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Defibrillation-Guided Radiofrequency Ablation of Atrial Fibrillation Secondary to an Atrial Focus

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OBJECTIVES Our aim was to evaluate a potential focal source of atrial fibrillation (AF) by unmasking spontaneous early reinitiation of AF after transvenous atrial defibrillation (TADF), and to describe a method of using repeated TADF to map and ablate the focus.

BACKGROUND Atrial fibrillation may develop secondary to a rapidly discharging atrial focus that the atria cannot follow synchronously, with suppression of the focus once AF establishes. Focus mapping and radiofrequency (RF) ablation may be curative but is limited if the patient is in AF or if the focus is quiescent. Early reinitiation of AF has been observed following defibrillation, which might have a focal mechanism.

METHODS We performed TADF in patients with drug-refractory lone AF using electrodes in the right atrium (RA) and the coronary sinus. When reproducible early reinitiation of AF within 2 min after TADF was observed that exhibited a potential focal mechanism, both mapping and RF ablation were performed to suppress AF reinitiation. Clinical and ambulatory ECG monitoring was used to assess AF recurrence.

RESULTS A total of 44 lone AF patients (40 men, 4 women; 32 persistent, 12 paroxysmal AF) with a mean age of 58 ± 13 years underwent TADF. Sixteen patients had early reinitiation of AF after TADF, nine (20%; 5 paroxysmal) exhibited a pattern of focal reinitiation. Earliest atrial activation was mapped to the right superior (n = 4) and the left superior (n = 3) pulmonary vein, just inside the orifice, in the seven patients who underwent further study. At the onset of AF reinitiation, the site of earliest activation was 86 ± 38 ms ahead of the RA reference electrogram. The atrial activities from this site were fragmented and exhibited progressive cycle-length shortening with decremental conduction to the rest of the atrium until AF reinitiated. Radiofrequency ablation at the earliest activation site resulted in suppression of AF reinitiation despite pace-inducibility. Improved clinical outcome was observed over 8 ± 4 months' follow-up.

CONCLUSIONS Transvenous atrial defibrillation can help to unmask, map, and ablate a potential atrial focus in patients with paroxysmal and persistent AF. A consistent atrial focus is the cause of early reinitiation of AF in 20% of patients with lone AF, and these patients may benefit from this technique. (J Am Coll Cardiol 1999;33:1217–26) © 1999 by the American College of Cardiology

The limitations of drug treatment in maintaining sinus rhythm in patients with atrial fibrillation (AF) include the relatively low efficacy rate, and the risk of proarrhythmia with the present medications (1–5). These limitations have sped the investigation of nonpharmacologic therapies for AF, such as the use of radiofrequency (RF) catheter ablation for ventricular rate control (6–11), and more recently, the use of atrial defibrillators for restoration of sinus rhythm (12). The former involves either the complete (6–8) ablation of the atrioventricular node with implantation of permanent pacemaker, or modification of the capacity of the atrioventricular nodal conduction (9–11). However, after RF ablation, the atria continue to fibrillate, and patients are deprived of the hemodynamic benefit of synchronized atrial contraction, and they continue to be exposed to the risk of thromboembolism.

It would be preferable to abolish AF by RF ablation of the atrium itself. Based on Moe's concept (13) that AF occurs as a result of multiple atrial wavelets, Cox et al. (14,15) devised ingenious surgical procedures that divide the atrium into separate compartments that are electrically

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Abbreviations and Acronyms

AF = atrial fibrillation
CS = coronary sinus
ECG = electrocardiogram, electrocardiographic
IV = intravenous
LA = left atrium
RA = right atrium
RF = radiofrequency
TADF = transvenous atrial defibrillation

connected but such that each is too small to sustain an AF wavelet, the so-called maze operation. This has been shown to be effective not only in achieving sinus rhythm, but also in maintaining normal atrial mechanical function in 80% of cases (15). Attempts have been made to reproduce the maze procedure by creating linear lines using catheter RF ablation around anatomical landmarks in either the right atrium (RA) or the left atrium (LA) or both (16–21). These have led to variable success rates, as ablation is limited by the difficulty in achieving a complete line of electrical block. The procedures tend to be lengthy and are associated with significant morbidity.

