Ablation of cardiac tissues by an electrode catheter technique for treatment of ectopic supraventricular tachycardia in adults

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ABSTRACT Five patients with chronic or recurrent ectopic supraventricular tachycardias unresponsive to drugs underwent programmed stimulation, endocardial mapping, and attempted catheter ablation of the arrhythmia focus. For attempted ablation, an intracardiac electrode catheter was positioned near the exit point of the tachycardia and served as the cathode while a chest wall patch served as the anode. In two patients with tachycardia originating near the coronary sinus, discharges of 200 or 400 J each were delivered to two electrodes at the earliest area of endocardial activation. These two patients with incessant tachycardia remain free of tachycardia for 17 and 11 months, respectively. In one patient with tachycardia originating from the right atrial appendage, both catheter and surgical ablation proved unsuccessful in that a new focus of atrial tachycardia supervened. This patient subsequently underwent successful catheter ablation of the atrioventricular junction. Two patients with junctional tachycardia underwent catheter ablation of the atrioventricular junction. Complete atrioventricular block followed atrioventricular junctional ablation and these patients required permanent cardiac pacing. The junctional tachycardia was replaced by sinus rhythm with episodes of unsustained atrial tachycardia. However, after 13 ± 5 months follow-up, neither of the patients require antiarrhythmic drugs. Catheter ablation can be effective for atrial foci near the coronary sinus or, and can be performed with preservation of atrioventricular conduction. Arrhythmia ablation is possible in those with atrioventricular junctional tachycardia but requires the sacrifice of atrioventricular conduction. After ablation, other automatic atrial foci may become operative and complicate use of dual-chamber pacemakers.


CHRONIC ECTOPIc tachycardias arising above the bifurcation of the bundle of His occur more commonly in children1-3 than adults.4 Gillette et al.3 have stressed the poor prognosis and risk of intractable heart failure or sudden death in some of these children. When ectopic supraventricular tachycardia resists pharmacologic suppression,3, 4 ablation of the atrioventricular junction is required to control the rapid ventricular rates.6-8 Recently, closed-chest catheter electrocautery has been used for local ablation of arrhythmogenic tissues.9-12 Direct electrode catheter ablation within the atrium is feasible if an ectopic focus can be localized precisely and the atrium can be safely exposed to high-voltage discharges. Preliminary experiments performed in nine dogs have indicated that the atria can be exposed to 50 to 100 J shocks without complication.13

To date, catheter ablation of atrial or junctional ectopic14 tachycardia has been reported in a small number of pediatric patients.15-17 This communication summarizes our experience with use of catheter ablation in adult patients with persistent or repetitive ectopic supraventricular arrhythmias refractory to drugs.

Methods

Clinical data. Five adults with incessant or recurrent drug-refractory supraventricular tachycardia were referred for evaluation and treatment. Symptomatic tachycardia had been present for 2 to 17 years in each. Two patients had automatic junctional tachycardia and three had automatic atrial tachycardia. Clinical data on these three men and two women are summarized in table 1.

The two patients with automatic junctional tachycardia demonstrated irregular junctional rate and atrioventricular dissociation during tachycardia. Rate of tachycardia accelerated during exercise and prevalence decreased after administration of β-
blocking drugs before adverse effects forced their discontinuation. Side effects included intolerable central nervous system depression in one patient (No. 1) and symptomatic bradycardia in the other (No. 2). One patient developed junctional tachycardia after a third surgical procedure for closure of atrial and ventricular septal defects and aortic valvuloplasty for congenital aortic insufficiency. The other patient had a structurally normal heart by echocardiography and radionuclide ventriculography.

Each of the three patients with automatic atrial tachycardia had one dominant P wave morphology during tachycardia. The frontal plane P wave axis of the tachycardia was superior in the two patients with atrial foci near the coronary sinus and normal in the patient with tachycardia arising from the right atrial appendage. Of the two patients with the atrial focus near the coronary sinus, one (patient 4) underwent closure of an atrial septal defect at age 3 and tachycardia developed 10 years later; one patient (No. 3) developed persistent atrial tachycardia after an unsuccessful attempt at surgical division of a posteroseptal accessory pathway. The patient with a right atrial appendage focus had documented persistent tachycardia for at least 16 years (patient 5). Previous unsuccessful drug treatments for each patient are listed in table 1.

