Radiofrequency Catheter Ablation of Supraventricular Arrhythmias in Patients With Congenital Heart Disease: Results and Technical Considerations

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Objectives. The aim of this study was to report the results and techniques of radiofrequency ablation for treatment of supraventricular arrhythmias in patients with congenital structural heart disease.

Background. The management of patients with congenital and other structural heart disease may be complicated by arrhythmias due to Wolff-Parkinson-White syndrome or by atrial arrhythmias after cardiac surgery. Ablation techniques using radiofrequency current are revolutionizing the management of arrhythmias, but reports have included few with structural heart disease.

Methods. Fifteen patients with significant heart disease underwent radiofrequency ablation: 11 with Wolf-Parkinson-White syndrome and 4 with intraatrial reentrant tachycardia after atrial surgery. Seven had Ebstein’s anomaly, complex in two, and the rest had other defects. Coexistence of structural defects introduced significant technical difficulties to radiofrequency ablation in patients with the Wolf-Parkinson-White syndrome and was accomplished by adaptation of current techniques. Ablation of Cardiac arrhythmias may complicate the management of patients with congenital structural heart disease. In the setting of impaired or marginal hemodynamic conditions, arrhythmias may be poorly tolerated, as may antiarrhythmic agents. The latter may impair myocardial function, worsen sinus node dysfunction or be proarrhythmic. Consequently, the use of ablation techniques may be indicated in some of these patients. Radiofrequency energy has been used for transcatheter ablation of accessory pathways (1-4), for atrioventricular (AV) node modification in AV node reentrant tachycardia (2,5) and for treating arrhythmias in children (6-11). However, there is scant information on the success of this technique in patients with congenital heart disease. We report here our experience with radiofrequency ablation for the treatment of several types of supraventricular tachycardia in children and adults with congenital cardiac disease.

Methods

Study patients. The patients included in this study were referred to our institution for management of recurrent tachycardia and were known to have congenital heart disease. Patients selected for radiofrequency ablation procedures either were known to be refractory to antiarrhythmic agents or were to undergo surgical correction or palliation of a congenital cardiac abnormality. All patients underwent an initial evaluation that included history, physical examination, electrocardiogram (ECG) and echocardiogram with color Doppler flow mapping to carefully delineate intracardiac anatomy. With intravenous sedation, an electrophysiologic study was performed for definition of arrhythmia
mechanism and for intracardiac mapping using previously described methods (7,12). In patients to be referred for surgical management of structural cardiac disease, a hemodynamic and angiographic study was also performed.

**Study protocol.** The diagnosis of accessory pathways was made by standard criteria. When the diagnosis of a reentrant atrial tachyarrhythmia was established, atrial flutter and intr atrial reentrant tachycardia were distinguished from each other as follows: Atrial flutter was diagnosed if there were clear flutter waves on the surface ECG and the atrial cycle length was ≤240 ms. Intraatrial reentrant tachycardia was diagnosed if discrete P waves were present, with an atrial cycle length >240 ms.

The protocol was approved by the Committee on Human Research, and written informed patient consent was obtained for the procedure. Patients 1, 3, 4, and 6 have been previously reported as part of an inclusive series of all patients undergoing catheter ablation at our institution (7,12).

After localization of arrhythmia substrates and anticoagulation with approximately 100 U/kg of intravenous heparin, radiofrequency lesions were created using a 7F or 6F tip-deflecting catheter with a large (4 mm) distal electrode (Mansfield Scientific, Mansfield, MA or EP Technologies). Radiofrequency current was supplied using a radiofrequency lesion generator (model RFG3B, RadioMedics Corp. or EP Technologies), which supplied up to 50 W of continuous, unmodulated current at 500 kHz delivered between the distal pole of the ablation catheter and a large surface area skin electrode. If a sudden rise in telemetered voltage and fall in current occurred indicating a rise in impedance, application of energy was immediately discontinued, the catheter removed and the distal electrode cleaned before reintroduction. Otherwise, energy was delivered for up to 100 s at each site.

