Transcatheter Ablation of Ectopic Atrial Tachycardia in Young Patients Using Radiofrequency Current

Edward P. Walsh, MD; J. Philip Saul, MD; J. Edward Hulse, MD; Larry A. Rhodes, MD; Allan J. Hordof, MD; John E. Mayer, MD; and James E. Lock, MD

**Background.** Ectopic atrial tachycardia (EAT) is a reversible cause of cardiomyopathy but may be quite difficult to control with conventional therapy. Transcatheter ablation with radiofrequency current was tested as an alternative to medical or surgical treatment of this condition.

**Methods and Results.** Twelve young patients (aged 10 months to 19 years) with drug-resistant EAT were treated with direct transcatheter ablation of the ectopic focus using radiofrequency (RF) energy. All had depressed left ventricular contractility by echocardiographic criteria, involving shortening fractions of 10–26% (median, 20%; normal, 28–35%). The EAT was mapped to the left atrium in seven cases and to the right atrium in five. Local atrial activation at the ectopic site preceded the onset of the surface P wave by 20–60 msec (median, 42 msec). Tachycardia terminated 0.5–13.0 seconds (median, 2.0 seconds) into a successful RF application. The ablation effectively eliminated EAT in 11 of 12 patients (92%), all of whom were discharged in sinus rhythm without medications after a median hospital stay of 48 hours. Ablation was unsuccessful in one patient with diffuse dysplasia of the anterior right atrium, who eventually did well after surgical resection of abnormal atrial tissue. Transient depression of sinus node function was noted in one patient who had successful ablation of an EAT focus in close proximity to the sinus node, although normal sinus node function returned within 72 hours. No other complications were encountered. During follow-up (3–21 months; median, 13 months), one patient had recurrence of a slower and less sustained EAT that was successfully eliminated at a second ablation session. All others remained in sinus rhythm, and all 12 subjects recovered normal ventricular function.

**Conclusions.** RF ablation appears to be a safe and effective therapeutic option for drug-resistant ectopic atrial tachycardia and may be the preferred first-line therapy for those patients with depressed ventricular function. *(Circulation 1992;86:1138–1146)*

**Key Words** • catheters • cardiomyopathy • supraventricular tachycardia

Ectopic atrial tachycardia (EAT) is an uncommon form of chronic supraventricular tachycardia that can ultimately lead to cardiomyopathy if unrecognized or uncontrolled. While it is seen most frequently in the pediatric age group, cases have been documented in the adult population as well. The cellular genesis of this arrhythmia is not completely understood, although the gross clinical behavior seems most consistent with a mechanism of abnormal automaticity arising from a single nonsinus atrial focus. EAT may resolve spontaneously in a small number of patients; however, the majority will require therapy, particularly if ventricular function is compromised. Unfortunately, treatment for this condition has been notoriously difficult. Chronic pharmacological suppression, when effective, often necessitates the use of class Ic or class III agents with the attendant risk of drug side effects. Surgical excision of EAT has also been used, with generally good results for left atrial foci but inconsistent success with right atrial sites.

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Transcatheter ablation techniques have been explored as alternative therapy for EAT in selected patients, although the experience has been limited to date. While some initial procedures involving direct current (DC) fulguration were promising, the technique did not gain wide acceptance due to the risks of acute myocardial damage inherent to DC methodology. With the advent of radiofrequency (RF) current as an energy source for cardiac ablation, interest in transcatheter therapy for EAT has resurfaced, heralded by recent attempts in both adult and pediatric patients. This report details our clinical experience and technique for RF ablation of ectopic atrial tachycardia in the pediatric age group.

