on surface electrocardiogram (ECG), whereas nine had atypical morphology.

RESULTS
A total of 24 circuits were identified. Three patients had IDAF only, five had IART only, seven had both, and one had a low right atrial wall tachycardia that could not be entrained. Twenty-two different reentry circuits were ablated. Successful ablation was accomplished in 13 of 14 (93%) IART and 9 of 10 (90%) IDAF circuits. There was one IART recurrence. The slow conduction zone involved the region of the right atriotomy scar in 12 of 14 (86%) IART circuits. No procedural complications and no further recurrences were seen after a mean follow-up of 24 months.

CONCLUSIONS
Both IDAF and IART are the most common mechanisms of atrial re-entrant tachyarrhythmias in patients with surgically corrected CHD, and they frequently coexist. The surface ECG is a poor tool for identifying patients with coexistent arrhythmias. The majority of IART circuits involve the lateral right atrium and may be successfully ablated by creating a lesion extending to the inferior vena cava. (J Am Coll Cardiol 2001;38:377–84) © 2001 by the American College of Cardiology

Intra-atrial tachyarrhythmias occur commonly after surgical correction of congenital heart disease (CHD) (1–4). The suggested mechanism is reentry around surgical scars and/or anatomic barriers. Antiarrhythmic agents have limited efficacy in the treatment of these arrhythmias, which carry increased morbidity and mortality (3,4). However, recent studies have demonstrated the feasibility of using radiofrequency (RF) ablation to produce a long-term cure (5–9).

Isthmus-dependent atrial flutter (IDAF) may occur in anatomically normal hearts as well as in surgically corrected CHD. Intra-atrial re-entrant tachyarrhythmias in patients with surgically corrected CHD have been referred to as “atrial flutter” in previous reports. In the present study, the term IDAF is confined to intra-atrial re-entrant tachycardias utilizing the tricuspid valve/inferior vena caval (IVC) isthmus. Intra-atrial tachyarrhythmias involving scars are referred to as intra-atrial re-entrant tachycardia (IART). The prevalence of IART and IDAF, and of their coexistence in patients with surgically corrected CHD, has not been methodically studied. The purpose of this study, therefore, was to assess the frequency with which IART and IDAF coexist in patients presenting with supraventricular tachyarrhythmias after surgical correction of CHD.

METHODS

Patient population. Sixteen consecutive patients were referred to the University of Virginia Medical Center for evaluation and management of recurrent tachycardia after surgical correction of underlying CHD in the period between August 1994 and July 2000. Patient details are summarized in Table 1. All patients had undergone at least one intracardiac surgical procedure for palliation or correction of CHD. All patients underwent electrophysiological (EP) testing and attempted RF ablation of the tachyarrhythmia. All patients had at least one 12-lead surface electrocardiogram (ECG) documenting their tachyarrhythmia. Typical atrial flutter (AF) was defined as negative saw-toothed P-waves in the inferior leads.

Electrophysiological assessment. Written informed consent was obtained from all patients or their legal guardians prior to the EP study. Antiarrhythmic medications were discontinued for at least five half-lives prior to EP testing, with the exception of two patients who were receiving amiodarone. Continuous 12-lead ECG monitoring was maintained during the EP study, and conscious sedation was achieved using intravenous midazolam and fentanyl. Electrode catheters were advanced into the heart through the right and left femoral veins. A 20-pole “Halo” catheter (Cordis Webster) was positioned on the tricuspid annulus, and quadripolar catheters (5-mm spacing, Cordis Webster)
were positioned at the right ventricular apex (except for patients with single ventricle) and, when possible, at the anteroseptal tricuspid annulus to record the His potential (Figs. 1 and 2). Both mapping and ablation were performed using a 4-mm tipped quadripolar steerable catheter (Blazer II, EP Technologies, San Jose, California). Surface ECG leads I, II, L and V1 and intracardiac electrograms from the electrode catheters were displayed on a multichannel oscilloscope, and the signals were stored on an optical disc (Bard Duo). Electrical stimulation was delivered through an external stimulator (Medtronic Model 532B) with a 2-ms pulse width at twice the diastolic threshold. The majority of patients were in ongoing tachycardia at the start of the studies. In other cases the tachycardia was initiated by programmed electrical stimulation and/or atrial burst pacing.

The diagnosis of typical IDAF was suggested by counterclockwise activation around the tricuspid annulus and was confirmed by entrainment mapping from the coronary sinus ostium. Atypical IDAF was defined as IDAF with clockwise activation around the tricuspid annulus. Termination of the tachycardia during isthmus ablation provided strong confirmation for the presence of IDAF.

