Electrophysiological Features of Atrial Tachyarrhythmias After the Creation of a Left Atrial Roof Line During Catheter Ablation of Atrial Fibrillation

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Summary

Left atrial roof line (LARL) can prevent the perpetuation of atrial fibrillation (AF) by delineation of the arrhythmogenic substrate, but it may be associated with an increased incidence of atrial tachycardia (AT). This study was performed to evaluate the characteristics and clinical implications of inducible AT after LARL.

A total of 139 consecutive patients with AF who underwent catheter ablation were prospectively enrolled in this study. LARL was required to prevent the perpetuation of AF in 98 of 139 patients (71%). LARL significantly reduced the incidence of inducible AF (before versus after: 100% versus 44%, respectively, \( P < 0.01 \)), whereas it significantly increased the incidence of AT (18% versus 63%, \( P < 0.01 \)). ATs were observed after LARL in 62 of 98 patients (63%), and these circuits were determined in 99 of 112 stable ATs (88%), including tricuspid isthmus-dependent \( (n = 35) \), mitral annulus \( (n = 22) \), septal \( (n = 15) \), surrounding right pulmonary veins (PVs) \( (n = 12) \), coronary sinus (CS) ostium \( (n = 4) \), upper loop \( (n = 4) \), surrounding left PVs \( (n = 4) \), and LA anterior wall \( (n = 3) \). Catheter ablation (CA) successfully terminated 111 of 122 stable ATs (91%) during CA. The occurrence of AT after CA was significantly higher in patients with than in those without residual AT (26% versus 2%, \( P < 0.05 \)).

Induced AT with a stable circuit after LARL creation could be mapped, and delineation of the induced AT may lead to a favorable outcome. (Int Heart J 2011; 52: 92-97)

Key words: Ablation, Atrial tachycardia, Atrial Fibrillation, Mapping, Left atrium, Roof line

Methods

Study population: The study population consisted of 139 consecutive patients with drug-refractory episodes of AF who underwent initial radiofrequency CA (Table I). The mean age of the patients was 62 years, 82 (57%) were male, and 41 (29%) had persistent AF lasting more than 7 days (long-standing AF more than 6 months; 15 (11%)). Patients were considered for ablation based on the presence of symptomatic AF resistant to \( \geq 1 \) antiarrhythmic drug. Cardiac structural findings assessed by ultrasonography are shown in Table II. No specific exclusion criteria were used. All antiarrhythmic agents were generally discontinued for at least 3 days before CA. Amiodarone was withdrawn at least 2 months before the procedure. All patients provided written informed consent for the electrophysiological study. This study was approved by our institutional review committee and the subjects provided informed consent.

Electrophysiological study and catheter ablation: Transesophageal echocardiography was performed to exclude any LA thrombi. A 10-pole or 20-pole diagnostic catheter was positioned in the coronary sinus (CS) for pacing and recording. The 20-pole catheter was located in the right atrium to cover...
the area of the tricuspid annulus or superior vena cava (SVC). The LA and PVs were accessed by a transseptal approach. We introduced 3 steerable catheters, including 2 spiral curve catheters, into the LA through a single transseptal puncture site. The PVs were mapped with a circumferential 10-pole or 20-pole catheter (IBI, Irvine, CA). The surface ECG and intracardiac electrograms filtered at 30 to 500 Hz were recorded simultaneously with a polygraph (DUO EP Laboratory; Bard Electrophysiology, Lowell, MA). A single bolus of 150 IU/kg of heparin was administered after the transseptal puncture and repeated to maintain an activated clotting time of > 300 seconds. Isoproterenol was continuously maintained during CA and burst pacing induction protocol (1-2 μg/minute).