**Methods**

**Patients**

The diagnosis of EAT was initially suspected on the basis of characteristic findings on ECG and Holter
TABLE 1. Profiles of Patients With Ectopic Atrial Tachycardia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Symptoms</th>
<th>Prior drug trials</th>
<th>EAT pattern</th>
<th>EAT rate (bpm)</th>
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<tr>
<td>1</td>
<td>18</td>
<td>Moderate</td>
<td>5</td>
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<td>100–170</td>
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<td>2</td>
<td>14</td>
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<td>10</td>
<td>Incessant</td>
<td>120–200</td>
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<tr>
<td>3</td>
<td>9</td>
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<td>1</td>
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<td>90–180</td>
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<td>8</td>
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<td>6</td>
<td>Episodic</td>
<td>100–280</td>
</tr>
<tr>
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<td>0.8</td>
<td>Severe</td>
<td>3</td>
<td>Incessant</td>
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<td>2</td>
<td>Incessant</td>
<td>120–170</td>
</tr>
<tr>
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<td>2</td>
<td>Incessant</td>
<td>140–220</td>
</tr>
<tr>
<td>8</td>
<td>11</td>
<td>Moderate</td>
<td>2</td>
<td>Episodic</td>
<td>160–180</td>
</tr>
<tr>
<td>9</td>
<td>13</td>
<td>Severe</td>
<td>2</td>
<td>Incessant</td>
<td>120–190</td>
</tr>
<tr>
<td>10</td>
<td>17</td>
<td>Severe</td>
<td>6</td>
<td>Episodic</td>
<td>100–180</td>
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<tr>
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<td>14</td>
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<td>4</td>
<td>Incessant</td>
<td>100–230</td>
</tr>
<tr>
<td>12</td>
<td>19</td>
<td>None</td>
<td>5</td>
<td>Episodic</td>
<td>160–210</td>
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</tbody>
</table>

EAT, ectopic atrial tachycardia; bpm, beats per minute.

recordings, including 1) prolonged episodes of rapid atrial rhythm at inappropriate rates for age, 2) wide variation in atrial rate (largely proportional to autonomic state) exhibiting “warm-up” at initiation and “cool-down” at termination, 3) abnormal axis and/or morphology for the P wave, and 4) episodic Mobitz I atrioventricular block without interruption of the primary atrial tachyarrhythmia. Historical features were also considered, such as 1) prior failed external cardioversion and 2) atypical response to conventional drug therapy. If the diagnosis remained uncertain, intracardiac electrophysiological study was used to demonstrate the generally accepted criteria for EAT, including 1) inability to initiate or terminate the atrial tachyarrhythmia with programmed stimulation techniques, 2) reset behavior in response to premature beats or prolonged rapid pacing similar to the sinus node, 3) atrial activation sequence supporting a nonsinus origin for initial depolarization, and 4) elimination of accessory atrioventricular pathways or atrioventricular nodal reentry as diagnostic possibilities.

Patients were considered eligible for attempted RF ablation if they had failed one or more trials of pharmacological control and had EAT that was either incessant or accounted for >50% of the cardiac rhythm over a 24-hour period. The procedure was performed under a protocol approved in January 1990 by the Clinical Investigation Committee at Children’s Hospital, Boston. Written informed consent was obtained from parents and all participants over age 12 years and included discussion of alternate therapy with additional drug trials or surgical excision.

Twelve patients who fulfilled criteria were studied between June 1990 and January 1992 (Table 1). Six of the 12 had previously undergone diagnostic catheterization with preliminary electrophysiological testing. Patients ranged in age from 10 months to 19 years (median, 12 years) with weights between 10 and 66 kg (median, 38 kg). The diagnosis of EAT had been recognized for periods of 2 weeks to 8 years (median, 4 years), during which time all subjects had failed trials of pharmacological control using one to 10 agents (median, 3.5). The EAT was incessant in eight patients. Tachycardia was intermittent in the remainder but present throughout 50–90% of the day. Ectopic focus rates ranged from a minimum of 90 min⁻¹ to a maximum of 280 min⁻¹, with an average rate of 167 min⁻¹.