The diagnosis of IART was made using the following criteria: 1) The atrioventricular relationship was not 1:1 during tachycardia; 2) the atrial activation sequence was not consistent with retrograde conduction either through the atrioventricular (AV) node or via an accessory pathway; 3) there was induction and/or termination of tachycardia with single atrial premature stimuli; 4) entrainment of tachycardia with either manifest or concealed fusion was possible with atrial stimulation; and 5) there was exclusion of IDAF.

Radiofrequency ablation. Ablation was performed using unmodulated RF energy at 50 MHz (EPT 1000 TC, Cardiac Ablation System). The power output was limited to 50 W, and RF energy was applied in the temperature-controlled mode (target 70°C). The IDAF was ablated using standard techniques by making a continuous line between the tricuspid annulus and the ostium of the inferior vena cava. Bi-directional isthmus conduction block was confirmed after apparently successful ablation by pacing from either side of the ablation line, and repeated 30 min later.

Zones of slow conduction were identified by low-amplitude fractionated electrograms and confirmed by entrainment mapping with a short postpacing interval. Ablation of IART was performed by extending an RF lesion to an anatomic barrier, usually the ostium of the IVC. Success was confirmed by programmed electrical stimulation. Dual extrastimuli to atrial refractoriness at a drive cycle length of 350 ms were applied immediately after apparently successful ablation, and 30 min later. Further mapping and ablation were performed if an atrial tachycardia was reinduced. Isoproterenol was not routinely used for reinduction after ablation.

### Table 1. Patient Demographics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Gender</th>
<th>Diagnosis</th>
<th>Corrective Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>25</td>
<td>F</td>
<td>Primum ASD</td>
<td>ASD repair</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>M</td>
<td>Perimembranous VSD</td>
<td>VSD repair</td>
</tr>
<tr>
<td>3</td>
<td>27</td>
<td>M</td>
<td>Perimembranous VSD/PDA</td>
<td>VSD/PDA repair</td>
</tr>
<tr>
<td>4</td>
<td>39</td>
<td>F</td>
<td>Tricuspid atresia, secundum ASD</td>
<td>Fontan procedure</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>M</td>
<td>VSD Eisenmenger’s</td>
<td>VSD repair</td>
</tr>
<tr>
<td>6</td>
<td>25</td>
<td>M</td>
<td>Mitral atresia</td>
<td>Modified Fontan procedure</td>
</tr>
<tr>
<td>7</td>
<td>34</td>
<td>M</td>
<td>ToF</td>
<td>Tetralogy repair</td>
</tr>
<tr>
<td>8</td>
<td>62</td>
<td>M</td>
<td>Secundum ASD</td>
<td>ASD repair</td>
</tr>
<tr>
<td>9</td>
<td>13</td>
<td>M</td>
<td>Double inlet left ventricle</td>
<td>Fontan procedure</td>
</tr>
<tr>
<td>10</td>
<td>39</td>
<td>F</td>
<td>ToF</td>
<td>Tetralogy repair</td>
</tr>
<tr>
<td>11</td>
<td>20</td>
<td>F</td>
<td>Atrioventricular septal defect</td>
<td>AV canal repair</td>
</tr>
<tr>
<td>12</td>
<td>20</td>
<td>M</td>
<td>ToF</td>
<td>Tetralogy repair</td>
</tr>
<tr>
<td>13</td>
<td>29</td>
<td>M</td>
<td>D-Transposition, Double inlet left ventricle</td>
<td>Fontan procedure</td>
</tr>
<tr>
<td>14</td>
<td>66</td>
<td>F</td>
<td>Secundum ASD</td>
<td>ASD repair</td>
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<tr>
<td>15</td>
<td>71</td>
<td>M</td>
<td>Secundum ASD</td>
<td>ASD repair</td>
</tr>
<tr>
<td>16</td>
<td>35</td>
<td>M</td>
<td>Secundum ASD</td>
<td>ASD repair</td>
</tr>
</tbody>
</table>

ASD = atrial septal defect; AV = atrioventricular; PDA = patent ductus arteriosus; ToF = tetralogy of Fallot; VSD = ventricular septal defect.

