Catheter Ablation of Multiple Unstable Macro-reentrant Tachycardia within the Right Atrium Free Wall in Patients without Previous Cardiac Surgery

Short title: Satomi, Right atrial free wall isolation for unstable AT


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Abstract

Background: Macroreentrant atrial tachycardia (AT) involving the right atrium free wall (RAFW) has been reported in patients without atriotomy. Catheter ablation (CA) of these ATs remains challenging due to the multiple morphologies of ATs with unstable reentrant circuits in some patients. The purpose of this study was to clarify the electrophysiological characteristics of these ATs and attempt the novel approach for CA.

Methods and Results: Electrophysiological study and CA were performed in 17 patients (14 men, 71 (67; 76) years) with reentrant ATs originating from the RAFW using 3-D mapping. All patients had no history of cardiac surgery. Clinical ATs with stable cycle length (CL) and atrial activation were identified in 11 patients (group A). All ATs were successfully ablated. In the remaining 6 patients, clinical tachycardia continuously changed with a different CL and P wave morphology and atrial activation sequence during mapping or entrainment study (group B). A complete isolation of the RAFW was attempted in Group B. After complete isolation was achieved in 5 of 6 patients, ATs were not induced in these 6 patients. The number of previous failed CAs and induced ATs were higher in group B than in group A. During 31 (19; 37) months of follow-up, AT recurrence developed in 27 % patients from group A and 33 % from group B.

Conclusions: Multiple and unstable macroreentrant ATs from the RAFW can occur in patients without a history of cardiac surgery. The RAFW isolation has the potential to abolish all ATs.

Key Words: Atrial tachycardia, Catheter Ablation, Clinical Electrophysiology, Mapping
It has been reported that macroleeentrant atrial tachycardia (Macro-AT) in the right atrium (RA) frequently occurs late after surgical repair of congenital heart diseases.\textsuperscript{1-3} Three-D mapping has been shown to facilitate understanding of the AT mechanism and identification of the critical isthmus of the Macro-AT.\textsuperscript{4} Recent studies have shown that RA free wall (RAFW) Macro-AT can also occur in patients without congenital heart disease and previous cardiac surgery and can be successfully ablated guided by 3-D mapping in patients with stable AT. \textsuperscript{5,6} However, in some patients multiple ATs can occur with variable tachycardia cycle length (CL) and different activation sequence. This can make the identification of the critical isthmus of the tachycardia very difficult and may result in ablation failure. In this manuscript we report the RA Macro-AT in patients without previous cardiac surgery and describe an ablation strategy in patients with stable and unstable ATs.

**Methods**

The study included 17 patients with RAFW Macro-AT who underwent catheter ablation. Fifty patients with RAFW Macro-AT who underwent catheter ablation at our department form January 1999 to July 2006. The 33 patients with previous cardiac surgery and the patients undergoing linear lesion in the RA after catheter ablation of atrial fibrillation were excluded from this study. This study was approved by the institutional ethical committee.

**Electrophysiological study**
After written informed consent was obtained, patients underwent electrophysiological study in the fasting state under deep sedation with intravenous propofol, midazolam and fentanyl. Antiarrhythmic medications were discontinued at least 5 half-lives before the procedure in all but 6 patients who were taking amiodarone. Multiple catheters were advanced into the RA via the femoral and subclavian veins and were positioned as follows: (1) a decapolar catheter within the coronary sinus (CS) via the left subclavian vein; (2) a 20-pole catheter around the tricuspid annulus to record the atrial activation from the RAFW; (3) a multi-polar catheter at the His-bundle region; and (4) a 7-F mapping catheter (Biosense-Webster, Diamond Barr, CA, USA) for mapping and ablation. If the patient was in sinus rhythm (SR) at the beginning of the procedure, it was attempted to induce the arrhythmia by programmed stimulation or burst atrial pacing. Intravenous isoproterenol was administered if the tachycardia was not induced. Group A was defined as a spontaneous or induced AT with a stable CL; group B was defined as induced or spontaneous tachycardia that converted easily into a different AT during mapping and/or entrainment mapping or spontaneous conversion into a different tachycardia within less than 2 minutes. This situation makes the identification of the critical isthmus very difficult.