Electrophysiologic study procedures. Before attempted catheter ablation, each patient underwent invasive cardiac electrophysiologic studies. Electrograms were recorded from the right atrium, His bundle, and right ventricle during tachycardia in all five patients, and from the coronary sinus in the three patients with atrial tachycardia. Atrial and ventricular overdrive pacing and programmed atrial and ventricular extrastimulation were performed during sinus rhythm in patients with atrioventricular junctional tachycardia. Similarly, overdrive pacing and programmed atrial stimulation were used for those with persistent atrial tachycardia. Overdrive pacing was continued for 30 sec at a paced cycle length at least 30 to 50 msec shorter than the tachycardia cycle length, after which overdrive was abruptly stopped and the escape rhythm was observed. The immediate effects of overdrive pacing for both the first and second postpacing cycle lengths were compared with the mean RR interval for 30 consecutive spontaneous tachycardia complexes recorded before atrial pacing to detect overdrive suppression or acceleration (see Statistical methods). It was believed that the first cycle length might be influenced by both entrance of the stimulated atrial depolarization and exit from the pacemaker site.

Junctional tachycardia was confirmed in two patients by a His bundle deflection preceding each ventricular complex and by atrioventricular dissociation. Atrial origin of tachycardia was confirmed in three patients during extensive right and left atrial (coronary sinus) endocardial mapping. Right atrial mapping was achieved by use of a single-lumen catheter with a Brockenbrough stylet (Elecath). This catheter was placed initially in the most superior portion of the right atrium and manipulated in all directions so that the atrial endocardial activation sequence was obtained at this level. The catheter was then withdrawn in 1 cm steps and the procedure was repeated to provide a detailed map of the right atrium. To map selected areas of the left atrium, the coronary sinus catheter was withdrawn from the most distant location to the coronary sinus os. A total of 18 to 20 atrial sites was mapped. The earliest surface P wave from three simultaneously recorded orthogonal leads was used as a reference.

Ablation procedure. Ablation was performed with use of a No. 6F quadrupolar electrode catheter (United States Catheters Incorporated, Billerica, MA) positioned at sites of earliest endocardial activation during tachycardia.

In the two patients with junctional tachycardia, the largest unipolar His bundle electrograms during tachycardia were 400 and 250 μV, respectively. The selected electrode was attached to the cathodal output of a standard defibrillator (Physio-Control Lifepak 6, Redmond, WA) and the anodal sink was connected to an 8 cm patch electrode (R-2 Corp., Skokie, IL) placed over the left scapula. To patients under sodium thiopental anesthesia, two 200 J shocks were delivered to the electrode showing the largest unipolar His bundle deflection. After atrioventricular junctional ablation, both patients developed complete atrioventricular block with ventricular escape cycle lengths of 1220 and 1300 msec (heart rates 46 and 50 beats/min), respectively. When antegrade atrioventricular block persisted more than 24 hr after ablation, permanent cardiac pacemakers were inserted.

In two patients, origin of tachycardia was mapped to the posteroseptal right atrium, near the coronary sinus os. The ablation catheter tip was placed in the coronary sinus for stabilization, and the proximal two electrodes were positioned carefully just outside the coronary sinus os under fluoroscopy. This technique has been described previously elsewhere.18 Two shocks were delivered between the two most proximal electrodes (cathode) to a patch on the anterior chest wall (anode) (see table 3). The total stored energy was 800 J for patient 3 and 400 J for patient 4. After ablation, sinus rhythm supervened in both.