We initially performed the PV isolation procedure using a double circular mapping catheter technique. The RF energy was principally applied during sinus rhythm. In the patients with AF persistency, direct cardioversion was initially attempted to restore sinus rhythm. The locations of PV ostia and carina were determined by left pulmonary venography with injection of contrast medium via the two long sheaths. The RF energy was circumferentially applied to isolate PV potentials and we confirmed the success of individual electrical PV isolation by monitoring the electrical isolation at the antrum level: approximately 1 cm from the ostium of both the right and left PVs. The complete disappearance of the potentials from all 4 PVs and antrum area inside the created line was confirmed in all patients. Left PV isolation was initially accomplished, followed by right PV isolation.

After the PV isolation procedure, atrial tachyarrhythmias were immediately induced by intense burst pacing. Atrial burst pacing was performed (10-s bursts) in decrements from 250 ms down to refractoriness at the maximum output or 150 ms from at least 3 sites including the distal CS, left atrial appendage, and right atrium. Inducible AF was defined as AF sustained for ≥ 1 minute. When the AF spontaneously terminated, induction was attempted 3 times from each of these sites. In cases showing induction of burst-inducible AF after the PV isolation procedure, an additional roof line was created.

To create the LARL, the ablation catheter was introduced through a long sheath to achieve stability and allow orientation of the catheter tip toward the LA roof. Radiofrequency energy was applied during left atrial appendage pacing, and we observed the local potential during CA. The electrophysiological endpoint of the ablation was the demonstration of a complete line of block joining the 2 superior PVs. Complete linear block was defined by the demonstration of double potentials along the entire LA roof. The second potential was then measured at the posterior LA close to the ablation line, which confirmed a detoured activation circumventing the right and left PVs, activating the posterior wall caudocranially with no conduction through the LA roof (Figure 1). In cases in which it was difficult to determine double potentials along the entire LA roof because of dull or poor potentials suspected to be caused by far-field potential due to scar creation, we found remarkable conduction differences between anterior and posterior sides of the LARL during LAA pacing and confirmed a remarkable time delay of more than 100 ms from LAA stimulus spike to the potential of the posterior wall side of the LARL.

After LARL creation, the same induction protocol was

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<th>Table I. Clinical Characteristics</th>
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<td>Number of patients</td>
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<td>Female (%)</td>
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<td>Persistent AF (%)</td>
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<td>SHD (%)</td>
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<td>Mean AF duration (months)</td>
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<td>Number of AADs</td>
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SHD indicates structural heart disease; AADs, anti-arrhythmic drugs; and LVEF, left ventricular ejection fraction. The AADs were classified by the Vaughan-Williams scale. AF duration is expressed as the period from AF onset to the ablation procedure.

<table>
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<th>Table II. Cardiac Structural Parameters</th>
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<td>LA diameter (mm)</td>
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<td>A-P</td>
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<tr>
<td>S-L</td>
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<td>MV-PV</td>
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<td>PV diameter (mm)</td>
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<td>LVEF (%)</td>
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LA indicates left atrium; A-P, antero-posterior; S-L, septal-lateral; MV-PV, mitral valve-pulmonary vein; LSPV, left superior pulmonary vein; LIPV, left inferior pulmonary vein; RSPV, right superior pulmonary vein; and RIPV, right inferior pulmonary vein.

Figure 1. Confirmation of complete block along the LARL. Elimination of the local potentials or formation of double potentials during left atrial appendage (LAA) pacing. (a) shows the double potentials with flat segment recorded on the LA roof. Conduction from LAA to the potential of the LA posterior wall side was remarkably delayed (169 ms). A curved 4-pole mapping catheter located along the LA posterior wall could help to confirm detoured activation of the LA posterior wall circumventing the right and left PV (b). The arrow shows the location of the mapping catheter. MAP indicates 4-pole mapping catheter; CS, coronary sinus; d, distal electrode; and p, proximal electrode.
performed (up to 150 ms from at least 3 sites including the CS and RA under isoproterenol infusion). In cases with induction of AT, the RF energy was appropriately applied to block the induced AT circuit. In the cases with induced AF, additional RF energy was appropriately applied to the area exhibiting complex fractionated atrial electrograms (CFAEs). The spontaneous arrhythmogenic foci in both atria were carefully mapped during the intravenous infusion of isoproterenol, and we attempted to delineate the arrhythmogenic foci from the non-PV foci.