**Ablation of accessory pathways.** As previously reported (7,12), accessory pathway location was carefully mapped using the ablation catheter. The site of earliest ventricular activation along the AV ring was determined during sinus rhythm, as well as the site of earliest retrograde atrial activation during supraventricular tachycardia. In some patients, mapping of the right AV ring was performed using an intracoronary mapping wire (13,14). The catheter was positioned against the AV ring on the atrial or ventricular side, and fluoroscopic evidence of concordant movement of the ablation catheter with the plane of the AV valve was seen, documenting good tissue contact. **Ophe** signals were recorded from these sites that were polymorphic and continuous, suggesting the presence of accessory pathway ("Kent") potentials. Radiofrequency lesions were made at these sites, with retesting to document lack of accessory pathway function 1 h after the last lesion.

**Slow pathway ablation for atrioventricular node reentry.** Slow pathway ablation was performed by the method described by Jackman et al. (15). Radiofrequency lesions were placed in the posterior septum close to or inside the mouth of the coronary sinus at sites with a large ventricular and a small atrial electrogram in sinus rhythm. Retesting was performed both with and without isoproterenol infusion to document lack of arrhythmia inducibility and loss of slow pathway function.

**Intraatrial reentrant tachycardia.** The diagnosis of intraatrial reentrant tachycardia was confirmed electrophysiologically by noting 1) an intraatrial activation sequence not consistent with retrograde atrial activation from the AV node; 2) induction and termination of tachycardia by single premature atrial contractions; and 3) the continuation of tachycardia despite AV block, either spontaneous or induced by the administration of adenosine intravenously. Multiple intraatrial electrode catheters were placed. Initial mapping involved localization of earliest atrial activation during tachycardia in relation to the onset of the P wave on the surface ECG. Further localization was accomplished by pacing from candidate sites during sinus rhythm at the tachycardia cycle length in an attempt to reproduce both the P wave axis and configuration on the 12-lead ECG, as well as the intraatrial activation sequence recorded from intraatrial catheters that was seen during tachycardia. During tachycardia, areas of atrial electrogram fractionation were sought near the regions of early activation. A fractionated electrogram was defined as an electrogram that was qualitatively more polyphasic than other electrograms obtained locally (16). Short test lesions were then administered to these sites of fractionation during tachycardia, and lesions that resulted in immediate tachycardia termination were continued for 100 s. Aggressive testing for inducibility was then performed and was repeated 1 h after the last lesion. This testing included the administration of isoproterenol.

**Follow-up study.** Echocardiograms were obtained on the day after the ablation procedure. Ambulatory 24-h ECGs were obtained on the day after the procedure and again at approximately 1 month after the procedure. Follow-up electrophysiologic studies were not routinely recommended in asymptomatic patients, but all patients were followed with periodic standard and 24-h ECGs. For success rates, 95% confidence intervals were calculated by the second method of Armitage (17).

**Results**

Fifteen patients with congenital heart disease underwent radiofrequency catheter ablation. They will be considered in two groups: those with a congenital substrate for arrhythmia (Group 1) and those with an acquired substrate (Group 2). All 11 patients in Group 1 had the Wolff-Parkinson-White syndrome, and 2 of these also had dual AV node pathways. All four patients in Group 2 had previously undergone cardiac surgery and had intraatrial reentrant tachycardia, and one of these also had AV node reentry.

The median age at time of ablation was 15 years (range 15 months to 61 years). One ablation procedure was performed in 11 patients, and two sessions were necessary in 4. The
Figure 1. Patient 3. A, Two-dimensional echocardiogram, apical four-chamber view, showing severe inferior displacement of the tricuspid annulus, right ventricular hypoplasia and a left ventricular diverticulum. AV = atrioventricular; LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle. B, Fluoroscopic studies in Patient 3. Left panel, Right coronary angiogram, slight left anterior oblique projection. Right panel, Right anterior oblique projection. The right coronary artery is cannulated with a custom mapping wire introduced through a right coronary angiographic catheter and the ablation catheter is positioned toward the tip of the mapping catheter.

average total duration of fluoroscopy was 81.8 min (range 33.2 to 144 [unavailable in two patients]) and the average duration of the procedures was 219 min (range 126 to 325) after introduction of the catheters. Ablation required a median of 22 applications of radiofrequency energy (range 4 to 30). Overall, 10 of 15 patients had complete elimination of tachycardia (67%, 95% confidence interval [CI], 42% to 85%), and 14 of 15 had clinical improvement (93%, 95% CI 70% to 99%) (17). No major or minor complications occurred as a result of either the electrophysiologic study or the ablation procedure.