After the creation of RF ablation lines, it was observed that in some cases of paroxysmal AF, the fibrillatory activity was converted to an "organized" activity (20). In some patients, atrial flutter, sustained monomorphic atrial tachycardia or repetitive extrasystoles that triggered episodes of AF were noted after linear ablation. These arrhythmias required separate ablation. More recently, a rapidly discharging focus has been reported to occur in patients with paroxysmal AF, which lead to an electrocardiographic (ECG) pattern of coarse AF (22). Ablation of these foci was associated with clinical cure of AF. However, in both cases it is difficult to locate the focus if the patient is in AF or develops AF during the procedure, as disorganized electrical activation during AF precludes the ability to accurately map, study and ablate the arrhythmia. Furthermore, the extent to which such foci occur in the overall population of patients with AF is unknown.

In about 30% of patients after external or transvenous atrial defibrillation (TADF), early reinitiation of AF within minutes of defibrillation has been observed (23–28). A consistent atrial ectopic is the cause of reinitiation in some cases. We hypothesize that a discharging focus is the cause of AF reinitiation after atrial defibrillation, and RF ablation to eliminate this focus will prevent AF reinitiation.

Our objectives in this study were 1) to examine the incidence of a rapidly discharging atrial focus acting as a precursor arrhythmia that leads to AF in patients without structural heart disease, and 2) to describe a new method by which defibrillation, mapping and ablation are used to identify and abolish this focus in patients who present in either sinus rhythm or AF.

METHODS

Patients. We performed TADF and electrophysiologic study in 44 consecutive patients (40 men, 4 women) with symptomatic, drug-refractory AF who had no significant underlying structural heart disease. Both TADF and the electrophysiologic study were performed in these patients for restoration of sinus rhythm and to evaluate nonpharmacologic therapy. There were 32 patients with persistent and 12 patients with paroxysmal AF, respectively, and the mean age was 58 ± 13 years. Patients had symptoms ranging from continuous (persistent AF patients) to once per week. Transvenous atrial defibrillation (TADF) was used as the first procedure in patients with persistent AF and was used to restore sinus rhythm after AF induction in patients who were initially in sinus rhythm at the time of study. The protocol of the study was approved by the ethics committee of the University of Hong Kong, and all patients gave written informed consent.

Procedure and catheter placement. The procedure was performed in the fasting state and consisted of a combination of TADF, mapping and RF ablation. Multipolar catheters were introduced from both femoral veins and the left subclavian vein under local anesthesia. A pair of 7F custom-made decapolar catheters (Elecath, Rahway, New Jersey) were advanced to the anterolateral RA and the distal coronary sinus (CS). Quadripolar catheters were also placed in the high interatrial septum, the atrioventricular junction and the right ventricular apex (Fig. 1A). Intravenous (IV) heparin, 3000 U, was given as a bolus once all catheters were in place, followed by 1000 U hourly during the remainder of the procedure. Sedation using IV midazolam and pethidine was commenced before TADF. Continuous recording of at least two surface ECG leads and intracardiac recordings (filtered at 30 to 500 Hz) were performed using a multichannel recorder (PPG, Midas 5000, Lenexa, Kansas or Prucka, Cardio Lab EP 4.0, Houston, Texas).

Transvenous atrial defibrillation. The details of TADF in our center has been reported (29). In brief, defibrillation was performed along the RA to CS shock vector using 3 × 3 ms biphasic shocks delivered from an external defibrillator (XAD, InControl, Redmond, Washington). After a test shock of 10 V, defibrillation testing was started at 180 V with the shock strength increased in 40-V steps until defibrillation was achieved. Conversely, a 40-V stepdown was used in patients in whom sinus rhythm was restored at 180 V until defibrillation failed. Repeated defibrillation was subsequently performed at least 40 V above this defibrillation limit. For patients who were in sinus rhythm, sustained AF was induced using burst atrial pacing and TADF was then performed. In one patient who had an implanted atrial defibrillator (Model 3000, Metrix, InControl, Seattle, Washington), TADF was performed using the implanted device.