**Electroanatomical mapping**

All patients underwent 3-D mapping. The activation map and the bipolar voltage map of the RA were used either during tachycardia or sinus rhythm. The 3-D RA geometry is reconstructed relative to the stable atrial activation recording from the CS. Mapping was complete when all regions of the atrium had been systematically sampled.
and a sufficient density of points (>150 points) had been acquired. In patients with sustained stable AT, mapping was performed during tachycardia. In patients with unstable circuits, only bipolar voltage maps were created. Electrical silence was defined as the absence of recordable atrial activity on bipolar voltage amplitude. These points are labeled as gray color on the 3-D maps (area of electrical silence; AES). Low-voltage regions were defined by the presence of a bipolar voltage <0.15 mV and appear red on the voltage maps. 7

The presence of Macro-AT was confirmed using electroanatomical mapping or entrainment study. Entrainment at multiple sites was performed to identify the reentrant circuit in all cases. Pacing sites with a post-pacing interval (PPI) that did not exceed the cycle length by 20 ms were considered within the reentrant circuit. The site with mid diastolic potential within reentrant circuit was defined as the potential critical slow conduction zones.

**Catheter Ablation**

Irrigated RF energy was delivered as previously described using a target temperature of 43°C, a maximal power of 40 Watts, and an infusion rate of 17 ml/min in all patients. The RF power was initially 30 Watts and titrated up to 40 Watts. In group A, RF ablation was initially performed in the tachycardia isthmus and subsequently in the cavitricuspid isthmus (CTI) in all patients.

In group B, in order to avoid injury to the sinus node or damage to pacemaker leads, the location of the sinus node was identified based on activation in sinus rhythm and marked on the 3-D map, and pacemaker leads were avoided using the fluoroscopic...
image. Additionally, the sites with phrenic nerve capture were tagged on the CARTO mapping. After complete mapping of the RA, an ablation site was initially chosen at the potential critical slow conduction zone, indicating the border zone with low amplitude and the area with fractionated/double potential during SR or tachycardia. Subsequently, if the AES extended toward the crista terminalis, each linear lesions was created at the border between two AES or an AES and an anatomic obstacle, such as the tricuspid annulus, inferior vena cava (IVC), or superior vena cava (SVC) (complex lesions). If AES was limited in the RA free wall, circumferential lesion was applied at the RA free wall anatomically including AES (circumferential lesion). (Figure 1) The end point of the ablation procedure was no induced AT after ablation or complete isolation of the RAFW, indicated by the disappearance of atrial activation recorded from the distal electrodes of the 20-polar catheter facing RAFW during SR or atrial pacing.

**Postablation Management**

All patients were maintained on anticoagulation and discharged under oral anticoagulation with warfarin for 3 months. All antiarrhythmic drugs including amiodarone were discontinued at the hospital discharge. Follow-up was obtained from the referring physician or our outpatient clinic.

**Statistical Analysis**

Continuous variables are summarized by median and quartile 1 and quartile 3 (Q₁; Q₃). Categorical variables are represented by absolute values and percentages. The probability of recurrent AT at 12, 24 and 36 months was estimated using the Kaplan-Meier method.
Results

Patient characteristics

Seventeen consecutive patients without cardiac surgery had an AT located within the right atrial free wall and underwent catheter ablation. Stable ATs were mapped in 11 patients (group A). All critical isthmuses were identified using 3-D mapping and were successfully ablated with irrigated energy. In the remaining 6 patients (group B), clinical ATs easily converted into a different AT with a different CL (Figure 2) and P wave morphology during mapping or pacing/entrainment (Figure 3). The ATs were easily induced by programmed stimulation, but reproducible induction of the AT with the same P wave morphology and CL was difficult due to the inducibility of multiple tachycardias.

Clinical characteristics are shown in Table 1. Structurally normal hearts were found in 12 of the 17 patients. Coronary artery disease with normal left ventricular ejection fraction was diagnosed in the remaining five patients. Five patients had sinus node dysfunction. In 4 out of these 5 patients a pacemaker had been implanted. Number of failed anti-arrhythmic agents was 1 in 11 patients, 2 in 5 patients and 3 in 1 patient, including amiodarone in 6 patients. Previous ablation procedures of RAFW macro-AT had failed in 10 patients. Also, CTI was ablated in 5 patients.