Congenital anatomic cardiac defects were not detected in any patient on the preprocedure echocardiogram (n=12) nor at a prior diagnostic cardiac catheterization (n=6). Left ventricular function (Table 2) was evaluated with echocardiography (n=12), radioisotope ejection fraction (n=1), or angiographic ejection fraction (n=1). In addition, measurements of pulmonary capillary wedge pressure (n=12) and cardiac index (n=8) were obtained at prior diagnostic catheterization and/or at time of ablation. All patients had depressed left ventricular shortening fractions on echocardiography, ranging from 10% to 26% (median, 20%; normal, 28–35%). Four patients had a dilated left ventricle, with an end-diastolic dimension exceeding the 97th percentile when corrected for body surface area. Pulmonary capillary wedge pressure was elevated above 10 mm Hg in seven subjects.

Three patients denied any symptoms, whereas six had moderate symptomatology (e.g., episodic dizziness, palpitations, exercise intolerance). Three patients had severe symptoms related to ventricular dysfunction, including a 10-month-old infant (patient 5) with low cardiac output and mitral regurgitation from left ventricular dilation and one teenager (patient 9) initially considered for cardiac transplantation before EAT was recognized.

Technique

The procedure was performed on-site at Boston Children’s Hospital for 11 of 12 patients. In one case (patient 9), the ablation was done in the laboratory of the referring institution (Columbia University Babies Hospital, New York). Before study, patients underwent a baseline cardiac evaluation consisting of physical examination and history review, ECG, 24-hour Holter, echocardiogram with Doppler, and chest radiograph. Antiarrhythmic drugs were discontinued for five half-lives in all except one patient who was receiving amiodarone until 1 week before the ablation attempt.

Sedation involved intravenous fentanyl and midazolam in the five oldest patients; however, all younger subjects were administered general anesthesia with intubation and mechanical ventilation. Two or more ves-
sels were cannulated percutaneously, after which patients were anticoagulated using 100 IU/kg intravenous heparin (maximum, 5,000 IU) with repeat doses guided by periodic measurement of activated clotting time (aiming for values approximately >50% of control). From two to five electrode catheters were then inserted, including at least one 6F or 7F deflectable-tip catheter with 2-mm spacing on the distal electrode pair and a 4-mm large-tip electrode (Mansfield/Webster, Watertown, Mass.) for detailed mapping and ablation. The simple two-catheter study used for the smallest patient involved a His-bundle recording and a single mapping/ablation catheter that was moved throughout both atria. For older patients, catheters were typically positioned at the His-bundle area, right ventricular apex, right atrium, left atrium, and, occasionally, coronary sinus. Intracardiac signals were recorded and filtered between 40 and 400 Hz for display on a 16-channel system (Bloom Associates, Reading, Pa.) along with four-surface ECG leads.

In 10 of 12 patients, EAT was present spontaneously upon arrival in the laboratory. Low-dose isoproterenol (0.01 μg/kg/min) was administered to two of these 10 in whom the EAT was only intermittent, which simplified mapping by converting the rhythm to incessant EAT. Two other patients (patients 4 and 10) were in sinus rhythm at the start of the procedure. Standard atrial extrastimulus testing (up to S3 and burst pacing) failed to induce tachycardia in these two subjects, although sporadic episodes of spontaneous EAT were eventually seen after beginning an infusion of isoproterenol.

With the patient in EAT, limited atrial stimulation involving single premature extrastimuli and burst pacing was performed to examine the gross electrophysiological behavior of the tachycardia. In no instance was EAT terminated with this stimulation. The reset response of tachycardia to these maneuvers was in agreement with the pattern previously described for EAT.5,16

Mapping of the ectopic focus was then undertaken by examining right and left atrial activation sequence. The left atrium was mapped directly in 11 of 12 patients (via a patent foramen ovale in four and via transseptal puncture using the Brockenbrough technique17 in seven). For one patient (patient 9) in whom the EAT focus had been clearly mapped to the right atrial appendage at a prior electrophysiology study, a coronary sinus wire was used to reflect left atrial activation time, and direct entry to the left atrium was not attempted. Local atrial activation times recorded from the distal electrode pair of the mapping/ablation catheter were indexed against the onset of the surface P wave to generate the map. If P wave onset was indistinct or obscured by T wave activity, a stable intracardiac signal (e.g., low septal right atrium) was initially used as the reference. Catheter manipulation in the right atrium was not difficult; however, left atrial mapping was often facilitated by the use of long curved vascular sheaths (USCI, Tewksbury, Mass. or Cordis, Miami, Fla.), even in patients with a patent foramen. These sheaths ensured left atrial access during catheter changes and improved torque transmission and overall catheter steering ability. Catheter positions were examined with biplane fluoroscopy, generally using the anteroposterior/lateral projections for left-sided foci and right and left anterior oblique projections for right-sided foci. During eight of the procedures, atrial angiograms were performed to clarify anatomic landmarks.