In patient 5, earliest atrial activity was localized to the right atrial appendage. After mapping, the Brockenbrough catheter was replaced by a No. 6F bipolar temporary pacing catheter and

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Clinical data</th>
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<tbody>
<tr>
<td><strong>Patient no</strong></td>
<td><strong>Age and sex</strong></td>
</tr>
<tr>
<td>1</td>
<td>20, F</td>
</tr>
<tr>
<td>2</td>
<td>23, M</td>
</tr>
<tr>
<td>3</td>
<td>42, F</td>
</tr>
<tr>
<td>4</td>
<td>17, M</td>
</tr>
<tr>
<td>5</td>
<td>27, M</td>
</tr>
</tbody>
</table>

AAT = atrial automatic tachycardia; AJT = automatic junctional tachycardia; CHD = congenital heart disease; NHD = no heart disease; CM = idiopathic cardiomyopathy; WPW = Wolff-Parkinson-White syndrome; Pr = propranolol; PA = procainamide; D = disopyramide; Q = quinidine; V = verapamil; Dig = digoxin; A = amiodarone.
five shocks were delivered to the right atrial appendage. Three 100 J shocks via the two distal electrodes, and a fourth and fifth shock of 100 and 50 J, respectively, were delivered to the single most distal electrode.

After ablation, inquiries about new symptoms were made and daily physical examinations were performed. Serum creatine kinase values were obtained every 4 to 6 hr in these patients. Serum levels of creatine kinase–MB were determined by electrophoresis (the normal values for our laboratory are 0 to 16 IU). Echocardiographic images were recorded before and after ablation, and changes in segmental motion were assessed with the use of Simpson’s rule.19 This protocol was approved by the Committee on Human Experimentation, University of California, San Francisco.

Statistical methods. All averaged variables are expressed as the mean ± SD. Significant differences between group means were believed present when the probability of no difference was less than .05. The 30 consecutive spontaneous tachycardia complexes were assumed to be normally distributed, and the mean and SD were determined as the tachycardia cycle length.

Results

Electrophysiologic studies. In the two patients with atrioventricular junctional tachycardia, a His bundle deflection initiated the tachycardia, followed by persistent ventriculoatrial dissociation (patient 1, figure 1) or episodes of ventriculoatrial dissociation (patient 2). This finding excludes atrioventricular reciprocating tachycardia and is strong evidence against atrioventricular junctional reentrant tachycardia. Tachycardias arose spontaneously and were not initiated by programmed stimulation in these two patients. Overdrive atrial pacing at various cycle lengths showed no consistent effects on the postpacing tachycardia cycle lengths (table 2; figure 2). There was no significant difference between the mean control cycle length of the tachycardia and the first and second postpacing cycle lengths (table 2). These observations are most compatible with automatic tachycardia arising at the atrioventricular junction. Although for purposes of this report, these two patients were considered to have supraventricular tachycardia, the available data cannot exclude the possibility that the tachycardia originated

![FIGURE 1. Recordings of a typical tachycardia from patient 1. Shown are simultaneous surface leads V1, I, and III and intracardia recordings from the high right atrium (HRA) and the His bundle electrogram (HBE). An irregular supraventricular tachycardia with atrioventricular dissociation compatible with a junctional ectopic tachycardia is present.](image)

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Tachycardia cycle length (msec)</th>
<th>Location</th>
<th>P axis (degrees)</th>
<th>Postspacing cycle length (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>SVT SR</td>
<td>First cycle</td>
</tr>
<tr>
<td>1</td>
<td>320–330 (327 ± 31)</td>
<td>AVJ</td>
<td>— —</td>
<td>350 ± 10</td>
</tr>
<tr>
<td>2</td>
<td>420–470 (448 ± 14)</td>
<td>AVJ</td>
<td>— —</td>
<td>458 ± 162</td>
</tr>
<tr>
<td>3</td>
<td>360–370 (363 ± 4)</td>
<td>CS os</td>
<td>—90 —30</td>
<td>427 ± 170</td>
</tr>
<tr>
<td>4</td>
<td>340–350 (347 ± 3)</td>
<td>CS os</td>
<td>—110 +75</td>
<td>258 ± 18</td>
</tr>
<tr>
<td>5</td>
<td>500–520 (507 ± 7)</td>
<td>RAA</td>
<td>+15 +45</td>
<td>700 ± 40</td>
</tr>
</tbody>
</table>

AVJ = atrioventricular junction; CS os = coronary sinus os; RAA = right atrial appendage; SVT = supraventricular tachycardia; SR = sinus rhythm.