Mapping and ablation of the AT in group A

In group A (11 patients), there were 9 spontaneous ATs and 6 induced ATs during the procedure. (Table 2) Three-D mapping was performed for the 1 ATs in the 8 patients, 2 ATs in 2 and 3 ATs in 1. Voltage mapping showed that a large area with low amplitude and fractionated/double potentials was found in the RAFW during tachycardia. The
critical isthmuses of the 15 ATs were found in the RAFW within the area with low amplitude in all 11 patients (figure 4). The isthmuses were located in the lateral free wall in 4, in the inferior wall (close to IVC) in 2, and in the posterior free wall (close to crista terminalis) in 6 patients. A median of 8 (5; 17) RF applications was delivered at the isthmuses, resulting in termination of all ATs and non-inducibility after the ablation. Long pauses or sinus bradycardia were not observed after tachycardia termination during or after RF ablation.

**Mapping and ablation of ATs in group B**

In group B (6 patients), there were 6 spontaneous ATs and 20 induced ATs during the procedure (table 2). Number of spontaneous or inducible AT in each patient was 3 in 1 patients, 4 in 3 patients, 5 and 6 in 2 patients. Fractionated electrograms, double potentials or isolated delayed components with low amplitude were recorded at the RAFW (Figure 5). Initially it was attempted to map the reentrant circuits of 1 spontaneous ATs in all 6 patients and additional 1 inducible AT in 1 patient. Voltage mapping showed that an area with low amplitude was consistently located in the RAFW. During mapping of the clinical tachycardia, circuits were converted constantly into other ATs by manipulation of the mapping catheter or entrainment pacing to identify whether the diastolic potentials in the RAFW were critical for the tachycardia circuits. In consequence, a linear lesion with 7 (4; 12) applications was delivered from the AES to an anatomical obstacle using CARTO during tachycardia (Figure 5) resulting in AT termination. After AT termination, ATs with different P wave morphology and different CL were still induced. Remapping of the RA was performed during SR. The isolation of R8
the RFFW was finally attempted during SR. In the first 3 patients, 3 linear ablation lines connected the AES to anatomical obstacles: one line connected the AES to the tricuspid annulus, one line connected the AES to the IVC, and a third line created cavotricuspid isthmus block if this was had not been done at a previous procedure (“complex lesions” pattern). In the other 3 patients, a long encircling lesion around the area with low amplitude was created (“circumferential lesion” pattern). (Figure 1)

In 5 out of the 6 patients, a mean of 17 (15; 19) RF applications were delivered and resulted in complete isolation of the RAFW (Figure 6). No ATs were induced by programmed stimulation or burst pacing with and without intravenous administration of isoproterenol after the isolation of RAFW. In one patient, isolation of the RAFW was not achieved even after 22 RF applications with irrigated RF energy. In this patient, AT was not inducible and the RAFW was activated via a conduction gap at the base of the RA appendage area close to the tricuspid annulus. In one out of the 6 patients, RF delivery at superior lateral RAFW resulted in a long sinus pause of 5.6 seconds, but stable sinus rhythm with a normal heart rate of 72 bpm recovered immediately after termination of RF energy.

**Clinical parameters in the two groups**

There was no difference between the groups in age, gender, structural heart diseases and fluoroscopy time. Previous catheter ablation had failed more frequently in group B (6 of 6; 100%) than in group A (4 of 13; 36%). The median number of spontaneous or inducible ATs before ablation was 1 (1; 2) in group A and 4 (4; 5) in group B. The average cycle length of ATs was 320 (293; 364) ms in group A and 375
(348; 440) ms in group B. The duration of the procedure was longer in group B: 350 (300; 355) min than group A: 240 (100; 285) min.

**Procedure data and follow-up**

No long pauses were observed on 24 hour holter after ablation in the 13 patients without pacemaker implantation. Right phrenic nerve injury was not observed during the ablation. During 31 (19; 37) months of follow-up without use of antiarrhythmic drugs (Group A: 31 (20; 36) months and Group B: 27 (12; 38) months), the recurrent ATs was observed 3 of 11 (27.3 %) in group A and 2 of 6 (33.3%) in group B. (Figure 7). The estimated probability of recurrent AT at 12, 24 and 36 months following an ablation procedure were 9%, 9% and 20% in group A and 20%, 20% and 40% in group B.