Abbreviations and Acronyms

- AF = atrial flutter
- ASD = atrial septal defect
- AV = atrioventricular
- CHD = congenital heart disease
- ECG = electrocardiogram
- EP = electrophysiologic
- IART = intra-atrial re-entrant tachycardia
- IDAF = isthmus-dependent atrial flutter
- IVC = inferior vena cava, inferior vena caval
- NYHA = New York Heart Association
- RF = radiofrequency
- ToF = tetralogy of Fallot
- VSD = ventricular septal defect
Patients were monitored overnight before discharge; they were followed on an outpatient basis, and recurrence of either symptoms or of atrial tachycardia was documented.

**Procedure duration and fluoroscopy times.** Procedure duration was defined as the time from catheter insertion until study termination. Pulsed fluoroscopy at a rate of seven frames per second was used, and the total fluoroscopy time was calculated for each procedure.

**Data analysis.** Data were expressed as the mean ± SD. Paired comparisons among conditions were performed with the two-tailed Student t test. Statistical significance was defined as p < 0.05.

**RESULTS**

**Patient characteristics.** Sixteen consecutive patients with surgically corrected CHD were studied (Table 1). Eleven patients were men, and the mean patient age was 32 ± 18 years (11 to 71 years). Cardiac anomalies included tetralogy of Fallot (ToF) (n = 3), atrial septal defect (ASD) (n = 5), ventricular septal defect (VSD) (n = 3), AV septal defect (n = 1) and single ventricle physiology (n = 4). Fourteen of the patients had one intracardiac surgical repair; two patients had two repairs. The functional status of the patients ranged from New York Heart Association (NYHA) functional class I to functional class IV.

**Presenting ECG.** Seven of the sixteen patients (44%) had typical AF morphology on their presenting 12-lead ECG, whereas nine patients (56%) had atypical morphology (Table 2). All patients who were subsequently found to have only IART on EP testing had atypical flutter morphology. All patients who were subsequently found to have only IDAF had typical flutter morphology on their surface ECG. Four of the seven patients (57%) who were found to have coexisting IDAF and IART had typical flutter morphology, whereas the other three patients (43%) had atypical flutter morphology (Table 2).

**Identification of IDAF and IART circuits.** An example of IDAF in a patient with ToF (Patient 12) is shown in Figure 3A. Note the counterclockwise activation sequence in the Halo catheter positioned on the tricuspid annulus, with high to low activation of the lateral right atrium. Pacing from the low right atrial septum in the region of the coronary sinus ostium resulted in entrainment with concealed fusion (i.e., the atrial activation sequence was unaffected by acceleration of the tachycardia from this site), and the postspacing interval matched the tachycardia cycle length.

Figure 4 shows an example of IART in patient 11, who had undergone repair of an AV septal defect. The AV relationship was 2:1 and the intra-atrial activation sequence recorded from the Halo catheter was not consistent with IDAF. The mapping/ablation catheter was positioned on the lateral right atrial wall, where the signal was fractionated and of low amplitude. Pacing from this site accelerated the tachycardia without affecting the activation sequence (concealed fusion), and the postspacing interval was almost identical to the tachycardia cycle length. Thus, this tachycardia was IART, dependent on slow conduction in the region of an old atriotomy scar.

In two patients, a transition to a new tachycardia morphology was observed during the application of RF energy to interrupt IDAF. An example of this is shown in Figure 3B. During ablation of the tricuspid valve/IVC isthmus, an abrupt change in tachycardia cycle length and morphology was observed. Subsequent mapping indicated that the new tachycardia was IART involving the region of the right atriotomy scar. After ablation of this arrhythmia, bidirectional isthmus conduction block was confirmed.
The mean cycle length for IART circuits was 308 ms (range 210 to 570 ms) and the mean cycle length for IDAF circuits was 291 ms (range 210 to 460 ms, p = 0.67). The zone of slow conduction was in the region of the atriotomy scar in 12 of the 14 IART circuits (86%). In one patient who had undergone the Fontan procedure (Patient 9), the zone of slow conduction was near the anastomosis of the right atrium to the pulmonary artery. In Patient 5, who had incomplete VSD repair, Eisenmenger’s physiology and significant right atrial dilation, the zone of slow conduction was located in the high posterior right atrium. In one patient with ToF repair (Patient 7), the tachycardia was located in the low right atrial free wall but could not be entrained.

