Electrophysiological Mapping and Ablation of Intra-Atrial Reentry Tachycardia After Fontan Surgery With the Use of a Noncontact Mapping System

Timothy R. Betts, MRCP; Paul R. Roberts, MRCP; Stuart A. Allen; Anthony P. Salmon, MD; Barry R. Keeton, MD; Marcus P. Haw, FRCS; John M. Morgan, MD

Background—Atrial tachyarrhythmias are a complication of Fontan surgery. Conventional electrophysiological mapping and ablation techniques are limited by the complex anatomic and surgical substrate and a high arrhythmia recurrence rate. This study investigates the use of noncontact mapping to identify arrhythmia circuits and guide ablation in Fontan patients.

Methods and Results—Eleven arrhythmias were recorded in 6 patients. Noncontact mapping improved recognition of the anatomic and surgical substrate and identified exit sites from zones of slow conduction in all clinical arrhythmias. Radiofrequency linear lesions were targeted across these critical zones in 5 patients. One patient underwent surgical cryotherapy. Although immediate success was achieved in 3 of 5 patients with radiofrequency ablation, 2 patients had a recurrence after a mean of 6.4 months of follow-up. The patient who underwent cryoablation remains free of arrhythmias.

Conclusions—Noncontact mapping can identify arrhythmia circuits in the Fontan atrium and guide placement of ablation lesions. Arrhythmia recurrence is high, possibly because of inadequate lesion creation rather than inaccurate mapping and lesion targeting. (Circulation. 2000;102:419-425.)

Key Words: mapping | Fontan procedure | ablation

Occurrence of atrial arrhythmias after Fontan surgery is well documented.1–4 Surgical and natural barriers to conduction contribute to the arrhythmia substrate. Antiarrhythmic drug therapy and antitachycardia pacing offer limited arrhythmia control.5 6 A few small series report the use of electrophysiological mapping and radiofrequency ablation techniques in patients with a variety of congenital heart conditions.5 7–13 Although initial success rate may be as high as 75%, recurrence rates of up to 50% are common during short-term follow-up. Results are less favorable in Fontan patients. Because conventional electrophysiological techniques are limited in such patients, we have explored the use of a noncontact, multisite mapping system to identify tachycardia mechanisms and guide ablation in 6 patients with atrial arrhythmias after Fontan surgery.

Methods

Patient Population

The study was approved by the Southampton and SW Hants Regional Ethics Committee. Informed consent was obtained from each subject. All procedures were performed in accordance with local guidelines. Six patients were studied. Three patients had tricuspid atresia and 3 had double-inlet single ventricles. Previous cardiac operations ranged from 1 to 3. Five patients had variations of the classic atriopulmonary connection; 2 had right atrial–to–pulmonary artery anastomoses, 3 had left atrial roof–to–pulmonary artery anastomoses. The sixth patient had a valved conduit from the right atrium to the right ventricle. The patients with double-inlet ventricles had their tricuspid valves patched or excluded by a pericardial baffle. One patient had incessant atrial tachycardia and 5 had documented paroxysms of symptomatic atrial tachyarrhythmias, either self-limiting or requiring repeated DC cardioversion. All patients had failed a median of 3 drug therapies. Assessment included detailed history and physical examination, ECG, 24-hour ambulatory monitoring, transthoracic echocardiography, and full cardiac catheterization. Mean right atrial diameter was 6.8 (±0.8) cm. Mean length of follow-up from first Fontan procedure was 13.5 (±3.9) years; mean follow-up from most recent Fontan procedure was 8.8 (±1.3) years. Only patient 6 had undergone a previous electrophysiological study.