**Discussion**

This manuscript describes in a large series of patients without cardiac surgery: (1) the clinical and electrophysiological characteristics of Macro-AT; (2) the catheter ablation guided by 3-D mapping of stable and unstable Macro-AT from the RAFW; (3) the isolation of the RAFW, which has the potential to abolish all unstable ATs.

**Macro-AT without atriotomy**

Macro-ATs in the RA are common late after surgical repair of congenital heart disease. Three-D electroanatomical mapping demonstrates that the critical tachycardia isthmus is frequently constrained between the atriotomy and natural anatomic obstacles. Catheter ablation of these tachycardias using electroanatomical mapping has a relatively
high success rate. In the present study of 17 cases, AES and the area with low amplitude and fractionated/delayed potentials during tachycardia or SR were located in the RAFW. No patients had previous history of cardiac surgery. The clinical features in our patients were similar to the previous publication in which Stevenson et al. reported that RA Macro-AT is associated with AES in patients without prior atrial surgery. In their report, there is a very high incidence of sinus node dysfunction indicating extensive RA pathology. This finding was also consistent with our finding that a pacemaker had been implanted in 4 out of 17 patients before the ablation. In these 17 patients, the pathological equivalent of low voltage amplitude and areas of electrical silence in the RAFW may be a “local RA cardiomyopathy”.

Clinical and electrophysiological characteristics of stable and unstable Macro-AT in patients without cardiac surgery

In this report, there were 11 patients with stable and 6 patients with unstable tachycardia due to spontaneous and/or pace-induced conversion of the tachycardia into multiple other ATs. In the 11 patients with stable AT, clinical ATs could be easily and reproducibly induced by programmed stimulation or burst pacing, suggesting reentry as a mechanism of AT. Activation mapping showed a large area with low amplitude and fractionated or/and delayed components in the RAFW during tachycardia and entrainment mapping showed a critical isthmus. This provides an anatomical substrate for Macro-AT. In the 6 patients of group B there were 26 ATs with a different P wave, tachycardia CL and atrial activation. Mapping demonstrated multiple AESs and a large area with low amplitude and fractionated and late potentials. This provides multiple
reentry circuits for macro-ATs with exit between several isolated channels or natural anatomical barriers. This substrate provides multiple reentry circuits with different exits for macro-ATs. Catheter manipulation and entrainment mapping frequently resulted in a change into a different AT, which made it difficult to identify the reentrant circuit. Electrophysiologically, this is similar to catheter ablation of complex reentrant ATs in patients after Fontan procedure for congenital heart disease. These patients without atriotomy had frequent failed CA and multiple ATs during the procedure, and the complex substrate with multiple AES and a large area with low amplitude in the RAFW made CA in these unstable ATs more complex and challenging.

**Isolation of the RAFW as an alternative approach in multiple unstable ATs**

Catheter ablation of multiple and unstable ATs is still challenging even with the use of 3-D mapping. Nakagawa et al recently demonstrated that elimination of all channels within the tachycardia substrate can abolish all multiple and unstable ATs in patients after surgical repair of congenital heart disease. Using this strategy, high-density mapping is required to identify the area with electrical silence and a line of double potentials during stable SR or atrial pacing. Also, identification of scar as an area without capture is required. Therefore, this strategy is time-consuming and it is difficult to eliminate all regions with abnormal electrograms in some patients. In this report, we performed complete isolation of the RAFW, including all potential channels between both AESs and between AES and the native anatomical barrier such as tricuspid annulus, IVC and SVC. Complete isolation was achieved in 5 of 6 patients, and no tachycardias were inducible in these patients. This is a similar strategy for ablation of unstable
ventricular tachycardia (VT) based on substrate mapping or surgical isolation of VT foci. To our knowledge, this is the first report that demonstrates the feasibility of the isolation of the RAFW to abolish all multiple and unstable ATs.