A general target area for possible ablation was identified when the local atrial activation preceded the onset of the surface P wave by ≥20 msec. Precision mapping was then performed in that region to localize the site of earliest atrial activity, at which point ablation was attempted. Radiofrequency electrical current (500 kHz) was generated from an electrosurgical device (Model RFG-3C, Radioinc Inc., Burlington, Mass.) and was delivered in a unipolar fashion from the distal electrode of the mapping/ablation catheter to an adhesive patch electrode positioned on the patient’s leg. During each RF application, generator output was monitored for voltage (V), power (W), current (mA), impedance (Ω), and duration (seconds). Initial generator output was set for 20–25 W, and RF application was commenced with constant rhythm monitoring. If the tachycardia was not affected within 10 seconds, RF discharge was terminated, and the catheter was repositioned for a repeat attempt. If EAT terminated or changed rate dramati-
cally during the 10-second “test application,” RF discharge was continued at the target area for 40–60 seconds, with the power increased slightly to 25–30 W.

After a successful lesion, patients were observed in the laboratory for 30 minutes, after which they were challenged with an infusion of isoproterenol (0.01–0.04 µg/kg/min). If no residual ectopic focus activity was observed, catheters were removed.

Postablation Evaluation and Follow-up

Continuous rhythm monitoring and a heparin infusion were maintained for 18–24 hours after the procedure. Chest radiograph, ECG, and echocardiogram were obtained within 6 hours of return from the laboratory. Early subjects had measurements of serum creatine phosphokinase (CPK) immediately after ablation and again 12 hours after the procedure, but this testing was later omitted from the protocol. Patients were observed for a minimum of 24 hours in the hospital and underwent acute follow-up testing with Holter monitoring and/or repeat isoproterenol challenge before discharge.

Outpatient follow-up visits were scheduled for 1, 6, and 12 months after the procedure. Routine testing included ECG, Holter monitoring, and repeat echocardiogram.

Results

Mapping and Ablation Results

The EAT focus was mapped to the left atrium in seven patients and to the right atrium in five (Figure 1). Left atrial foci tended to cluster near the pulmonary veins but were also seen on the anterior atrial roof and near the orifice of the left atrial appendage. Right atrial foci were usually mapped toward the right atrial appendage, except for one case (patient 5) where the EAT arose from the high posterior roof of the right atrium in close proximity to the sinus node. “Sinus node reentry” seemed quite unlikely in this patient based on the wide rate variation of the clinical arrhythmia and failure to terminate tachycardia with attempted overdrive pacing.

Atrial mapping data (Table 3) identified local electrical activity at the EAT focus that preceded the onset of the surface P wave by 20–60 msec (median, 42 msec). The signals were otherwise unremarkable and contained no evidence of early extra potentials, fractionation, or continuous electrical activity (Figures 2A and 2B). In two cases, there was transient EAT termination from local catheter pressure at the target site during precision mapping, which correctly predicted the site for successful ablation. In all other instances, activation time alone was used to locate the focus.

The number of 10-second “test lesions” used for this series ranged from 0 to 61 (median, 7), and the number of full duration applications ranged from 1 to 11 (median, 2). No impedance rises were encountered. As noted in Table 2, the initial response of the EAT focus to a successful RF lesion was somewhat variable but always rapid. On eight occasions, the EAT simply stopped abruptly during the initial few seconds of RF application (Figure 3A). In two patients, there was transient EAT acceleration before the focus was extinguished (Figure 3B); in one case, the EAT slowed over a few beats before termination (Figure 3C). The time between onset of RF output and initial rate change varied from 0.5 to 6.0 seconds (median, 2.0 seconds), and the time to EAT termination ranged from 0.5 to 10 seconds (median, 2.0 seconds).