*p < .05.
from the bundle of His. In both patients, rate and prevalence of tachycardia increased with exercise and decreased after propranolol, but β-adrenergic–blocking drugs caused intolerable side effects when taken over a long term.

In all three patients with persistent atrial tachycardia, periods of atrioventricular block were recorded after carotid massage or administration of β-blockers, thus excluding an atrioventricular reciprocating mechanism. In addition, the tachycardia could not be terminated by either overdrive or coupled atrial or ventricular extrastimuli. Atrial overdrive pacing resulted in transient acceleration of tachycardia (patient 4) or transient suppression of the first and second postpacing cycle lengths (patients 3 and 5, table 2). These findings were interpreted as being most consistent with enhanced automaticity of an atrial focus.4, 20–23 A representative atrial endocardial map from patient 4 is shown in figure 3. Exercise increased the rate of atrial tachycardia to 160 and 200 beats/min in patients 4 and 5, respectively, but symptoms of tachycardia were unimproved by maximal tolerable doses of β-adrenergic blockers in all three patients.

The ablative procedure was uncomplicated in each patient (figure 4). None developed chest pain, hemodynamic instability, or new arrhythmias. Twelve-lead electrocardiograms showed change in P wave contour and rate in each patient, associated with reversion to

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**FIGURE 2.** Atrial overdrive pacing was initiated during tachycardia in patient 2. Surface leads V₁, I, and III are shown with the right atrial (RA) and His bundle electrograms (HBE). The baseline cycle length of tachycardia was 448 ± 14 msec. When atrial pacing at cycle length 300 msec is abruptly halted, an irregular junctional tachycardia is present.

**FIGURE 3.** Schema of atrial endocardial map for patient 4. The earliest area of atrial endocardial activation relative to the surface P waves was −50 msec and occurred at the os of the coronary sinus (CS). There was concentric spread of atrial activation to other atrial sites. SVC = superior vena cava; IVC = inferior vena cava; TV = tricuspid valve.
sinus rhythm (figure 4; table 2). Patients undergoing atrioventricular junctional ablation had complete atrioventricular block after shock and their course was uncomplicated.

**Myocardial injury.** Peak values for myocardial creatine kinase isoenzyme were recorded 12 to 24 hr after ablation and ranged from 12 to 81 IU/liter (table 3). No patient developed a new murmur or evidence of congestive heart failure, and serial chest x-rays revealed no cardiopulmonary congestion in the initial week after ablation. Technitium pyrophosphate scans (two patients) revealed focal atrial uptake in patient 1 and no uptake in patient 4.

**Myocardial function.** Comparison of echocardiographic images obtained before and after ablation revealed transient hypokinesis of the summit of the ventricular septum at 24 hr in patient 1, with significant improvement in septal motion at 72 hr. The remaining four patients developed no detectable change in cardiac chamber size or motion over the 72 hr period immediately after atrial ablation.

**Clinical follow-up.** All antiarrhythmic medications were stopped after ablation. Over a follow-up period of 7 to 48 months, one patient developed recurrence of the preablation atrial tachycardia. The two patients with junctional tachycardia developed transient episodes of non-sustained atrial tachycardia and two patients remained free of symptomatic atrial tachycardia after ablation.

The two patients with junctional tachycardia experienced symptomatic improvement after ablation, including a reduction in palpitations and improved tolerance of exertion with resolution of dizziness, dyspnea, or palpitations at rest. Both received DDD pacemakers (Intermedics Cosmos 283) capable of high upper tracking rates (180/min). Postablation radionuclide ventriculograms showed normal resting ventricular function and appropriate increases in cardiac output and stroke volume with exercise during 1:1 atrioventricular pacing. However, ambulatory electrocardiograms confirmed brief periods of atrial tachycardia in both patients. Atrial rates up to 145 and 180/min, respectively, were recorded (figure 5) since the pacemaker allowed for transmission of impulses to the ventricles. One patient (No. 2) preferred conversion to the VVI pacing mode and is presently fully active (plays racketball) and without symptoms. The second patient (No. 1) opted to retain DDD-type pacing since her work required rapid rates and she experienced only infrequent symptoms due to the spontaneous atrial tachycardia.