The other important finding was that there was no difference in AT recurrence in the two groups after a successful ablation despite more complicated ATs and more RF lesions in the patients who underwent RAFW isolation. This may be due to careful mapping and more clear evidence of eliminating the tachycardia substrate. Also, this approach needs longer procedure duration although the 3-D mapping can reduce the fluoroscopic time. Injury to the sinus node or the phrenic nerve may theoretically occur during isolation of the wide area of RAFW. In one patient, a long pause occurred during ablation at the RA. However, there was no evidence of sinus node injury on 24-holter ECG and during following-up. Such complications did not occur in our series. This may be due to our ablation strategy. First, we performed detailed mapping of the sinus node and marked the phrenic nerve course on the CARTO map by pacing maneuvers. Second, the pathological area with low amplitude and abnormal atrial activation was located anterior to the sinus node in these 6 patients. Based on these findings, isolation of the RAFW can become an alternative approach for multiple and unstable ATs within the RAFW.

Limitations

There are several limitations. First, pacing at the AES was not performed in all patients. It was impossible to differentiate baseline noise from potentials with very low amplitude within the AES. Therefore, some site with the AES may not present as an...
acquired pathological scar. Second, concealed entrainment mapping was not systematically performed in the 6 patients with multiple and unstable ATs due to continuous transformation into a different ATs. Third, complete RAFW isolation was not achieved in 1 patient because ATs could not be induced after extensive ablation most likely due to a thick and trabeculated myocardium at the base of RA appendage area close to tricuspid annulus. However, no AT was induced after the ablation. Fourth, pacemakers had been implanted in 4 of these patients before ablation; therefore dysfunction of the sinus node could not be assessed in these 4 patients. Finally, multiple and unstable AT can occur in the patients after the repair of congenital heart disease in the clinical practice, we have not included this patients due to very limited number of these patients. This approach can theoretically be used in the patients with multiple and unstable AT after the repair of congenital heart disease.

**Conclusions**

Complex (multiple and unstable) macroreentrant tachycardias were observed in patients without a surgical incision in the RAFW. It is crucial to ablate all channels of tachycardias. The compartmentalization of RAFW using electroanatomical mapping is an acceptable technique for eliminating such atrial tachycardias.
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Disclosure: None
References


Table 1: Patients characteristics

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<th>Group B</th>
</tr>
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<tr>
<td></td>
<td>N=11</td>
<td>N=6</td>
</tr>
<tr>
<td>Age, (yr), median (Q1;Q3)</td>
<td>58 (52;67)</td>
<td>71 (67;76)</td>
</tr>
<tr>
<td>Gender, (m/f)</td>
<td>8/3</td>
<td>6/0</td>
</tr>
<tr>
<td>Structural heart disease, (n)</td>
<td>2 (18%)</td>
<td>3 (50%)</td>
</tr>
<tr>
<td>Coronary heart disease, (n)</td>
<td>2 (18%)</td>
<td>3 (50%)</td>
</tr>
<tr>
<td>SSS, (n)</td>
<td>2 (18%)</td>
<td>3 (50%)</td>
</tr>
<tr>
<td>Number of Pts with failed AAD, (n)</td>
<td>8</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Prior CA</td>
<td>4 (36%)</td>
<td>6 (100%)</td>
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</table>

SSS = sick sinus syndrome, Pts = patients, AAD = anti-arrhythmia drugs, CA = catheter ablation.
Table 2: Electrophysiological Result