Procedure results are summarized in Figure 4. For 11 of 12 patients (92%), the EAT was successfully eliminated using RF energy. Two ablation sessions were required in one patient (patient 4), who was receiving amiodarone until 1 week before study and arrived in the laboratory for the initial procedure with sinus rhythm. High-dose isoproterenol was used to initiate the EAT, but tachycardia was quite intermittent, thereby complicating both mapping accuracy and assessment of efficacy. Although the ablation initially appeared successful, recurrent EAT (at slower rates compared with baseline) was noted 2 months later by follow-up Holter. This patient returned for a second ablation session at which time the focus was more active and could be accurately mapped and ablated in a 1.5-hour procedure. One additional patient (patient 10), who was on sotalol until 1 week before the procedure, had no spontaneous EAT and minimal response to isoproterenol at an initial catheterization performed under general anesthesia. Accurate mapping was not possible. The focus was successfully ablated at a second procedure performed with light intravenous sedation 1 week later when EAT was nearly incessant.

Ablation failed despite prolonged efforts in only one patient (patient 7). The EAT focus in this instance was well mapped to the orifice of the right atrial appendage, where local activation preceded the P wave by 45 msec. Of interest, signals recorded from adjacent atrial tissue in this area were noticeably fractionated and low amplitude. Several RF applications were attempted but resulted only in acceleration of the EAT, which ultimately degenerated to atrial fibrillation. After cardioversion, EAT was still present at a faster rate with a more irregular pattern compared with baseline. At this point, an atrial angiogram was performed and revealed the presence of a large saccular aneurysm at the anterior right atrium (Figure 5) that had not been appreciated on the preprocedure echocardiogram due to its retrosternal location. A decision was made to proceed to surgery, where diffuse fibrous dysplasia of the anterior right atrium was noted in addition to the aneurysm. Abnormal atrial tissue was resected, and the patient did well with normal sinus rhythm thereafter.

![Figure 1. Diagrammatic map of ectopic atrial tachycardia foci locations (circles). Numbers identify the 12 study patients as listed in Table 1.](http://circ.ahajournals.org/doi/10.1161/CIRCULATIONAHA.111.032581)
Intracardiac electrograms and surface ECG recorded during mapping of a left-sided (A) and right-sided (B) ectopic atrial focus. In both examples, the distal electrode pair of the mapping/ablation catheter (MAPd) is located at a site where successful ablation was achieved. Local atrial activation precedes the onset of the P wave (dotted line) by more than 20 msec.
Procedure times ranged from 1.5 to 7.0 hours (median, 4.0 hours) with fluoroscopy times of 37–157 minutes (median, 77 minutes). Total hospital stays (excluding the patient sent to surgery) ranged from 36 hours to 4 days (median, 48 hours).

Measurements of serum CPK were available from the first six patients in this series. Maximum values for total CPK varied between 22 and 464 mU/ml with an MB fraction of 6–17%. No clear correlation was found between CPK data and RF lesion number or procedure duration. This testing was not performed in later patients.

**Complications**

One 10-month-old child (patient 5) had EAT with an atrial rate in excess of 200 min⁻¹ mapped to the right atrium just anterior to the sinus node. Although sinus node damage was considered possible with ablation in this region, the patient was quite ill with severe mitral regurgitation and low cardiac output (mixed venous saturation, 31%) such that the risk was deemed justified. The EAT was successfully ablated, although the initial recovery rhythm was slow sinus alternating with a junctional escape. After 3 days of observation, sinus rhythm at physiological rates returned, and he thereafter remained in normal rhythm.
No additional complications were encountered. Post-procedure echocardiograms showed no evidence of pericardial effusion, deterioration in ventricular function, or intracardiac thrombus. No blood product administration was required.