Five patients were considered for surgical conversion to total cavopulmonary connection (TCPC), indicated on hemodynamic grounds (gradient across the atriopulmonary connection, significantly elevated right atrial pressures, limiting symptoms of heart failure, or protein-losing enteropathy). Arrhythmia mapping was performed as a prelude to possible surgical cryoablation at the time of TCPC conversion. Radiofrequency ablation was attempted as a palliative measure while surgical treatment was considered. Patient details and results of the study are summarized in Tables 1 and 2.
TABLE 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/Sex</th>
<th>Congenital Defect</th>
<th>Relevant Surgery</th>
<th>Time Since Most Recent Surgery, y</th>
<th>Arrhythmia</th>
<th>NYHA Status</th>
<th>Previous Drug Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22/M</td>
<td>Double-inlet ventricle</td>
<td>Direct RA-PA anastomosis</td>
<td>8</td>
<td>Incessant IART</td>
<td>II</td>
<td>Amiodarone, atenolol</td>
</tr>
<tr>
<td>2</td>
<td>15/F</td>
<td>Tricuspid atresia</td>
<td>LA-PA anastomosis; baffle from ASD to LA roof</td>
<td>10</td>
<td>Paroxysmal IART</td>
<td>II</td>
<td>Sotalol, amiodarone, digoxin, propafenone</td>
</tr>
<tr>
<td>3</td>
<td>16/M</td>
<td>Double-inlet LV</td>
<td>LA-PA anastomosis; baffle from RA floor to LA roof</td>
<td>10</td>
<td>Recurrent IART; DC cardioversion ×3</td>
<td>III</td>
<td>Amiodarone, disopyramide, digoxin, cardioversions</td>
</tr>
<tr>
<td>4</td>
<td>20/M</td>
<td>Double-inlet ventricle</td>
<td>Direct RA-PA anastomosis</td>
<td>6</td>
<td>Paroxysmal IART</td>
<td>II</td>
<td>Amiodarone, digoxin, sotalol, disopyramide, cardioversions</td>
</tr>
<tr>
<td>5</td>
<td>18/M</td>
<td>Tricuspid atresia</td>
<td>LA-PA anastomosis; baffle from ASD-LA roof</td>
<td>10</td>
<td>Paroxysmal IART</td>
<td>II</td>
<td>Atenolol, amiodarone, propafenone</td>
</tr>
<tr>
<td>6</td>
<td>20/F</td>
<td>Tricuspid atresia</td>
<td>RA-RV conduit</td>
<td>8</td>
<td>Paroxysmal IART</td>
<td>III</td>
<td>Atenolol, amiodarone, digoxin</td>
</tr>
</tbody>
</table>

NYHA indicates New York Heart Association; RA, right atrium; PA, pulmonary artery; IART, intra-atrial reentry tachycardia; LA, left atrium; ASD, atrial septal defect; and LV, left ventricle.

Noncontact Mapping System

The noncontact mapping system (EnSite 3000, Endocardial Solutions Inc) consists of a 9F multielectrode array (MEA) mapping catheter, a custom-designed amplifier, and a Silicon Graphics workstation. The methods of chamber geometry construction, inverse solution reconstruction of endocardial potentials, and the validation of noncontact mapping in the left ventricle and the right atrium have previously been reported.14–16

Mapping Procedure

Patients were studied under general anesthesia, off all antiarrhythmic medication. A bipolar catheter was positioned in the high right atrium, and a 24-pole catheter (Orbiter, Bard) was positioned around the atrial wall. When accessible, a quadripolar catheter was positioned in the coronary sinus. A 7F, 4-mm-tip, steerable ablation catheter (Stinger, Bard) was used to create chamber geometry, record contact electrograms, and deliver radiofrequency energy. Contact catheter data and 12-lead ECGs were recorded simultaneously on the Duo Laboratory system (Bard). A continuous heparin infusion was administered to keep the activated clotting time >300 seconds.

The 64-electrode MEA was passed into the right atrium through a femoral vein (Figure 1). Approximate positions of anatomic landmarks (atriotomy incisions, patches, baffles, and anastomoses) were initially identified from operation notes, angiography, and echocardiography. More precise localization was then attempted. Atriotomy incisions were identified by examining ablation catheter electrograms for double potentials during tachycardia or sinus rhythm. Patches, baffles, and atrioventricular anastomoses were identified by absent or low-amplitude electrograms, palpable ridges, and areas where a pacing stimulus failed to capture at maximum output. The margins of these identified areas together with any other identified inert or diseased myocardium were labeled onto the right atrial geometry by steering the locator signal to the ablation catheter tip. These areas were then examined with virtual unipolar electrograms and during color mapping, with a change from low to high amplitude confirming the border between inert structures and viable myocardium.

Atrial arrhythmias were induced by means of a programmed extrastimulation protocol with or without isoproterenol infusion. Data were recorded during sinus rhythm and atrial tachyarrhythmias.