Of the 24 different reentry circuits identified, 22 were ablated successfully (92%). Nine of the 10 IDAF circuits (90%) were successfully ablated. The single failure was in Patient 4, who had tricuspid atresia and had undergone the Fontan procedure as an infant. Patients with tricuspid atresia do not have a true annulus but instead have a muscular floor that often exhibits excellent conduction. The tachycardia had a counterclockwise appearance resembling IDAF and was entrained from multiple sites in the putative isthmus region. The cycle length increased from 460 ms to 490 ms with RF energy application but it did not terminate. None of the other patients with IDAF had recurrent supraventricular tachycardia after successful ablation. Fourteen IART circuits were identified in 13 patients. Thirteen of the 14 circuits (93%) were ablated successfully. The single failure was in Patient 14, who had undergone an ASD repair. The patient’s tachycardia was short-lived and terminated with overdrive pacing, which precluded adequate entrainment mapping and ablation. No complication was observed during the electrophysiologic studies or during RF ablations.

**Electrophysiologic study and RF ablation.** Twenty-four discrete re-entrant circuits were identified in the 16 patients (Table 2). This included 14 IART and 10 IDAF circuits. The mean cycle length for IART circuits was 308 ± 105 ms (range 210 to 570 ms) and the mean cycle length for IDAF circuits was 291 ± 84 ms (range 209 to 460 ms, p = 0.67). Isthmus-dependent atrial flutter alone was found in three patients (19%), and IART alone was found in six patients (37%). The two coexisted in seven patients (44%).

The zone of slow conduction was in the region of the atriotomy scar in 12 of the 14 IART circuits (86%). In one patient who had undergone the Fontan procedure (Patient 9), the zone of slow conduction was near the anastomosis of the right atrium to the pulmonary artery. In Patient 5, who had incomplete VSD repair, Eisenmenger’s physiology and significant right atrial dilation, the zone of slow conduction was located in the high posterior right atrium. In one patient with ToF repair (Patient 7), the tachycardia was located in the low right atrial free wall but could not be entrained.

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**Table 2. Arrhythmia Characteristics and Procedure Details**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Tachycardia Type</th>
<th>Cycle Length (ms)</th>
<th>ECG Morphology</th>
<th>Procedure Success</th>
<th>Procedure Time (min)</th>
<th>Fluoroscopy Time (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>IDAF</td>
<td>230</td>
<td>Typical</td>
<td>Y</td>
<td>55</td>
<td>1.6</td>
</tr>
<tr>
<td>2</td>
<td>IDAF/IART</td>
<td>220/285</td>
<td>Typical/Atypical</td>
<td>Y</td>
<td>247/219</td>
<td>NA/6.3</td>
</tr>
<tr>
<td>3</td>
<td>IDAF/IART</td>
<td>246/286</td>
<td>Atypical</td>
<td>Y</td>
<td>118</td>
<td>6.1</td>
</tr>
<tr>
<td>4</td>
<td>IDAF</td>
<td>460</td>
<td>Typical</td>
<td>N</td>
<td>275</td>
<td>19.1</td>
</tr>
<tr>
<td>5</td>
<td>IART</td>
<td>570</td>
<td>Atypical</td>
<td>Y</td>
<td>110</td>
<td>NA</td>
</tr>
<tr>
<td>6</td>
<td>IART</td>
<td>232</td>
<td>Atypical</td>
<td>Y</td>
<td>49</td>
<td>2.6</td>
</tr>
<tr>
<td>7</td>
<td>IART</td>
<td>260</td>
<td>Atypical</td>
<td>Y</td>
<td>54</td>
<td>3.2</td>
</tr>
<tr>
<td>8</td>
<td>IDAF/IART</td>
<td>352/380</td>
<td>Atypical</td>
<td>Y</td>
<td>71</td>
<td>5.7</td>
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<td>9</td>
<td>IART</td>
<td>496</td>
<td>Atypical</td>
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<td>NA/7.4</td>
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<td>10</td>
<td>IDAF/IART</td>
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<td>Typical</td>
<td>Y</td>
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<tr>
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<td>Typical</td>
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<td>61</td>
<td>3.2</td>
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<tr>
<td>13</td>
<td>IART</td>
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<td>130</td>
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<tr>
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<td>400/320</td>
<td>Atypical</td>
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<td>11.4/12.1</td>
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<td>IDAF/IART</td>
<td>260/250</td>
<td>Typical</td>
<td>Y</td>
<td>130</td>
<td>6.8</td>
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</table>