The patient with tachycardia mapped to the right atrial appendage had recurrence of the original arrhythmia 24 hr after ablation and underwent surgical removal of the right atrial appendage 9 days after ablation. Intraoperative mapping localized the origin of tachycardia to the right atrial appendage. The appendage was resected and encircling cryogenic lesions were applied to its base. The resected atrial appendage was carefully examined for evidence of new injury related to ablation. Grossly, it contained several thickened, dark red lesions. Histologically, these thickened areas showed hemorrhage, myocardial focal necrosis of epicardial fat, and granulation tissue with mild chronic inflammation (figure 6). Other regions of the atrial wall showed patchy interstitial fibrosis without inflam-
TABLE 3

Effects of ablation and follow-up

<table>
<thead>
<tr>
<th>Patient no</th>
<th>Energy (J)</th>
<th>CK-MB Peak U/liter (% of total CK&lt;sup&gt;a&lt;/sup&gt;)</th>
<th>Echocardiographic findings</th>
<th>Arrhythmia recurrence</th>
<th>Symptoms</th>
<th>Follow-up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>200 × 2</td>
<td>12 (8%)</td>
<td>Transient ventricular septal dyskinesia</td>
<td>Unsustained atrial tachycardia</td>
<td>Rare palpitations</td>
<td>48</td>
</tr>
<tr>
<td>2</td>
<td>200 × 2</td>
<td>81 (13%)</td>
<td>No change</td>
<td>Unsustained atrial tachycardia; converted to VVI pacing</td>
<td>None</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>400 × 2</td>
<td>14 (2%)</td>
<td>No change</td>
<td>Atrial flutter&lt;sup&gt;b&lt;/sup&gt;</td>
<td>None</td>
<td>17</td>
</tr>
<tr>
<td>4</td>
<td>200 × 2</td>
<td>49 (2%)</td>
<td>No change</td>
<td>None</td>
<td>None</td>
<td>11</td>
</tr>
<tr>
<td>5</td>
<td>100 × 4</td>
<td>29 (12%)</td>
<td>No change</td>
<td>Recurrence of original arrhythmia in 24 hr&lt;sup&gt;c&lt;/sup&gt;</td>
<td>None</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>50 × 4</td>
<td></td>
<td></td>
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</tbody>
</table>

CK-MB = myocardial isoenzyme of creatine kinase.

<sup>a</sup>Normal <3%.

<sup>b</sup>Transient atrial flutter with episode of pleuropericarditis 2 months after ablation.

<sup>c</sup>Underwent surgical resection and finally catheter ablation of the atrioventricular junction.

Discussion

Our initial experience using electrode catheters for ablation of ectopic supraventricular tachycardia foci indicates that this technique is promising. For example, in two of the three patients with atrial ectopic tachycardia, control of the arrhythmia was possible without the need for permanent cardiac pacing. Of the patients with automatic junctional tachycardia, one is completely asymptomatic while the other is much improved after ablation. In four of the five patients arrhythmia was controlled without need for long-term drug therapy, and the fifth is similarly asymptomatic but only after undergoing ablation of the atrioventricular junction.

Before attempting atrial ablation in man, we evaluated the safety of catheter electrocautery in the atrium.
FIGURE 6. A, Photomicrograph of the atrium with ablation injury. A pedicle with intramural hemorrhage is attached to a region with transmural inflammation and focal necrosis of adjacent epicardial fat (hematoxylin-eosin, original magnification × 20). B, Higher magnification of the ablation injury showing an area of myocardial necrosis and granulation tissue (hematoxylin-eosin, original magnification × 200).
FIGURE 7. Photomicrograph of atrial appendage from a region remote from the delivered shock. The wall shows areas of fibrosis intermixed with normal myocardial fibers (hematoxylin-eosin ×75) intermixed with normal myocardial fibers (hematoxylin-eosin ×75).