Group 1

Patients. Eleven patients with the Wolff-Parkinson-White syndrome and significant congenital heart disease had ablation of accessory pathways. Moderate Ebstein's anomaly was present in four patients, severe Ebstein's anomaly with associated problems in three (Fig. 1) and other diagnoses were present in four (Table 1). Even brief tachycardia was very poorly tolerated hemodynamically in Patients 2, 3, 4 and 11 because of the coexisting structural heart disease. In three patients ablation was performed as preparation for surgical correction or palliation of serious congenital heart disease (Patients 1 to 3). In each case the planned surgical procedure involved cardiopulmonary bypass, and it was believed that the additional time for intraoperative accessory pathway ablation by the endocardial approach would significantly prolong the time on cardiopulmonary bypass, thereby increasing the risks of the procedure.

Results. Fifteen accessory pathways were found in 11 patients, and 13 of 15 pathways were successfully ablated in 15 ablation sessions (2 sessions in each of 4 patients). In Patient 9, who had Ebstein's anomaly, a pathway with a short anterograde effective refractory period was successfully ablated, but a second pathway with a longer refractory period could not be ablated at the same session.

Accessory pathway conduction returned after initially successful ablation in four patients. A second ablation procedure was successful in two patients and not attempted in one. One of the latter, Patient 11 with Shone's complex, had recurrence of pre-excitation without tachycardia 48 h after the initial procedure, and supraventricular tachycardia was not inducible at repeat electrophysiologic study by the esophageal route. Patient 2, the other patient with recurrence of pre-excitation, is described in the following section.

Patients 1 and 2 subsequently underwent uncomplicated reparative or palliative procedures with a cardiopulmonary bypass duration of 18 min in both cases. Patient 3 has been accepted for a Fontan procedure but has not yet undergone surgery. Neither patient experienced intraoperative or immediate postoperative supraventricular tachycardia. Patient 2 had a recurrence of pre-excitation 3 days postoperatively with a single short episode of supraventricular tachycardia. She is currently receiving propranolol and has no tachycardia. Her condition is considered improved because the arrhythmia was not controlled with this or any other medication before the ablation procedure.

Patient 4 had cardiomyopathy with severe progressive left ventricular dysfunction unrelated to the Wolff-Parkinson-White syndrome or tachycardia. The patient had a recurrence of pre-excitation the evening after the first procedure and the following day had a second procedure that was successful. He had no episodes of tachycardia after ablation and underwent heart transplantation 1 month later. Examination of the heart revealed a discrete, well demarcated radiofrequency lesion at the tricuspid annulus.

Technical considerations. In five of the seven patients with Ebstein's anomaly, mapping of retrograde atrial activation during supraventricular tachycardia and anterograde ventricular activation during sinus rhythm was performed using a mapping wire inserted into the right coronary artery (custom device, Viggo-Spectramed). Right coronary artery mapping allowed precise localization of the accessory pathways and more rapid evaluation in the presence of multiple
Table 1. Radiofrequency Ablation of Arrhythmia With Congenital Substrate