Radiofrequency (RF) energy was delivered for 30 to 60 seconds at each site using an 8-mm-tip dumbbell-shaped catheter (Japan Life Line Co., Ltd., Tokyo). The RF energy was delivered with a power of 35 W toward the PVs and 40 W toward the LARL. In cases in which a complete block line could not be obtained at these energy levels, RF energy was increased up to 50 W. The temperature was limited to 55°C.

**Determination of the AT circuit in the LA:** The regularity of the ATs was assessed using recordings from the right atrium and coronary sinus. In the case of a cycle length variability of less than 20%, and basic cycle length greater than 170 ms, the AT circuit was defined as stable and we attempted to determine and terminate the AT circuits. Unstable ATs basically represented stable AT circuits, although these circuits demonstrate spontaneous or pacing-induced variability of cycle length of less than 20% included during AT persistency. AF was defined as a cycle length more than 20% with/without variable dispersion of atrial activation. Mapping of the ATs was performed using multisite entrainment techniques and by observing the P wave morphology and activation sequence recorded from the atrial electrodes. Entrainment with concealed fusion and a postpacing interval within 20 ms of the tachycardia cycle length was located within the AT circuit (Figure 2).

First, we examined whether there was an AT with a macro-reentrant mechanism. Recordings with the continuous activation pattern with the same direction through the LA roof on the anterior and posterior walls, and demonstration of entrainment with concealed fusion at the roof between the left and right upper PVs, anterior wall, and posterior wall were confirmed. Mitral annulus-dependent AT was defined as that which demonstrated the entire tachycardia cycle length occurring surrounding the mitral annulus and showed entrainment with concealed fusion at the mitral isthmus, peri-mitral anterior wall, and peri-mitral posterior wall. A septal AT was defined as that which demonstrated the entire tachycardia cycle length around the site of LA transseptal puncture and showed entrainment with concealed fusion of the anterior, posterior, upper, and lower sides around the LA side of its transseptal site.

After macro-reentrant AT was ruled out, we attempted to determine the location of a focal or localized AT. The ablation catheter was used to identify any centrifugal patterns or areas exhibiting a long conduction time with fractionated potentials. Repeated pacing maneuvers with measurement of the postpacing interval were also used to determine the precise location. RF energy was applied between the anatomical obstacles that allowed activation to cross a critical isthmus of the macro-reentrant circuit, with a long conduction interval with fractionated potentials representing localized reentry and the site of earliest activation of the focal mechanism.

**Follow-up:** All patients were discharged home 3 days after the CA procedure and were seen in our hospital at 1-2-month intervals. The AF episodes were adequately assessed by the symptoms, 12-lead ECG, and 24-h Holter ECG recordings. AF recurrence was defined as the occurrence of atrial tachyarrhythmias after a 2-month blanking period after the CA procedure. The mean follow-up duration was 583 days (range, 301-1075 days). AF recurrence was defined as episodes lasting more than 30 seconds.

**Statistical analysis:** Continuous variables are expressed as the mean ± SD. Variables were compared by paired t test or χ² test. All analyses were performed using SPSS 10.0 statistical software (SPSS Inc., Chicago, IL), and a P < 0.05 was considered statistically significant.