<table>
<thead>
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<th>Group A</th>
<th>Group B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N=11</td>
<td>N=6</td>
</tr>
<tr>
<td>No of AT, (n), median (Q1;Q3)</td>
<td>1 (1; 2)</td>
<td>4 (4; 5)</td>
</tr>
<tr>
<td>No of Pts with AT, (n)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 AT</td>
<td>8</td>
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</tr>
<tr>
<td>2 ATs</td>
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<tr>
<td>3 ATs</td>
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<td>1</td>
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<tr>
<td>4 ATs</td>
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<td>3</td>
</tr>
<tr>
<td>5 ATs</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>6 ATs</td>
<td>-</td>
<td>1</td>
</tr>
<tr>
<td>CL of AT, (ms), median (Q1;Q3)</td>
<td>320 (293;364)</td>
<td>375 (348;440)</td>
</tr>
<tr>
<td>Number of total applications, (n), median (Q1;Q3)</td>
<td>8 (5;17)</td>
<td>26 (19;37)</td>
</tr>
<tr>
<td>Duration of procedure, (min), median (Q1;Q3)</td>
<td>240 (100;285)</td>
<td>350 (300;355)</td>
</tr>
<tr>
<td>Fluoroscopy time, (min), median (Q1;Q3)</td>
<td>28 (18;31)</td>
<td>37 (22;51)</td>
</tr>
<tr>
<td>Recurrence during follow-up, (n)</td>
<td>3 (27%)</td>
<td>2 (33%)</td>
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AT = atrial tachycardia, Pts = patients, CL = Cycle length,
Figure legends

Figure 1
Schematic representation of ablation lines at the right atrium free wall.
If the area of electrical silence (AES) area extended toward the crista terminalis, a linear lesion between the superior region of AES and the tricuspid annulus, a linear lesion between the inferior region of the AES and the IVC and a cavo-tricuspid isthmus line were created to close the RA free wall (“complex lesions” pattern). If AES area was limited to the RA free wall, RF applications were delivered in a circular pattern at the RA free wall including low potential areas or identifiable isthmuses of tachycardias according to the electroanatomical map (“Circumferential lesion” pattern).
SVC = superior vena cava, IVC = inferior vena cava, TA = tricuspid annulus, RAO = right anterior oblique

Figure 2
Twelve lead ECG of pleomorphic ATs
Six types of ATs were induced in one patient in group B. All ATs had different cycle lengths.

Figure 3
Twelve lead ECG of unstable AT
Morphology of P wave and CL of AT were changing during mapping in a patient in Group B

Figure 4
Twelve lead ECG and activation map during tachycardia

R20
Electroanatomical activation mapping during an atrial tachycardia (AT) in a patient in group A demonstrated the exit at the superior part of the area of electrical silence (AES) at the RA free wall and the entrance at the inferior part of it. This tachycardia was terminated by applications at the exit region. The duration obtained by the activation map covered only 56% of AT cycle length (189ms of 340ms). That indicated that most part of the slow conduction zone was located in the AES.

Figure 5
Voltage map and RF application in the patients with AT with no identified isthmus

The electroanatomical voltage maps of RA during sinus rhythm showed the area of electrical silence (AES) at the RA free wall, indicated by a gray area. Note multiple scattered AES located on RA free wall, extending from the tricuspid annulus to the crista terminalis in left panel. Right panel shows the voltage map of another patient with extensive AES at the RA free wall. Low voltage areas were demonstrated around the AES in both patients. Two ablation lines were created to connect the free wall AES and anatomical obstacles (“complex lesions” pattern), such as AES to tricuspid annulus and scar to IVC. This patient had prior cavo-tricuspid isthmus block. (Left panel) An encircling lesion was attempted around the free wall AES (“circumferential lesion” pattern) (Right panel). Red dots indicated the point of applications.

Figure 6
Intracardiac recording at the time of right atrium free wall isolation

The electrogram recorded by 20-polar catheter showed sudden elimination of signal (star) during radiofrequency application at the right atrium free wall region, indicating the electrical isolation of RA
free wall. Note low amplitude before isolation on almost all right atrial free wall bipoles suggestive of scar.

RA = right atrial free wall, CS = coronary sinus, MAPp = mapping/ablation catheter proximal, MAPd = mapping/ablation catheter distal

Figure 7

Flow chart showing the result of ablation and follow-up.

See text for details.

AT=atrial tachycardia, RA = right atrium
Complex lesions

Circumferential lesion
Right atrial macro-reentrant AT without an atriotomy

Group A (N=11)
9 spontaneous
6 induced

Isthmus identified AT 15

AT isthmus ablation
Inducible AT 0
Recurrence AT N=3/11 (27%)

Group B (N=6)
6 spontaneous
20 induced

Isthmus identified AT 7

AT isthmus ablation
No isthmus identified AT 19
RA isolation
Inducible AT 0
Recurrence AT N=2/6 (33%)

31 (19; 37) months of follow-up
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