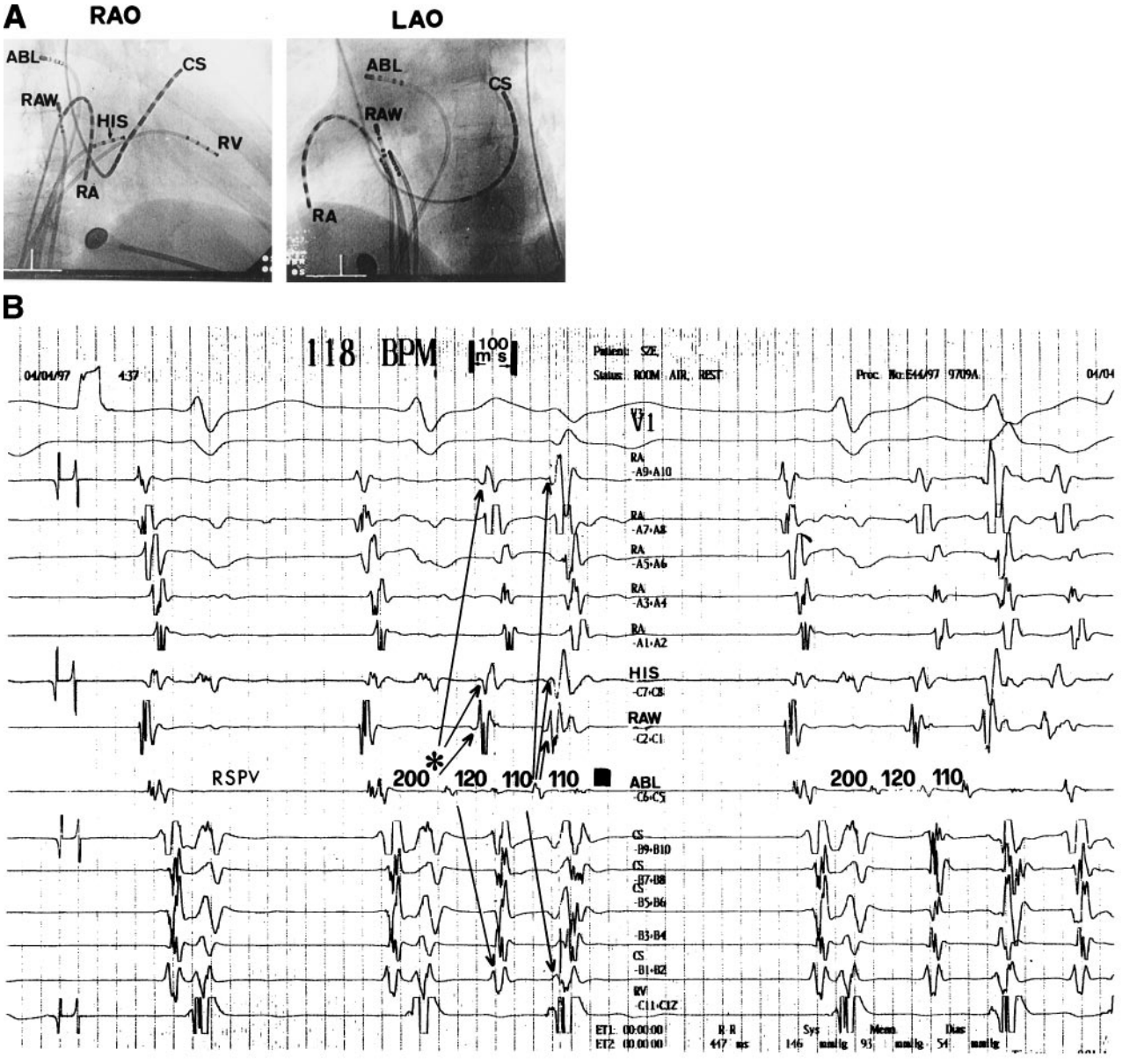

Figure 1. Cineangiographic views **(A)** of catheter layout in a patient undergoing defibrillation-guided mapping and RF ablation of AF secondary to a focus in the right superior pulmonary vein, just inside the orifice (Patient 2). **(B)** Recording of spontaneous AF reinitiation in a patient with a focus just inside the RSPV (same Patient as in **A**). At the site of putative focus, the atrial electrogram during sinus rhythm is fragmented. A rapidly discharging focus with fragmented electrograms and a reproducible coupling interval of 200 ms to the sinus beat initiates a nonsustained, then a sustained run of AF. The first beat of the focus (*) is 115 ms (not labeled) ahead of the high right atrial electrogram (RA A9–A10) There was shortening of the cycle length after the first beat of the focus before the onset of AF. Because of the rapidity of the tachycardia, some electrograms appeared to be blocked (second and fourth beat after *). Note a similar activation sequence of the RA, but earlier activation of HIS and RAW electrograms at the first beat of the tachycardia compared to the sinus beat, with advancement of all CS electrograms suggestive of a LA focus. ABL = ablation catheter (at RSPV); CS = coronary sinus (B9, B10 = proximal CS; B1, B2 = distal CS); HIS = His bundle; LAO = left anterior oblique view; RA = right atrium (A9, A10 = high RA; A1, A2 = low RA); RAO = right anterior oblique view; RAW = high right interatrial septum; RV = right ventricular apex; RSPV = right superior pulmonary vein. Each calibration mark on the left represents 1 mV for that and the following recordings until a change in calibration was made.

Mapping of focal AF. Immediately after sinus rhythm was restored by a TADF shock, a switch box was used to generate multiple biopolar intracardiac electrograms from the RA and CS defibrillation catheters. These electrograms were recorded via the switch box onto the recorder. Continuous recordings at 100 mm/s were made for up to 2 min after the shock or until reinitiation of AF occurred. After an additional 2 min of AF, defibrillation was again carried out to see whether there was repeatable reinitiation of AF from a consistent site. In case of absence of recurrence within