Identification of Reentrant Circuit Anatomy and Suitable Ablation Sites

Activation maps and reconstructed unipolar electrograms were examined in review mode at a range of high-pass filter settings (0.1 to 32 MHz) and a low-pass filter setting of 300 MHz to determine Suitable Ablation Sites

<table>
<thead>
<tr>
<th>Patient</th>
<th>Procedure/Fluoroscopy Time, min</th>
<th>No. of Induced Arrhythmias (Clinical Arrhythmias)</th>
<th>Principal Arrhythmia Circuit</th>
<th>Cycle Length, ms</th>
<th>Breakout Site/Critical Isthmus</th>
<th>Ablation Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>270/45.2 390/79.5</td>
<td>2 (1)</td>
<td>RA macroreentry</td>
<td>285, 305</td>
<td>Between low atriotomy and lateral TV patch</td>
<td>APC-ASD, CS os-IVC, ASD-IVC, TV patch-ASD</td>
</tr>
<tr>
<td>2</td>
<td>300/59.5</td>
<td>2 (1)</td>
<td>Rotation around atriotomy scar</td>
<td>210, 220</td>
<td>Between low atriotomy and IVC</td>
<td>Atriotomy-IVC</td>
</tr>
<tr>
<td>3</td>
<td>405/46</td>
<td>3 (2)</td>
<td>Biatrial; slow conduction through baffle suture line</td>
<td>340, 375</td>
<td>Interposterior margin of interatrial baffle</td>
<td>Baffle-IVC; APC-SVC; Atriotomy-IVC</td>
</tr>
<tr>
<td>4</td>
<td>320/73 140/50.5</td>
<td>1 (1)</td>
<td>RA macroreentry</td>
<td>235</td>
<td>Between TV patch and IVC</td>
<td>TV-IVC; APC-TV patch</td>
</tr>
<tr>
<td>5</td>
<td>420/58</td>
<td>3 (1)</td>
<td>RA macroreentry</td>
<td>200, 230, 260</td>
<td>Inferior end of atriotomy incision</td>
<td>Atriotomy-IVC</td>
</tr>
<tr>
<td>6</td>
<td>Not recorded</td>
<td>1 (1)</td>
<td>Rotation around RA-RV conduit</td>
<td>220</td>
<td>Between inferior margin of conduit and IVC</td>
<td>IVC-CS-conduit</td>
</tr>
</tbody>
</table>

RF indicates radiofrequency; RA, right atrium, TV, tricuspid valve; APC, atrio pulmonary connection; ASD, atrial septal defect; CS, coronary sinus; IVC, inferior vena cava; SVC, superior vena cava; RV, right ventricle; and cryo, cryoablation.
the sequence of right atrial activation (Figures 2, 3, 4, and 5). The isopotential color maps were referenced to the surface P wave. Positioning of virtual electrograms over the color maps plus correlation with bipolar contact electrograms at marked sites representing the electrodes on the multipolar catheter helped distinguish true depolarization from artifact, far-field potentials, and repolarization. Higher filter settings were used to eliminate repolarization artifact. During global activation, color contrast and offset settings were high to show only depolarization of significant areas of atrial myocardium. As activation was taken back in time from the onset of atrial systole into late diastole, the color offset and contrast settings were brought down close to zero and electrogram amplitudes were increased to look for low amplitude, presystolic, and late diastolic activation. Careful manipulation of offset and contrast settings allowed identification of the earliest site of systolic activation. Lower filter setting were used to determine earliest activation, particularly in virtual electrograms at exit sites from a zone of slow conduction. An initial sharp, negative deflection in the unipolar virtual electrograms (QS complex) suggested a true exit site. Filter settings were applied after the acquisition of raw data and thus could be changed at the press of a button during review.

Once the earliest site of activation and diastolic pathway of slow conduction were determined, the position and margins of the planned linear lesion were labeled onto the geometry. Linear lesions were chosen that bridged the narrowest gap between the anatomic or surgical barriers that enclosed the earliest site of activation and the preceding zone of slow conduction.

In real-time mode, the ablation catheter was then steered to the identified areas by means of the locator signal. Contact electrograms were recorded, local activation times referenced to the surface P wave, and, when possible, pace mapping and entrainment were performed.