Follow-up

Outpatient follow-up extended from 2 months to 21 months (median, 13 months). Except for the patient who required a second ablation session and the patient sent for surgery, all others remained in sinus rhythm without the need for medications after a successful first procedure. No “new” arrhythmias related to the RF lesion(s) have been detected on follow-up Holter monitoring. All patients recovered normal ventricular size and shortening fraction on follow-up echocardiograms, including the patient initially referred for cardiac transplantation. The mitral regurgitation noted before ablation in the 10-month-old child resolved completely as ventricular dilation regressed. The time course for

<table>
<thead>
<tr>
<th>Patient</th>
<th>Focus location</th>
<th>Time before P wave (msec)</th>
<th>10-Second RF applications (No.)</th>
<th>Long RF applications (No.)</th>
<th>Response to successful RF application</th>
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<tbody>
<tr>
<td>1</td>
<td>LA</td>
<td>50</td>
<td>11</td>
<td>2</td>
<td>Terminate at 2.5 seconds</td>
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<td>LA</td>
<td>60</td>
<td>61</td>
<td>11</td>
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<tr>
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<td>RA</td>
<td>50</td>
<td>6</td>
<td>5</td>
<td>Terminate at 2.0 seconds</td>
</tr>
<tr>
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<td>20</td>
<td>8</td>
<td>2</td>
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<td>RA</td>
<td>35</td>
<td>0</td>
<td>1</td>
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</tr>
<tr>
<td>5</td>
<td>RA</td>
<td>30</td>
<td>21</td>
<td>4</td>
<td>Terminate at 5.0 seconds</td>
</tr>
<tr>
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<td>0</td>
<td>1</td>
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</tr>
<tr>
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<td>RA</td>
<td>45</td>
<td>26</td>
<td>3</td>
<td>Failed (surgery)</td>
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<tr>
<td>8</td>
<td>LA</td>
<td>25</td>
<td>8</td>
<td>4</td>
<td>Terminate at 4.0 seconds</td>
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<tr>
<td>9</td>
<td>RA</td>
<td>45</td>
<td>7</td>
<td>2</td>
<td>Accelerate at 6.0 seconds; terminate at 13.0 seconds</td>
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<tr>
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<tr>
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<tr>
<td>12</td>
<td>LA</td>
<td>30</td>
<td>2</td>
<td>1</td>
<td>Decelerate at 0.5 seconds; terminate at 2.0 seconds</td>
</tr>
</tbody>
</table>

Median, 42 msec Median, 7 Median, 2 Median termination time, 2.0 seconds

RF, radiofrequency; LA, left atrium; RA, right atrium.
improvement in ventricular performance was generally rapid (within 1–2 months), although echocardiographic variables did not normalize in two cases until 6 months (patient 10) and 9 months (patient 2) after the ablation.

Discussion

EAT is well recognized as a correctable cause of cardiomyopathy. Although it is a rare form of supraventricular tachycardia in both children and adults, the hemodynamic consequences of poor rhythm control may ultimately necessitate aggressive therapy. Transcatheter ablation is now evolving as a realistic alternative to chronic pharmacological or surgical management of this condition. There are two potential targets for effective ablation. The first involves permanent interruption of the normal atrioventricular conducting system, allowing the EAT to persist but controlling the ventricular rate. This technique has been employed successfully for EAT and other refractory atrial arrhythmias but creates pacemaker dependence. A more attractive option is direct eradication of the EAT focus. Experience with this later method had been limited until the introduction of RF current as an ablation energy source, since the technique of DC fulguration carried a potential risk of perforation through the relatively thin atrial wall.