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2 min, AF reinduction was performed by burst atrial pacing, and the TADF protocol was repeated for up to a total of three times. If there was no AF recurrence, mapping of a potential focus for AF was abandoned.

For patients who developed early reinitiation of AF, the following observations were noted: 1) electrogram characteristics of the earliest activation site during AF reinitiation; 2) coupling intervals of the first three atrial electrical activations before degeneration into AF; and 3) pattern of AF reinitiation, which included the earliest site of activation that consistently reinitiated AF. If this initial mapping of the reinitiation of AF was indicative of an atrial focus, additional right and left atrial mapping was performed to locate the focus site more precisely. Left atrial mapping and ablation were performed using standard Brockenbrough transeptal needle puncture technique and a long sheath (SL-2, Daig, Minnetonka, Minnesota). Cycle lengths of potential sites of the focus were measured from the ablation catheter electrogram using the onset of high-frequency electrical activity when present or from the initial rapid departure from baseline when absent.

RF ablation. An 8F 4-mm tip mapping catheter (Marinr, Medtronic Inc., Minneapolis, Minnesota) was used to deliver RF energy at the earliest atrial focus that initiated AF. With a maximum wattage set at 50 W, RF energy was applied to achieve a temperature of 55° to 60°C for up to 2 min using a RF generator (Atakr, Medtronic Inc., Minneapolis, Minnesota). Successful ablation was defined as disappearance of early reinitiation of AF. A "safety" application was routinely applied at the successful site. After ablation, reinduction of AF by burst atrial pacing was attempted using the ablation catheter at the successful site and at the high RA. Reinduction of AF and repeated TADF, with and without isoprenaline, were performed to assess the suppression of early reinitiation of AF.