**RESULTS**

Figure 3 shows a summary of the atrial tachyarrhythmia induction protocol. AF occurred during the PV isolation procedure in 20 of 139 patients (14%), and the temporal sinus rhythm was restored successfully by direct cardioversion after PV isolation in all patients. Of the 139 patients enrolled in this study (paroxysmal, n = 98; persistent, n = 41), AF was induced by a burst pacing protocol in 98 patients (71%: paroxysmal, 64%; persistent, 85%), and then an additional LARL was created in these inducible AF patients. Complete PV electrical dissociation was confirmed in all patients. Complete electrical block across the LARL was achieved in 94 of 98 (95%) patients. The mean procedural time was 159 ± 40 minutes, the mean fluoroscopy time was 48 ± 17 minutes, and the mean RF duration was 38 ± 12 minutes. Cardiac tamponade occurred in one patient, which was resolved by pericardial drainage without cardiac surgery. Antiarrhythmic drugs were administered after the CA in 29% of the patients (persistent, 46%; paroxysmal, 21%).

**Characteristics of the AT before and after creating the LARL:** The characteristics of the stable ATs before creating the LARL are shown in Figure 4. A total of 40 ATs were observed in 25 of the 139 patients (18%: paroxysmal, 20%; persistent, 12%). The estimated circuits were determined in 35 of 40 stable ATs (87%), and were shown to include common atrial flutter (n = 15), roof-dependent AT (n = 12), Cs ostium AT (n = 3), mitral annulus AT (n = 2), AT surrounding left PVs (n = 1), AT sur-
rounding right PVs (n = 1), and septal type AT (n = 1). Unstable ATs were observed in 4 patients (paroxysmal, n = 2; persistent, n = 2).

The characteristics of the stable ATs after creating the LARL are shown in Figure 5. LARL was performed a median of 14 minutes (range, 4-30 minutes) after the PV isolation procedure. A total of 112 stable ATs were observed after creating the LARL in 54 of 98 patients (55%: paroxysmal, 58%; persistent, 48%). The estimated circuits were successfully determined in 99 of 112 stable ATs (88%), and were shown to include common atrial flutter (n = 35), mitral annulus AT (n = 22), septal AT (n = 15), AT surrounding right PVs (n = 12), Cs ostium AT (n = 4), upper loop AT (n = 4), AT surrounding left PVs (n = 4), and left atrial (LA) anterior wall AT (n = 3). The possible localized AT mechanism was observed in 6 ATs (anterior wall AT, n = 3; surrounding left PV, n = 3), and the possible focal mechanism was observed in 8 ATs (Cs ostium AT, n = 4; surrounding right PV, n = 4). Mean cycle length of common atrial flutter was 224 ± 35 ms, and that of mitral annulus AT was 232 ± 40 ms. Unstable ATs were observed in 16 patients (paroxysmal, n = 9; persistent, n = 7).

Common atrial flutters were successfully terminated by creating a tricuspid isthmus block line between the inferior vena cava and tricuspid annulus in all patients (100%). The mitral annulus ATs could be terminated by adding an additional mitral isthmus or LA anterior line block in 15/22 patients (68%). The septal ATs could be terminated by left side septal ablation (from the septum to the right PVs) in 12 of 15 patients (80%). We did not confirm the complete block line in the septum. The ATs surrounding the right PVs could be terminated in all patients (conduction gaps, n = 4; carina between the upper and lower PVs, n = 12). The upper loop ATs could be terminated by ablating the upper to mid-crista conduction gap in all patients (100%). The Cs ostium ATs could be terminated by ablating the site at the Cs ostium exhibiting the earliest activation in 3 of 4 patients (75%). The LA anterior wall ATs were successfully terminated in all patients (100%) by ablating the areas that exhibited prolonged activation with fractionated potentials or early activation sites. The ATs surrounding left PVs were successfully terminated in all of these patients by ablating the LAA–LSPV ridge in 3 cases and the earliest activation site in 1 case. Finally, the stable ATs with confirmed reentrant circuits occurring during the CA (ATs before LARL 23, ATs after LARL 99) were successfully terminated in 111 of 126 stable ATs (87%).