**Linear Lesion Creation**

Radiofrequency energy was delivered during tachycardia by a 4-mm-electrode in a point-to-point fashion. Temperatures were limited to 65°C and energy delivery to 70 W. Each radiofrequency lesion position was labeled on the geometry. Arrhythmia termination was an indication that the chosen site was appropriate. The line was continued if tachycardia stopped before the distal margin of the lesion had been reached. Once the line was completed, if tachycardia had terminated, reinduction was attempted. If the arrhythmia was noninducible, bidirectional pacing was attempted. If the arrhythmia was not terminated or was reinduced, further mapping was performed and gaps in the line where color broke through were targeted. If no obvious gap was present, the entire line was reattempted.

The surgical cryotherapy lesions for patient 6 were created with the use of an 11-mm probe (Spembly Ltd). Tip temperature was reduced to −50°C for 90 seconds for each lesion. No further mapping was undertaken to assess linear lesion continuity and conduction block.

Patients were followed up with clinic visits at 1, 3, and 6 months and with regular 24-hour Holter monitoring.

**Results**

Data were collected during atrial tachyarrhythmias in all 6 patients. Apart from the patient with incessant atrial tachycardia, all other arrhythmias were initiated with programmed extrastimuli. In all 6 patients, at least 1 clinical arrhythmia was induced (similar P-wave morphology and cycle length to previously documented tachyarrhythmias). The mean procedure time was 320 (±98) minutes; the mean fluoroscopy time was 59 (±13) minutes. Of 11 different atrial tachyarrhythmias recorded, 7 were thought to be clinically relevant.

**Mapping Procedure**

The noncontact mapping balloon catheter was positioned without difficulty in each case. There were no difficulties with balloon stability or displacement. No part of the atrial chamber was >6 cm from the balloon center. On one occasion when the area of interest could not be mapped with sufficient accuracy and was beyond 4-cm distance from the balloon center, the catheter was repositioned.

Exact boundaries between surgical barriers and myocardium were hard to determine in some instances. Atriotomy incisions were characterized by double potentials in only 2 patients. The remainder had absent or low-amplitude electro-
grams in the anterolateral atrial wall where the scar was presumed to be. Color map activation correlated well with bipolar contact electrograms. Techniques such as pace mapping and entrainment were difficult to perform and unreliable because of intermittent capture, poor catheter-tip stability, and unreliable P-wave morphology. Diseased, inert tissue, typical of the Fontan atrium, limited the number of electrograms that could be recorded from contact catheters.

There were no procedural complications. The noncontact mapping catheter did not cause obstruction to blood flow or perforation of the cardiac chamber. There were no thromboembolic episodes and no bleeding complications arising from anticoagulation.

**Arrhythmia Characteristics**

All arrhythmias were induced and terminated with programmed extrastimuli. Mean tachycardia cycle length was 262 (±58) ms. All arrhythmias were sustained and hemodynamically tolerated. Color mapping demonstrated that in every case the arrhythmia mechanism was macroreentry involving anatomic and surgical barriers forming protected areas of slow conduction. Ten of the 11 induced arrhythmias had reentry circuits that were confined to the right atrium. In 3 patients, the zone of slow conduction was between the lower end of the atriotomy scar and the inferior vena cava or tricuspid valve patch. Patient 4 had a right atrial macroreentrant circuit similar to common atrial flutter (Figure 5). The patient with the conduit from the right atrial appendage to the right ventricle had a circuit that rotated around the conduit anastomosis with slow conduction between the conduit and the inferior vena cava (IVC). The arrhythmia induced in patient 3 was biatrial, with slow conduction occurring across the interatrial baffle. The left atrial portion of the circuit immediately adjacent to the baffle was indirectly visualized as low-amplitude, far-field activity on the color map.

The site of earliest activation was identified in all 11 arrhythmias. A zone of slow conduction could be mapped in its entirety in 4 of 11 arrhythmias and was only partially seen in 7 of 11 arrhythmias.
Radiofrequency Ablation
The mean number of applications was 32 (±21) at a median of 2 sites. Three of the 5 patients had a second or third site selected after repeated attempts were ineffective at creating the initial line of block. Poor catheter tip contact and stability were common during radiofrequency energy applications. Radiofrequency application terminated the clinical tachycardia in 3 of 5 patients. In 2 patients, this was during the one and only linear lesion created. The other 2 patients required multiple lesions. After successful ablation of the clinical arrhythmia, 2 patients had inducible arrhythmias that were judged to be nonclinical.