Postablation care and follow-up. All catheters were removed after the procedure, and hemostasis was achieved using direct pressure. Low-molecular-weight heparin in a dose of 10,000 U/d was given subcutaneously for two weeks after the procedure for patients with a history of paroxysmal AF, and was changed soon after the procedure to warfarin anticoagulation, continued for three months at a therapeutic international normalized ratio, in patients with persistent AF. All patients were given an event recorder and underwent periodic Holter monitoring for documentation of arrhythmia recurrence. They were seen in the arrhythmia clinic at 2, 4, and then every 8 weeks for evaluation of AF recurrence. Routine transthoracic echocardiography was performed before, immediately after and at three months' postablation to assess left ventricular function, presence or absence of pericardial effusion, left atrial clots and pulmonary arterial pressure. Doppler flow measurements of the pulmonary veins were made at approximately three months' postprocedure using transthoracic methods. Transesophageal echocardiography was performed on follow-up to assess potential pulmonary vein stenosis after ablation.

Statistical analysis. Results are expressed as mean ± SD. Differences were evaluated by paired or unpaired Student *t* test for continuous variables. A value of $p < 0.05$ was considered statistically significant.

RESULTS

A total of 44 patients with lone AF underwent TADF. Of these, 12 patients had paroxysmal AF and the remaining 32 patients had AF for at least 1 month (mean: 27 ± 29 months, range: 1.5 to 120 months). Six of 12 patients with paroxysmal AF had early recurrence of AF during the TADF procedure, of which five patients (42%) showed a consistent atrial activation pattern and coupling intervals to the sinus beat suggestive of a focal initiation. Of 32 patients with persistent AF, 10 had early reinitiation of AF, of which 4 (13%) showed an atrial activation pattern suggestive of a consistent focus. These 9 patients were younger than those without a consistent focus for reinitiation of AF (40 ± 10 vs. 62 ± 9 years, $p < 0.001$). The left atrial diameter and ejection fraction on echocardiography were 37 ± 4 mm and 68 ± 11%, respectively. Of these nine patients, seven consented to participate in RF ablation, which was performed as a one-stage procedure in two patients, and on separate occasions in the remaining patients. These patients had daily episodes of palpitation despite the use of a class Ic drug and sotalol.

Earliest site of reinitiation of AF. Four patients were in AF at the time of the study, and AF was induced in the remaining patients. In all nine patients, AF was successfully defibrillated with an atrial defibrillation limit of 240 ± 30 V (approximately 1.6 J). Early reinitiation of AF occurred with a mean of 23 ± 7 s (range 3 to 120 s). In all patients, the RA (including the right posterior interatrial septum) electrograms of the first beat of tachycardia showed a similar pattern, with subtle differences, as compared with sinus rhythm (Fig. 1B; Fig. 2). Conversely, the CS electrograms were significantly advanced as compared with sinus rhythm, suggestive of a LA focus. Of the seven patients undergoing RF ablation, four foci were mapped to the right superior pulmonary vein, just inside the orifice, and the remaining three foci to the left superior pulmonary vein, just inside the orifice. The earliest site determined to be the focus was 86 ± 38 ms (range 40 to 140 ms) ahead of the high RA reference electrogram.