<table>
<thead>
<tr>
<th>Pt No</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Cardiac Anomalies</th>
<th>Accessory Pathway Locations</th>
<th>Initial Success</th>
<th>Fluoro Time (min)</th>
<th>Follow-Up (mo)</th>
<th>Clinical Status at Follow-Up</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13/3</td>
<td>M</td>
<td>Ebstein's anomaly, ASD</td>
<td>Lt PostLat</td>
<td>Yes</td>
<td>90.2</td>
<td>31</td>
<td>No recurrence</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>2/F</td>
<td>F</td>
<td>Pulmonary atresia, hypoplastic RV, B-T shunt</td>
<td>PostSept; Rt PostLat</td>
<td>Yes; Yes</td>
<td>55.7</td>
<td>24</td>
<td>Recurrence, improved</td>
<td>Had B-T shunt and tricuspid valve closure</td>
</tr>
<tr>
<td>3</td>
<td>26/M</td>
<td>M</td>
<td>Ebstein's anomaly, hypoplastic RV, Potts anastomosis</td>
<td>Rt Lat</td>
<td>Yes</td>
<td>40.0</td>
<td>22</td>
<td>No recurrence</td>
<td>To undergo Fontan procedure</td>
</tr>
<tr>
<td>4</td>
<td>61/M</td>
<td>M</td>
<td>Ebstein's anomaly, severe LV dysplasia</td>
<td>Rt Lat</td>
<td>Yes</td>
<td>N/A</td>
<td>1</td>
<td>No recurrence</td>
<td>2 sessions. Had heart transplantation 1 mo later</td>
</tr>
<tr>
<td>5</td>
<td>16/M</td>
<td>M</td>
<td>Ebstein's anomaly</td>
<td>Rt Ant; Lt Lat; Rt PostLat; Lt Lat</td>
<td>Yes; Yes; Yes; Yes</td>
<td>60.4</td>
<td>23</td>
<td>No recurrence</td>
<td>2 sessions</td>
</tr>
<tr>
<td>6</td>
<td>28/M</td>
<td>M</td>
<td>Small to moderate VSD</td>
<td></td>
<td></td>
<td>N/A</td>
<td>21</td>
<td>No recurrence</td>
<td>Moderate LV enlargement due to VSD</td>
</tr>
<tr>
<td>7</td>
<td>13/F</td>
<td>F</td>
<td>L-transposition, VSD</td>
<td>Lt Post (with AVNRT)</td>
<td>Yes</td>
<td>131.5</td>
<td>19</td>
<td>No recurrence</td>
<td>AVNRT not approached</td>
</tr>
<tr>
<td>8</td>
<td>8/F</td>
<td>F</td>
<td>Ebstein's anomaly</td>
<td>Rt PostLat</td>
<td>Yes</td>
<td>68.5</td>
<td>13</td>
<td>No recurrence</td>
<td>2 sessions</td>
</tr>
<tr>
<td>9</td>
<td>15/M</td>
<td>M</td>
<td>Ebstein's anomaly, ASD</td>
<td>Rt Lat</td>
<td>Yes</td>
<td>144</td>
<td>13</td>
<td>No recurrence</td>
<td>Short ERP pathway ablated</td>
</tr>
<tr>
<td>10</td>
<td>32/M</td>
<td>M</td>
<td>Severe Ebstein's anomaly, patent foramen ovale, right to left shunt</td>
<td>Rt Post; Rt Post</td>
<td>No; No</td>
<td>124</td>
<td>10</td>
<td>Not improved</td>
<td>Now controlled on medication</td>
</tr>
<tr>
<td>11</td>
<td>1/M</td>
<td>M</td>
<td>Parachute mitral valve, supravalvar mitral ring, s/p correction and s/p anomalous Lt coronary reimplantation</td>
<td>Lt Post nonseptal (with dual AV node pathways)</td>
<td>Yes</td>
<td>95.3</td>
<td>5</td>
<td>Recurrence, improved</td>
<td>Slow pathway ablation also performed. Pre-excitation without SVT recurred at 48 h. SVT not inducible at restudy</td>
</tr>
</tbody>
</table>

Ant = anterior; ASD = atrial septal defect; AV = atrioventricular; AVNRT = atrioventricular node reentrant tachycardia; B-T = Blalock-Taussig; ERP = effective refractory period; F = female; Fluoro = fluoroscopic; Lat = lateral; Lt = left; LV = left ventricular; M = male; N/A = not available; Post = posterior; PostLat = posterolateral; PostSept = posteroseptal; Rt = right; RV = right ventricular; s/p = status post; SVT = supraventricular tachycardia; VSD = ventricular septal defect.