Cryoablation
A continuous line from the IVC to the conduit and then to the coronary sinus os was created with 8 cryotherapy lesions. After 5 months of follow-up, the patient has been arrhythmia free.

Follow-Up
Immediate procedural success, defined by the inability to reinduce the clinical tachycardia, was achieved in 3 of 5 patients (excluding the surgical patient who did not have further provocative testing). Two patients had inducible arrhythmias that were deemed to be nonclinical. Mean follow-up is 6.4 months. One patient remains in incessant tachycardia and is being considered for surgical conversion to TCPC together with surgical cryoablation and pacemaker implantation. Two of 6 patients remain free of symptoms, with no arrhythmias documented on 24-hour Holter monitoring. Three patients continued to have repeated episodes of tachycardia. In 1 patient, this has not been recorded on an ECG. In the other 2 patients, ECG recordings confirmed recurrence of their original clinical tachycardia. The 3 patients with recurrent tachycardias have subsequently undergone conversion surgery to TCPC, together with surgical cryoablation and pacemaker implantation.

Discussion
This study reports the use of noncontact mapping in the investigation and treatment of atrial arrhythmias that occur after the Fontan operation. Clinically relevant arrhythmia circuits with their corresponding anatomic boundaries were demonstrated in every patient. The locator signal allowed accurate positioning of the ablation catheter and guided linear lesion creation. Radiofrequency energy application terminated the arrhythmia in 3 of 5 patients, but 2 patients had early recurrence. The patient who underwent surgical cryoablation has had no further arrhythmias.

Previous published series represent a heterogeneous group of congenital heart conditions, with only a small proportion having undergone Fontan surgery. Initial studies targeted sites with early activation and fractionated electrograms. Focal rather than linear radiofrequency lesions were created. Subsequently, other series adopted a more anatomic approach. Regions of previous surgical intervention were sought with the use of operative reports and contact electrogram characteristics. Pacing techniques, such as entrainment, were then attempted. All these series indicate that Fontan patients respond less well to mapping and ablation than patients who have undergone other forms of congenital heart disease surgery. Long-term results are poor.

These studies may have failed because of the limitations of contact mapping with a small number of electrodes in an anatomically complex substrate. Electrogram recording requires good catheter contact, which may be difficult in large

Figure 5. Activation sequence of 1 cycle of IART in patient 4: Open view of right atrium is displayed. Chamber has been “cut” along posterolateral wall and peeled open to display entire endocardial surface. Center image is reference frame with anatomic structures labeled for orientation purposes. Negative potentials on endocardial surface (depolarization) are manifest as colors superimposed on purple (isoelectric or positive potentials) background. Low-amplitude, fractionated, slow conduction spreads down anterolateral wall, beginning 170 ms before P-wave onset. Conduction spreads around the lateral margin of tricuspid valve patch and across tricuspid valve–IVC isthmus before heading up septum, posterior to coronary sinus os. More global, higher-amplitude activity then occurs at P-wave onset. For this patient, linear lesion was created in tricuspid valve–IVC isthmus. For an animated version of this figure, please refer to the journal’s Web site (www.circulationaha.org).
chambers. We noted that only a minority of electrodes on our multipolar catheters were in contact with electrically active myocardium. Conventional techniques are unable to locate and record catheter position and thus cannot allow reconstruction of a 3D picture of atrial anatomy. Noncontact mapping allows precise localization of the ablation catheter tip with reference to a 3D reconstruction of chamber geometry. Detection of anatomic and surgical margins is limited when using electrogram morphology and pacing techniques alone. We discovered fractionated electrograms in areas of myocardium that had not been subjected to surgical alteration and were not critical components of the reentrant circuit. In our patients, atriotomy scars were not always associated with double potentials and could manifest as low-amplitude, electrically inert areas with no distinct borders. Additionally, we found that pacing techniques were limited by intermittent capture and poor catheter tip stability.