Characteristics of the atrial electrogram. During sinus rhythm, the atrial site of the earliest activation showed a fragmented electrogram pattern identified by first entering one of the pulmonary veins and withdrawing the catheter until a fragmented atrial electrogram was recorded. In five of seven patients, the electrogram during sinus rhythm showed an initial low frequency component followed by a high-frequency component (Fig. 2). At the first tachycardia beat,

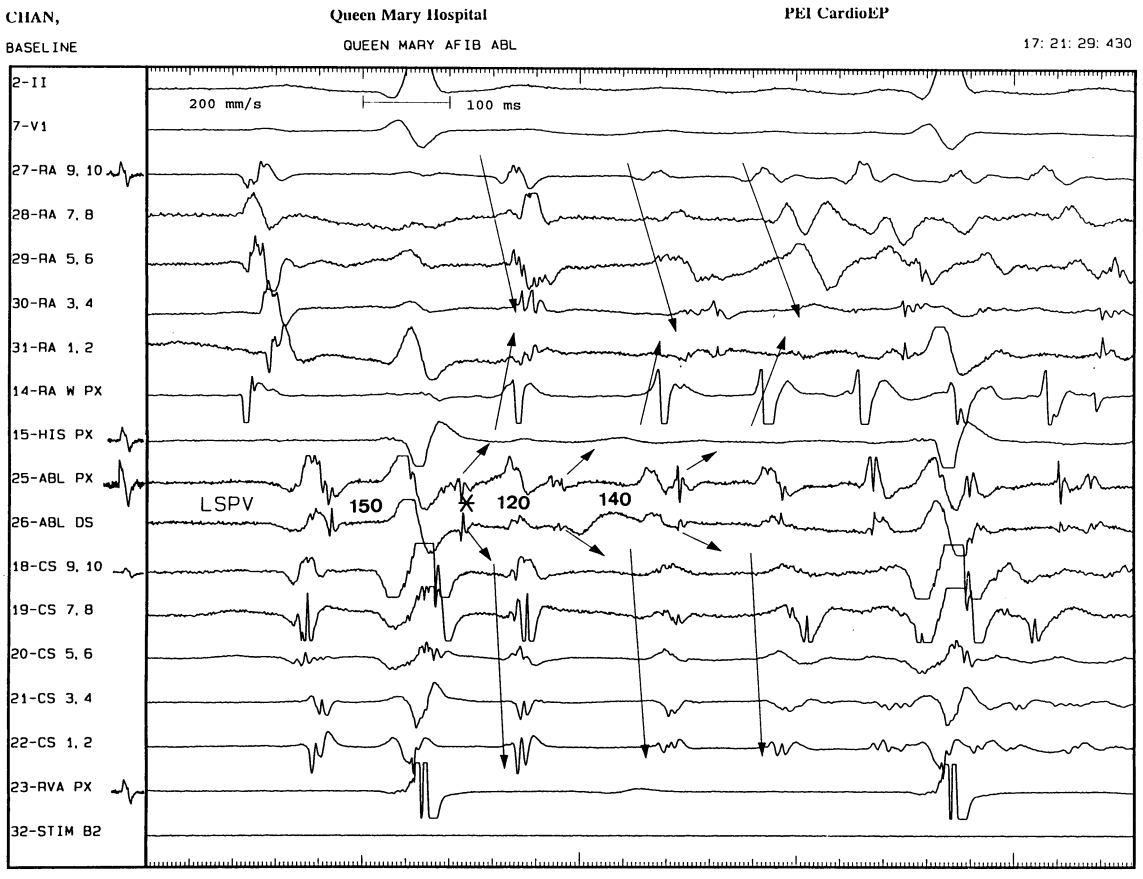

Figure 2. Activation of a focus leading to AF in patient 7 from just inside the left superior pulmonary vein (LSPV, HIS potential was of low amplitude). The first beat of the focus (*) was 58 ms (not labeled) ahead of the high RA electrogram. In this patient, decremental conduction occurred from the focus to the rest of the atrium, and advancement of CS electrograms compared to the sinus beat. At the ablation catheter, the atrial electrogram shows an initial rounded then a fast component during sinus rhythm, which appears to reverse and separate at the first beat of tachycardia. Abbreviations as in Figure 1. Each calibration mark on the left represents 1 mV for that and the following recordings until a change in calibration was made.

the high-frequency component appeared to advance ahead of the low-frequency component. In the other two patients, only a single electrogram signal was observed and it advanced in time at the onset of the tachycardia with respect to both the surface P-wave and high RA electrogram. There was a shortening of the cycle lengths of the second and the third beat of the focus compared with the first one before AF initiation (Table 1); the focus thereafter was usually overdriven by the fibrillation wavelets. Progressive decremental conduction to the remainder of the atrium was observed prior to AF reinitiation.