ECG = electrocardiographic; IART = intra-atrial re-entrant tachycardia; IDAF = isthmus-dependent atrial flutter; NA = not available.
Figure 3. (A) Typical isthmus-dependent atrial flutter (IDAF) in a patient with ToF (patient 12). The patient has had heart block since the surgical correction. Note the counterclockwise initial activation sequence in the Halo catheter positioned on the tricuspid annulus, with high-to-low activation of the lateral right atrial wall. Pacing performed through the distal ablation catheter (ABLATE-D) located near the ostium of the coronary sinus did not affect the activation sequence, and the postpacing interval was nearly identical to the tachycardia cycle length. HALO-1 = distal; HALO-10 = proximal. (B) Recording obtained during ablation of the isthmus between the tricuspid annulus and the inferior vena cava (IVC) in the same patient. The cycle length of the IDAF is prolonged in comparison with (A) as a result of partial ablation of the isthmus. The IDAF abruptly terminated during the RF energy application and was immediately replaced with an intra-atrial re-entrant tachycardia (IART) of different cycle length and activation sequence (arrows). This was an IART dependent on the region of the atriotomy scar in the lateral right atrium. Successful ablation of the tricuspid valve/IVC isthmus was confirmed after ablation of the IART. The surface electrocardiographic leads are I, L, F and V1.
to the transient nature of the arrhythmia (see preceding text), and this tachycardia recurred as well. A second attempt at ablation was unsuccessful. One patient expired from Eisenmenger’s syndrome after an incomplete VSD repair, although no further atrial tachyarrhythmia was documented during follow-up. The remaining patients were both arrhythmia- and symptom-free.

**DISCUSSION**

*Electrophysiology study and electrocardiography.* Patients who have undergone surgical correction of CHD are predisposed to the development of atrial tachyarrhythmias, and IART has been described as a mechanism accounting for a significant number of these arrhythmias (10). Although both IART and IDAF are well documented in patients with surgically corrected CHD, neither the relative prevalence of each arrhythmia nor their coexistence has been studied systematically. The present study shows that the most common underlying mechanisms of atrial tachycardia in an unselected series of patients with corrected CHD are IART and IDAF and that the two mechanisms frequently coexist. Coexistent IART and IDAF were found in 44% of the patients; IART alone was inducible in 37% of the patients, and IDAF alone in 19%.

The surface ECG was a poor guide for the diagnosis of the underlying rhythm. Patients with IDAF alone or IART alone had predictable typical or atypical flutter morphology, respectively. However, in patients with coexistent arrhythmias, either typical or atypical flutter morphology could be present on the ECG. Thus, although the surface ECG can suggest the etiology of the presenting tachycardia, it does not usually help in identifying patients with multiple tachycardias. Hence, the presence of both IART and IDAF should be assessed in these patients regardless of the tachycardia morphology on surface ECG.

*Radiofrequency ablation.* The success of RF ablation in terminating AF in patients with anatomic normal hearts has led to its use in the treatment of atrial tachyarrhythmias in patients with surgically corrected CHD. The target site for RF ablation in these patients depends on the placement of suture lines. Ideally, it is where the shortest line of block could be formed to eliminate the necessary circuit that maintains the tachycardia. Canine models of the Mustard procedure have shown that re-entrant circuits involve the right atrial free wall and that extension...
of the atriotomy to the tricuspid annulus eliminated the tachycardia (11). Similarly, canine models of the modified Fontan operation have demonstrated that the re-entrant circuit involves the lateral tunnel suture line (12). In clinical studies, several different zones of slow conduction have been described in patients with IART (5–7). These include areas between the atriotomy and the venae cavae and between the atriotomy and the tricuspid annulus. Sites of slow conduction after ASD repair were found between the atrial septal patch and the tricuspid annulus. Sites of slow conduction after ASD repair were found between the atrial septal patch and the tricuspid annulus. Patients who had undergone the Fontan procedure had sites near the atriopulmonary anastomosis. The present study not only confirms the above findings but also establishes that the region in the lateral right atrium is involved in the majority of IART circuits. Radiofrequency energy application between the zone of slow conduction and the IVC was effective in ablating 13 of the 14 (93%) IART circuits.