While RF ablation is currently very well established as definitive therapy for tachyarrhythmias due to accessory atrioventricular pathways in patients of all ages, this report represents the first large series to describe its application to EAT. Unlike accessory pathways, accurate localization of this ectopic atrial focus must involve a three-dimensional map and is further confounded by the absence of reliable electrogram markers (such as accessory pathway potentials) apart from local activation times. Despite these difficulties, the technique was successful in 92% of our patients, with the sole failure occurring in a patient who had diffuse atrial fibrosis requiring surgical excision. All subjects had prompt reversal of the ventricular dysfunction caused by the tachyarrhythmia, and all remain in sinus rhythm without the need for medications. While some series describing surgical therapy for EAT report arrhythmia recurrence after excision of right atrial foci, we detected recurrence in only one patient with a suboptimal map at initial study due to residual amiodarone effect. Successful ablation was later achieved in this case when the focus was more active. Although the follow-up for this group is short term (median, 13 months), the initial results remain quite encouraging and suggest that the technique is at least as effective as a surgical procedure.

The mapping/ablation features that best predicted successful EAT elimination included 1) local electrical activity preceding onset of the surface P wave by 20–60 msec (median, 42 msec), 2) rapid resolution of EAT within less than 10 seconds of beginning RF application, and, on 2 occasions, 3) transient EAT suppression due to local catheter pressure over the target site. The critical requirement for a high-quality map was the presence of nearly incessant tachycardia, which occasionally required the administration of isoproterenol. As demonstrated by two patients in this series, residual antiarrhythmic drug effects and/or the deep level of sedation accompanying general anesthesia can suppress the EAT in the laboratory environment. Except for cases involving very young children (in whom we prefer a general anesthetic), it may be advisable to perform EAT ablation under light intravenous sedation whenever possible.

Since this series marked our early experience with EAT ablation, we tended to be very compulsive with the mapping technique. Direct left atrial recording was preferred over a coronary sinus electrode, even if this required transeptal puncture. In cases where the focus can be clearly mapped to the right atrial appendage, this degree of intervention may not be necessary for an accurate map. However, for EAT foci at all other locations, we found a coronary sinus recording to be less than adequate for localization. Indeed, many left atrial foci produce a similar misleading signal on the coronary sinus electrode involving earliest activation near the mouth of coronary sinus. There does not appear to be a reliable substitute for direct left atrial mapping if the EAT involves any site other than the right atrial appendage.

RF lesions at the atrial level have been shown to produce transmural necrosis that heals with a well-organized fibrous scar and have not been associated with significant risk for either early or late perforation. The RF technique is also free of the barotrauma and catheter fling seen with DC ablation, further decreasing the likelihood of acute atrial trauma. RF current would thus appear well suited for atrial muscle ablation, including the fragile areas near the atrial appendages. This report supports the general safety of the technique in children as young as 10 months.

Fluoroscopy times and procedure times encountered in this series were longer than those for a typical diagnostic electrophysiology study but were not dissimilar to times reported for RF ablation of accessory pathways. Innovations in mapping catheter design may permit faster simultaneous mapping of multiple atrial sites and are under investigation at our center. Additionally, laboratory equipment is now available with the capacity for pulsed fluoroscopy at slow frame rates (7.5 sec−1), which will reduce radiation exposure to the patient and operator without significantly compromising catheter control. Overall procedure times still require reduction but will likely shorten as more experience is gained with this technique.

Since atrial RF lesions are transmural, one cannot safely comment on whether the foci ablated in this series involved endocardium, epicardium, or atrial myocytes. However, in contrast to the aforementioned surgical reports, which suggested that some cases of EAT represent diffuse atrial disease, 11 of the 12 patients treated in this series (including four of five with right atrial EAT) appeared to have a point source arrhythmia that could often be eliminated by a single RF lesion. The ultimate size of such lesions is unlikely to be more than 10 mm in surface diameter. Furthermore, early disappearance of EAT at a median of 2.0 seconds into the RF application suggests that an EAT focus could actually be smaller then even 2 mm.

In this early experience involving 12 patients with drug-resistant EAT, catheter ablation with RF current appeared to be an effective and safe therapeutic option. At our institution, RF ablation is now recommended as first-line therapy for any age child who has developed
myocardial dysfunction from the tachyarrhythmia. For patients with well-preserved ventricular performance, an initial drug trial with β-blocker is still attempted, reserving ablation as second-line therapy to prefer for more potent antiarrhythmic drugs or surgery.

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