Electroanatomic mapping of intra-atrial tachycardia after Fontan surgery with the CARTO system (Biosense Ltd) has addressed some of the limitations of conventional mapping. A 3D reconstruction of atrial geometry was created; however, mapping was still dependent on contact electrograms. A voltage map assisted in the identification of nonexcitable tissues. Concealed entrainment was not helpful because it is performed over a larger area of atrial tissue, necessitating an anatomic approach to ablation. Noncontact mapping provides a 3D reconstruction of atrial geometry, instant mapping of entire endocardial activation without the need for catheter contact, reconstruction of unipolar electrograms at any site on the endocardial surface, the ability to map multiple arrhythmias without rebuilding chamber geometry, and a locator signal that can be applied to any conventional catheter to determine its position. All of these features contributed to successful mapping of intra-atrial reentry tachycardia (IART) in our patients.

Despite the use of noncontact mapping to characterize atrial tachyarrhythmias and identify sites suitable for ablation, our intermediate follow-up results are similar to previous studies that used conventional techniques. We believe that noncontact mapping accurately documented the arrhythmia circuits because activation displayed as isopotential color maps correlated well with contact electrograms. The tachycardia circuit was initially broken with radiofrequency ablation in the majority of cases only to return at a later date, suggesting that the selected site was appropriate. Where pace mapping and entrainment could be performed, they confirmed that selected sites were critical to the reentry circuit. By using the locator signal, we were able to extend the linear lesions up to and beyond the anatomic boundaries. We believe that procedural failure was due to inadequate lesion creation. In the dilated Fontan atria, catheter tip stability was often poor and together with the characteristic hypertrophied and fibrotic atrial musculature may have prevented full-thickness lesions. Furthermore, the capacious right atria with slow, stagnant blood flow may have increased heat loss into the surrounding blood pool. Our patients, with very dilated, scarred atria and longer follow-up periods (6 to 10 years since their most recent surgery), when compared with other studies, offered a particularly challenging substrate. In 3 patients who have subsequently undergone surgical conversion to TCPC, examination of the endocardial surface revealed no macroscopic or histological evidence of radiofrequency lesions despite their having received numerous applications.

The high rate of arrhythmia recurrence after ablation indicates that additional technology is required to ensure adequate lesion depth and continuity in the thickened Fontan atrium. Large-tipped ablation catheters and irrigated-tip technology may provide that step. Intracardiac echocardiography may aid identification of surgical and anatomic structures. However, none of these techniques alter the arrhythmia substrate, and new arrhythmia circuits may emerge. For selected patients with exhausted right atria, a surgical approach involving conversion to TCPC accompanied by cryoablation offers patients both hemodynamic improvement and prevention of arrhythmias. Surgical ablation has the added advantage of direct visualization of cardiac structures and boundaries, ensuring that linear lesions extend to their anatomic margins. It may also assess whether lesions are transmural. Preoperative mapping is still helpful to determine cryoablation position, and noncontact mapping is the most appropriate technique.

Study Limitations
A concern regarding dilated chambers is the accuracy at sites >4.0 cm from the balloon center. Although electrogram morphology may become less accurate, the location of endocardial potentials is not affected. Loss of accuracy is gradual, so that timing differences between adjacent areas of myocardium are minimal. In common with previous studies, identification of anatomic landmarks may have been inaccurate despite the use of contact and noncontact electrogram characteristics. When creating linear lesions, we deliberately extended the line over our marked boundaries until we were sure that we had crossed the margin.

The continuity of linear lesions was principally assessed by termination of the arrhythmia during radiofrequency application and noninducibility. We found bidirectional pacing not to be helpful. When pacing was attempted at sites adjacent to the linear lesion, noncontact mapping was inaccurate because pacing artifact often swamped subsequent activation. The complex anatomy prohibited pacing from distant sites. It has been demonstrated that noncontact mapping may successfully assess lesion integrity in common atrial flutter. More recent software has pace-blanking facilities, and we now believe that bidirectional pacing should be used to assess lesion continuity in complex arrhythmias. We did not perform bidirectional pacing with contact catheters to record activation times, which may have contributed to procedural failure and arrhythmia recurrence.

Conclusions
Complex arrhythmias require high-density, multisite mapping techniques, such as noncontact mapping, which provide accurate 3D maps of arrhythmia circuits in structurally abnormal atria. The mapping and radiofrequency ablation of atrial arrhythmias in Fontan patients should be considered for those in whom surgical ablation is unwarranted. Noncontact mapping is effective at determining arrhythmia circuits, but
advanced ablation technologies are almost certainly required for adequate lesion formation.

Acknowledgments
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References
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