Effects of LARL on inducibility of atrial tachyarrhythmias: LARL significantly reduced the incidence of inducible AF (before versus after: 100% versus 44%, respectively, P < 0.01), whereas it significantly increased the incidence of AT (18% versus 63%, respectively, P < 0.01). The incidence of inducible AF significantly decreased after LARL creation (44% vs 18%, P < 0.01). The LARL significantly reduced the incidence of paroxysmal AF (63% vs 45%, P < 0.01) and increased the incidence of persistent AF (34% vs 55%, P < 0.01). The estimated circuits were successfully determined in 35 of 40 stable ATs (87%). In cases with cycle length variability of less than 20%, the AT circuit was defined as stable, and we mapped these ATs.
ble stable ATs that originated from the LA was significantly increased after compared with before LARL (mitral annulus AT: 1.4% versus 22.4%, \( P < 0.01 \); septal: 1.0% versus 15.3%, \( P < 0.01 \); surrounding right PV: 1.0% versus 12.2%, \( P < 0.01 \); LA appendage: 0% versus 2.0%; and LA anterior: 0% versus 3.1%).

**Clinical outcome:** Antiarrhythmic drugs were administered after the CA in 41 of 139 patients (29%; paroxysmal, 19%; persistent, 51%) (Vaughan-Williams scale: class I, 16%; class II, 12%; class III, 21%; class IV, 4%). The occurrence of AT after CA was significantly higher in patients with residual ATs than in patients without residual ATs (26% versus 2%, respectively, \( P < 0.05 \): paroxysmal, 10%; persistent, 14%). The occurrence of AF after CA did not differ significantly between the two groups (10% versus 7%, respectively; NS: paroxysmal, 9%; persistent, 9%).

**Discussion**

In this study, we evaluated the effects of LARL creation on inducible ATs after electrical PV isolation in patients with AF, and assessed the electrical characteristics of the ATs before and after LARL creation. The LARL significantly reduced the incidence of AF, but increased the incidence of reentrant ATs originating from the LA. The LARL prevented perpetuation of the AF; however, the creating of a secondary boundary by the LARL may likely be associated not only with the increased macro-reentrant ATs occurring away from the PV ostium, but also with increased latent micro- or localized reentrant ATs without central anatomical obstacles. Further, inducible ATs with stable circuits could also be successfully terminated by additional RF energy applications (91%), and the following AT episodes were significantly higher in the patients with than in those without residual ATs (26% versus 2%, respectively).

**Effects of a roof line on AF:** PVs are dominant sources of triggers initiating AF in patients with paroxysmal AF; however, a PV electrical isolation strategy alone for patients with an enlarged atrium or persistent AF may be quite limited. It is now recognized that the development of AF leads to electrical and structural changes within the atria that could allow for the promotion and maintenance of localized high-frequency activity (focal or reentrant), meandering multiple wavelet reentry, and macro-reentry. In addition, the structural changes further promote the inconsistency and prolongation of the atrial conduction, which leads to maintenance of the perpetuation of AF.

Nademanee, et al.\(^9\) reported that the LA roof represents a region demonstrating highly fragmented electrophors, perhaps indicating the presence of a substrate capable of sustaining localized reentry or focal activity to maintain the AF. In addition, ablation along the LA roof creating a complete block line resulted in a slowing of the AF process and termination of AF in 47% of the patients undergoing creation of an LARL during AF\(^10\) with a favorable clinical course. These observations implied that the LA roof plays a major role in maintaining fibrillatory circuits in most AF patients. In the present study, the LARL significantly reduced the percentage of AF inducibility by 44% in cases of inducible AF. This result suggested that the LARL facilitates delineation of one of the main substrates of AF and simplifies the multiple AF reentrant circuits, causing a change from an unstable to a stable reentrant circuit.

**Characteristics of the AT after creating the roof line:** The incidence of LA tachycardia after CA appears to be higher in patients treated with anatomic linear lesions around the PVs, ranging from 10% to 31%,\(^10-14\) and the rate of ATs seemed to be dependent on the technical aspects. Circumferential left atrial ablation around the PVs was associated with a greater incidence of ATs either due to macro-reentries using the PV isolated area as the central or lateral border,\(^9\) and the RF ablation targeting CFAEs yielded an incidence of left ATs of about 8%\(^9\).