pathways, as well as the plane of the AV groove in these patients with Ebstein's anomaly. This was particularly important in Patient 3, who had severe right ventricular hypoplasia (Fig. 1). Patient 4 had undergone a diagnostic electrophysiologic study several years earlier and was thought to have two free wall pathways, anterior and posterior, when mapping was performed by conventional means using a catheter rotated through 360°. However, right coronary artery mapping at the time of the ablation demonstrated a single right lateral pathway. In each patient with Ebstein's anomaly, the pathways were approached with an ablation catheter passed from the inferior vena cava. Because of the inferior displacement of the tricuspid valve leaflets into the ventricle, positioning of the catheter tip under the valve leaflet would not have been useful. Therefore, the ablation catheter was positioned against the AV ring either directly or after flexion of 180°. Particular attention was paid to obtaining a catheter position with equal atrial and ventricular electrogram amplitudes to avoid administering energy in the atrialized right ventricle.

The congenital heart disease in Patients 2, 3, 7 and 11 added significant technical difficulties to the ablation procedure. Patient 7 had ventricular inversion with l-transposition of the great arteries; therefore the ablation of a left posterior para-septal pathway required positioning of the ablation catheter on or under a left-sided tricuspid valve. The ventricular septum was in a sagittal rather than an oblique plane, thereby complicating catheter manipulation. Initially a patent foramen ovale was crossed, but ablation attempts on the atrial side failed to eliminate pre-excitation. Subsequently, the pathway was successfully ablated by the retrograde route. Patient 11, who had Shone's complex with aortic stenosis, a stenotic small parachute mitral valve and a stenosing supravalvar mitral ring, had previously undergone...
repair of aortic coarctation and reimplantation of an anomalous left coronary artery that arose from the pulmonary artery. These anatomic factors necessitated a transseptal rather than a retrograde approach to a left posterior paraseptal pathway. The patient also had dual AV node pathways, and slow pathway modification was also successfully performed.

Group 2

Patients. Four patients underwent catheter ablation of intraatrial reentrant tachycardias that were considered sequelae of atrial surgery (Table 2). Two patients had undergone patch closure of secundum atrial septal defects, one had undergone a modified Fontan procedure, and one with single ventricle and D-transposition had undergone a palliative Mustard procedure to decrease cyanosis. The atrial cycle lengths during intraatrial reentrant tachycardia ranged from 310 to 430 ms. In Patient 13, who had had the Fontan operation, tachycardia was associated with the rapid development of marked hepatomegaly. In Patients 12 to 14, atrial flutter was inducible only by aggressive atrial stimulation and was not present clinically.

Results. All four patients had initially successful radiofrequency ablation, with noninducibility of the previously easily inducible atrial tachycardia. Patient 12 had a single recurrence of atrial tachycardia at a slightly longer atrial cycle length and with a different P wave axis and morphology, suggesting a second circuit. This has not recurred. Patient 14 had a recurrence of atrial tachycardia with a similar P wave morphology and axis 3 months after the procedure, but at a markedly longer cycle length (415 ms vs. 330 ms before ablation) and has been completely asymptomatic without hepatomegaly despite continued tachycardia.

Technical considerations. In the two patients who had had atrial septal defect repair, positioning of diagnostic catheters was routine, and left-sided signals were obtained from the coronary sinus. In the patient with the Fontan procedure (Patient 14), left atrial recordings were not obtained because no atrial communication existed and the coronary sinus had been left to drain into the left atrium. In the patient with a Mustard procedure and single ventricle, the coronary sinus was entered directly from the inferior vena cava. The pulmonary venous atrium was reached by passing a catheter from the inferior vena cava through the baffle into the left ventricle and subsequently backward across the other AV valve into the pulmonary venous atrium.

In Patients 12 and 15, a site of early atrial activation during tachycardia was found at the lateral right atrium at the probable site of atriotomy, with fractionation of the local electrograms. Application of radiofrequency energy at this site during tachycardia caused termination of the tachycardia, and it could not be reinduced. In addition, Patient 15 had a second discrete substrate for intraatrial reentrant tachycardia with early atrial activation and fractionation in the region of the atrial septal defect patch, which was similarly ablated, as well as AV node reentry, which was eliminated by ablation of the slow pathway.