The successful site of ablation in patients with the Fontan procedure has been reported to be distinctly different from sites in patients who have had other forms of corrective surgery in that it often involves the Fontan anastomosis (5,6). In patients with the Fontan procedure, the IART circuits may be located in the lateral or anterior right atrium, and only a minority are located at the tricuspid annulus-IVC region (13,14). Results of the present study are in accord with these findings. Of the four patients who had undergone the Fontan procedure, two had circuits involving the region of the right atriotomy, and one patient had a circuit in the region of the atriopulmonary anastomosis. One patient with tricuspid atresia had a counterclockwise circuit in the region of the muscular floor replacing the annulus. The sites chosen for ablation were areas of slow conduction suggested by low-amplitude fractionated electrograms and confirmed by entrainment mapping (6). This approach is often time-consuming and exposes both patients and operators to significant amounts of radiation. Previous studies have reported mean procedure durations up to 8.8 h, with mean fluoroscopy times ranging from 52.6 to 80 min (6,9). However, these studies included patients with atrial redirection procedures who underwent hemodynamic studies. This could have significantly increased procedure durations and fluoroscopy times. The mean procedure duration was significantly lower in the present study (130 ± 73 min). The fluoroscopy duration was also significantly shorter, although comparisons are difficult because pulsed fluoroscopy is used in our laboratory. However, it seems that a mapping strategy targeting the lateral right atrium and the tricuspid annulus-IVC isthmus may identify the majority of circuits and may minimize procedure duration.

Recurrence and prevention. Recurrence of atrial tachycardia after RF ablation in this patient population has been previously described (5–9). Initial studies have demonstrated short-term success rates between 77% and 81% in ablating IART circuits (5–7), with intermediate- and long-term recurrence rates of 27% to 52% (5–7). In the present study, there was one IART recurrence, and it will be of

Figure 5. Example of intra-atrial re-entrant tachycardia (IART) from patient 6, who had undergone the modified Fontan procedure for mitral atresia. The atrial remnant was too small to accept the Halo catheter, so right atrial activation was recorded using two decapolar catheters positioned on the atrial septum (RAA) and on the lateral right atrial wall (RAP). Pacing was performed through the distal ablation catheter (ABLATE-D). Mapping revealed early, low-amplitude, fractionated potentials on the low lateral right atrial wall. Pacing from this site demonstrated concealed entrainment with a postspacing interval close to the tachycardia cycle length. The surface electrocardiographic leads are I, L, F, and V₁.
interest to follow the patients in the long term to assess them for further recurrence. There are reports that IART can be prevented by modifying the surgical technique (15). Gandhi et al. (16,17) demonstrated in a chronic canine model that lateral tunnel suture lines provided electrophysiological substrate for promotion of AF. The inducibility of AF was increased if suture lines were placed along the crista terminalis. Adoption of newer surgical techniques of avoiding injury to the terminal crest during the operation has been shown to reduce the incidence of atrial re-entrant tachycardia after the operation (12). In the present study, the majority of IART circuits appear to involve the region between the atriotomy scar and the IVC. Creation of a linear lesion at the atriotomy scar, and they were eliminated by RF lesions of IART circuits appear to involve the region of the atriotomy scar and the IVC. We postulate that extending the atriotomy incision to the IVC at the time of corrective surgery may reduce the incidence of these tachyarrhythmias.

Study limitations. One has to be careful in analyzing the results of the present study, considering the relatively small number of patients. The study was composed of patients with various forms of CHD. However, those who had undergone atrial redirection operations such as the Senning or Mustard procedure were not represented. The inclusion of such patients may have significantly increased procedure durations and fluoroscopy times, owing to the complexity of their underlying CHD.

The sites targeted for initial mapping were selected after an extensive review of each patient’s medical and surgical record. The zone of slow conduction in the majority of IART circuits involved the lateral right atrium. Even though the atriotomy scar is likely to be involved, the current catheter-based mapping techniques lack sufficient resolution to demonstrate conclusively its role in these circuits. Finally, this study presents short to intermediate-term follow-up results. Despite the high immediate success and low recurrence rates, no conclusions can be made regarding long-term efficacy and recurrence of arrhythmias.

Conclusions. Isthmus-dependent atrial flutter and IART are frequently found in patients with surgically corrected CHD who present with supraventricular tachyarrhythmias, and the mechanisms frequently coexist. The surface ECG is not useful in identifying patients with coexistent mechanisms. Radiofrequency ablation of both tachyarrhythmias is highly successful and safe and can be performed with relative ease and minimal radiation exposure. The majority of IART circuits appear to involve the region of the atriotomy scar, and they were eliminated by RF lesions placed between the zone of slow conduction in the lateral right atrium and the IVC. We postulate that scar may be important in the development of IART and that extension of the surgical atriotomy incision to the IVC at the time of corrective surgery may reduce the incidence of these tachyarrhythmias.

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