The following AT also commonly occurs after surgical procedures. Macro-reentrant AT may occur in either atrium and may be the result of the proarrhythmic effects of these operations. Surgical boundaries, if incomplete, may promote intra-arrhythmic reentrant circuits,\(^2\) whereas complete lesions could also create an environment for macro-reentrant circuits that depend on the tissue with anisotropic conduction properties if a functional central obstacle is protected by surgical boundaries. Central functional barriers, such as the crista terminalis, tricuspid annulus, superior vena cava, coronary sinus, and mitral annulus, could provide an obstacle to maintaining a stable AT circuit, which may make it possible for this anisotropic obstacle to assume a role in organized conduction.

In this study, the addition of a complete LARL after PV isolation significantly increased the incidence of inducible ATs from 18% to 63%. The incidence of induced ATs was higher in the present study compared to those reported previously. As AT was induced from multiple sites with administration of isoproterenol, the aggressive induction protocol may have had a significant impact on the increased AT inducibility.\(^6\) LARL creation further promoted the increased incidence of macro-reentrant ATs originating from the LA, including mitral annulus-dependent ATs, ATs surrounding right PVs, and septal ATs. The additional created LARL, the boundary zone anterior to the right PVs corresponding to the limbus of the fossa ovalis, and the mitral annulus acting as a lateral functional barrier may have supported the stable circuit of these types of macro-reentrant ATs originating from the LA.\(^7\) In this study, stable ATs from the right atrium, such as tricuspid isthmus-dependent ATs, were also commonly inducible. Therefore, the following macro-reentrant ATs in the right atrium could have emerged after LARL creation.

LA anterior wall ATs and ATs surrounding right or left PVs could be observed after circumferential PV isolation or the stepwise approach,\(^16,19\) and the mechanism responsible for the ATs located in the left anterior wall could be localized reentry within a low voltage zone. In addition, the intervenous ridge, the so-called “PV carina,” where the transmural myocardial thickness of the PV-atrial junction and epicardial atrial fibers acting as an electrical connection across the ablation line, may have facilitated development of the substrate mainly responsible for the ATs surrounding right and left PVs.\(^20-23\) The structural and functional characteristics of these regions, including the narrow and thick musculature, complex myofibril arrangement, and proximity to the autonomic ganglia and the ligament of Marshall may support the formation of a slow conduction obstacle of reentry due to the nontransmurality of the AF ablation lesions.\(^23\)

**Study limitations:** We did not use a 3D electroanatomical mapping system to evaluate the circuit of the induced ATs, or to confirm the block line along LARL. Therefore, there were
some limitations in this study regarding assessment of the course of induced ATs. First, we may have misdiagnosed the course of the induced ATs because we could not assess the location of the low voltage zone and the entire reentrant course of the inducible ATs using a 3D mapping system. Second, the recovery of conduction along the LARL may be associated with an increased incidence of inducible ATs, especially in cases with mitral ATs or surrounding PV ATs. Third, we could not confirm the frequency of AF episodes, and the AF duration was determined according to the patients’ symptoms. Therefore, we could not fully evaluate the asymptomatic AF episodes in the present study.

Conclusions: The noninducibility of LA tachyarrhythmias achieved by additional linear ablation may be associated with improved clinical outcome.25-27 Delineation of the inducible ATs with noninducibility after circumferential PV isolation may be helpful to prevent the recurrence of ATs.10 In the present study, the inducible ATs after creating the LARL could be successfully mapped and delineated in almost all cases by using a deductive entrainment mapping technique. Moreover, the absence of residual inducible ATs at the endpoint of the CA procedure seemed to predict prevention of the occurrence of ATs during follow-up. Thus, a vigorous strategy to delineate the ATs after creating the LARL may lead to a favorable outcome after CA.

References