In Patient 13, intraatrial reentrant tachycardia could be induced only from the pulmonary venous atrium, but very early atrial electrograms with fractionation were found at the posterior right AV ring just beneath the mouth of the coronary sinus. Application of radiofrequency energy at this site during tachycardia terminated the rhythm, and it could not be reinduced.

In Patient 14, early atrial activation during intraatrial reentrant tachycardia was found close to the right AV groove anterolaterally, with fractionated electrograms found at the isthmus formed between the hypoplastic tricuspid annulus and the origin of the conduit, which connected the right atrium to the pulmonary artery (Fig. 2). Application of radiofrequency energy at this site caused termination of tachycardia, and it could not be reinduced.

Discussion

Congenital and acquired arrhythmia substrates. The association of supraventricular arrhythmias and congenital heart disease may have either a congenital substrate, such as W: ni-Parkinson-White syndrome, or a surgically acquired...
Implantation of permanent pacemakers in these patients (27).

A common problem in patients after extensive atrial surgery, mic agents are known to exacerbate sinus node dysfunction, likely to be higher in this group. Most available antiarrhythmic postoperative patients, the risks of antiarrhythmic agents are

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Substrate, particularly after atrial surgery. The congenital substrate for arrhythmia is not rare. Of patients with Wolff-Parkinson-White syndrome followed up from birth, 20% to 37% have associated congenital heart disease (18-21). Conversely, of patients with Ebstein’s anomaly of the tricuspid valve, as many as 26% will have accessory pathways, and these are often multiple (22-24). Acquired substrates for arrhythmias may be the result of atrial dilation, atrial suturing or scar, or both, or injury to the conduction system. Consequently, patients undergoing cardiac surgery are at risk of developing new cardiac arrhythmias, and these may cause significant morbidity and mortality. The latter is of particular concern in patients who have undergone a Mustard or Senning procedure for D-transposition and those with the Fontan procedure (or its modifications) for various forms of functional single ventricle. These patients are known to be at risk of sudden cardiac death, and it has been suggested that the mechanism is rapid conduction of atrial arrhythmias to the ventricle (25,26).

Because of associated hemodynamic abnormality, patients with structural heart disease may tolerate tachycardias more poorly than patients with a structurally normal heart. In view of this and the often abnormal conduction system of postoperative patients, the risks of antiarrhythmic agents are likely to be higher in this group. Most available antiarrhythmic agents are known to exacerbate sinus node dysfunction, a common problem in patients after extensive atrial surgery, and the possibility of such exacerbation often requires implantation of permanent pacemakers in these patients (27).

Agents such as quinidine and flecainide have an increased proarrhythmic potential in patients with ventricular dysfunction (28-30) and should be avoided. Finally, most antiarrhythmic agents cause or exacerbate systemic ventricular dysfunction (31), a problem of particular concern in patients after the Fontan procedure. For all these reasons, radiofrequency ablation deserves careful consideration in the management of arrhythmias in patients with congenital heart disease.

Technical considerations. The use of transcatheter radiofrequency techniques in this patient group requires a thorough knowledge of such techniques in patients with a structurally normal heart, a knowledge of congenital heart disease and experience with the catheterization of patients with congenital and other structural heart defects, and the ability to improvise and adapt techniques for the particular situation encountered. Coexisting defects may limit the options for catheter approach, as in Patient 11 with conotruncal and mitral stenosis, and therefore may require the use of transseptal puncture in small patients. Such defects may also direct the placement of the ablation catheter, as in Patient 14 with intraatrial reentrant tachycardia involving the Fontan conduit. There is a clear advantage to performing a diagnostic cardiac catheterization at the time of electrophysiologic study with catheter ablation in preparation for cardiac surgery, because it minimizes the frequency of vascular entry in these patients.