of nine dogs. This confirmed that permanent transmural atrial injury could be achieved with one to two direct-current discharges, 50 to 100 J each, delivered to a single catheter electrode against the atrial free wall. At this energy level, the acute injury was limited to the atrial wall, without perforation of the atrial free wall or thrombosis of adjacent coronary arteries. Similar observations have been noted by others. The histologic appearance of ablation injury in these canine atria after 1 week is similar to that seen in the resected atrial appendage of patient 5 nine days after catheter ablation. The atrial appendage contained transmural injury with an intense inflammatory response, granulation reaction, and necrosis of epicardial fat, but no perforation. A more diffuse, healed myocardial reaction was also present that appeared to antedate the ablation, perhaps related to the patient’s clinical cardiomyopathy. Significantly, patchy fibrosis within the atrial appendage and the recurrence of tachycardia after surgical resection suggested the potential for latent arrhythmogenic areas remote from the ablation site.

Results of electrophysiologic study and endocardial catheter mapping in three patients with atrial tachycardia were compatible with a focal rather than a reentrant mechanism. The ease of catheter mapping and stable catheter position during ablation may have influenced outcome. The coronary sinus os allows for precise mapping of the posterior septum as well as stable catheter placement during ablation. In contrast, catheter mapping in the right atrial appendage was awkward and stable placement of the catheter during the procedure was difficult. These technical factors may have contributed to our failure in patient 5. Alternatively, the focus of tachycardia may have been localized to a very small number of cells and present techniques lack adequate precision for its identification.

After ablation, the two patients with junctional tachycardia experienced palpitations due to episodes of unsustained atrial tachycardia. In one, these episodes were sufficiently frequent to require reprogramming of his pacemaker to the VVI mode. These atrial arrhythmias may have been present in preablation tracings, but would have been very difficult to distinguish from the dominant junctional tachyarrhythmias. Both
patients experienced marked improvement in symptoms, since before ablation both had experienced severe dizziness or syncope associated with palpitations. The findings in the two patients with junctional tachycardia and in patient 5 appear to indicate propensity for emergence of other ectopic atrial foci after ablation of a dominant tachycardia substrate. The emergence of these new foci during periods of activity or excitement suggests sensitivity to sympathetic tone or circulating catecholamines, behavior similar to that of the ablated tachycardia substrate in these patients. Whether this reflects multifocal potential of arrhythmogenic tissues or atrial damage incident to catheter or surgical ablation is not known. In addition, unsustained atrial tachycardia is frequently recorded on 24 hr ambulatory recordings of patients with and those without heart disease.

In conclusion, our limited experience suggests that catheter ablation may play a useful role in the management of selected patients with drug-refractory ectopic supraventricular tachycardia. The chief limitation in our series was the emergence of other atrial arrhythmias confounding the use of DDD pacemakers. These patients may respond best to pacemakers with rates governed by nonelectrocardiographic physiologic changes. The precise role of attempted catheter ablation of ectopic supraventricular tachycardia vs the surgical approach is still unclear. Of note is the finding that three of our five patients developed their arrhythmias after a cardiac surgical procedure and one developed a new atrial focus after surgical amputation of the right atrial appendage. Attempted catheter ablation would appear to be a reasonable approach for selected patients to avoid the morbidity and risks of cardiac surgery. However, great caution should be used in applying these catheter techniques to foci located in the thin-walled areas of the superior right atrium and appendage because of risk of myocardial perforation. These factors must be carefully weighed in the decision to attempt catheter ablation.

References
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FIGURE 8. A, Preablation 12-lead electrocardiogram showing an atrial tachycardia (144/min). Note the upright P wave in leads II and aVF, with a frontal plane P wave axis +75 degrees. B, Four months after surgical resection of the right atrial appendage, a new atrial tachycardia is present. The P waves are inverted in leads III and aVF and the mean P wave frontal axis is -60 degrees.
THERAPY AND PREVENTION—CATHETER ABLATION

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