Despite the difficulties inherent in performing ablation procedures in patients with serious structural heart disease, 10 of the 15 patients reported here had complete elimination of tachycardia and 14 of 15 had clinical improvement. Although not as completely successful as our experience in children without structural heart disease (7), these early results indicate that radiofrequency ablation is a highly effective alternative to surgery or lifelong antiarrhythmic medication, or both, in this patient group.

Risks of ablation therapy in children. No major or minor complications were evident either initially or at follow-up in any of the patients in this report. Studies from individual centers and one multicenter study (7,8,10,32,33) have shown that the short-term risk of complications is quite low. The long-term risks have yet to be determined. These risks could conceivably include arrhythmias or coronary artery problems. Of some concern also is the exposure to ionizing radiation that accompanies an ablation procedure. The amount of exposure to the patient is similar to that received during other interventional or even diagnostic catheterizations. One report (34) estimated the lifetime risk of a fatal malignancy from a typical catheter ablation procedure in an adult as 0.07%. This risk may be higher in children because of longer life expectancy, and efforts to reduce the radiation exposure associated with radiofrequency ablation are needed.

Ablation as preparation for heart surgery. Patients with abnormal tachycardia who are to be referred for surgical correction or palliation of a structural heart defect represent
a special problem. In the past, several approaches have been available:

1) Patients may undergo surgical division of an accessory pathway at the time of the other operation (3). If this procedure is performed by the endocardial approach, it adds significantly to the duration of cardiopulmonary bypass and perhaps might increase the risk of the operation as a whole (35,36). A major perioperative neurologic complication has been reported in at least one such patient (3). Either ablation by the epicardial approach (37) or radiofrequency ablation before surgery would seem a lower risk approach; the latter is certainly more convenient because it frees the surgeon from the need to ablate the pathway as part of an already complex procedure.

2) The patient might undergo surgical correction without an attempt at accessory pathway division. Theoretically, postoperative tachycardia can be converted repeatedly by programmed stimulation using epicardial pacing wires. However, endogenous or exogenous catecholamines may bring about nearly incessant tachycardia, which may be poorly tolerated in the immediate postoperative period. We (38) have observed two such patients before the advent of radiofrequency techniques, both of whom did quite poorly postoperatively, and others (21) have reported similar experiences. The advantage of preoperative radiofrequency ablation is that the potential for abnormal tachycardia due to the accessory pathway is eliminated before surgery, simplifying the operative procedure and lowering the risk.

Atrial tachycardia. Ectopic (automatic focus) atrial tachycardia has been successfully approached by transcatheter techniques (32,39), and there is increasing experience with the ablation of intraatrial reentrant tachycardia by these means. Some investigators have had encouraging results in adults with atrial flutter and an otherwise normal heart, but there has been a significant recurrence rate of tachycardia (39). In general, reentrant rhythms require an area of slow conduction, as well as an area of unidirectional block (which may be an anatomic or functional barrier), for their initiation and maintenance. Some patients who have undergone atrial surgery could conceivably have a surgically created substrate for intraatrial reentrant tachycardia because suture lines may create barriers to transmission of cardiac conduction (40), protecting a zone of myocardium that then becomes available for participation in a reentrant circuit. In Patient 14, for example, the conduit probably acted as a barrier, protecting an isthmus of tissue between the conduit and the tricuspid valve ring that, in turn, acted as the substrate for intraatrial reentrant tachycardia. Future work in this area should focus on a careful study of the possible barriers to conduction created by different forms of cardiac surgery, as well as application of these techniques to patients with atrial flutter.

Conclusions. We have found transcatheter techniques using radiofrequency energy to be effective in the definitive treatment of patients with structural heart disease and tachycardia due either to the Wolff-Parkinson-White syndrome or to intraatrial reentry. The use of these techniques in this patient group requires a thorough knowledge of cardiac catheterization techniques in patients with congenital and other structural heart disease. In patients who are to undergo surgical correction or palliation, preoperative ablation of the substrate for tachycardia is effective and may be preferred to operative accessory pathway division. The ablation of intraatrial reentrant tachycardia shows promise in the management of patients who have undergone extensive atrial surgery; it may eventually become the preferred approach, particularly when there are contraindications to the use of antiarrhythmic agents.

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References