RF ablation. Radiofrequency energy (mean number of RF applications: 13 ± 7) was applied at the putative site of the AF-initiating focus and resulted in suppression of AF reinitiation in all patients (Fig. 3A and B). In four patients, modification of the focus occurred, resulting in only single ectopic beats that did not reinitiate AF. During the "safety" application of RF energy, these single ectopics were abolished in all patients. In the patient with an implanted atrial defibrillator, sustained AF was induced and lasted for over 1 h during the procedure. The RF ablation at the focus resulted in termination of the AF, followed by single ectopics and finally normal sinus rhythm. Burst pacing both at the successful RF ablation site and at the RA reinduced AF in all patients despite successful RF ablation to prevent AF reinitiation. No procedural-related complications occurred, nor was there elevation of serum creatine phosphokinase.

Follow-up. One patient (Patient 6) with a history of persistent AF had recurrence of AF on day 2 after the procedure. He refused further attempts to restore sinus rhythm, and he continued to receive atrioventricular nodal blocking agent. The remaining patients were asymptomatic, and no symptomatic AF occurred in the postprocedural period via review of Holter or event recordings. No significant change occurred in left ventricular function nor LA size during follow-up, nor was there elevation of pulmonary arterial pressure as measured by echocardiography. There were no evidence of pulmonary vein stenosis on transesophageal echocardiography.

Table 1. Characteristics of Atrial Focus and RF Ablation

Patient	AF	RFA Site	HRA-ABL (ms)	1st CL (ms)	2nd CL (ms)	3rd CL (ms)	AF RA	Types LA	Fluoroscopic Time (min)	Duration (h)	No. of RFs
1	Paroxysmal	RSPV	−40	130	120	120	II	III	91	3.2	26
2	Paroxysmal	RSPV	−115	120	110	110	II	II	90	4.2	10
3†	Paroxysmal	LSPV	−55	270	180	160	I	II	109	4.2	6
5	Paroxysmal	RSPV	−115	220	180	170	I	I	110	4.2	12
6	Persistent	LSPV	−140	150	116	108	III	II	100	4.3	17
7	Persistent	LSPV	−58	150	120	140	III	III	102	3.8	16
9	Persistent	RSPV	−80	200	180	170	III	III	90	3.6	7
Mean ± SD			−86 ± 38	177 ± 55	144 ± 34*	140 ± 27*			99 ± 9	3.9 ± 0.4	13 ± 7

*$p < 0.01$ compared with 1st CL. †Patient with an implanted atrial defibrillator.
AF = atrial fibrillation; HRA-ABL = difference in timing between the ablation catheter and the high right atrial electrogram at the first beat of the focus; CL = cycle length of atrial focus; LA = left atrium; LSPV = left superior pulmonary vein, just inside the orifice; RFA = radiofrequency ablation; RSPV = right superior pulmonary vein; TADF = transvenous atrial defibrillation.

DISCUSSION

Precursor arrhythmias for AF. Early reinitiation of AF has been described (23–27), and it seems to occur in up to 30% of patients after either external (23,24) or transvenous atrial defibrillation (25–27). The mechanisms of AF reinitiation are probably multiple, but a consistent atrial ectopic activation sequence was observed in some patients. These ectopic beats usually exhibit a higher prematurity index than those that do not initiate AF (26,27). Early reinitiation of AF is also associated with a higher incidence of late AF recurrence despite early suppression with class Ic antiarrhythmic agents (24), but may be reduced with intracellular calcium-lowering medications (28).

In this study we observed that a consistent atrial focus was responsible for early reinitiation of AF in 20% of patients with lone AF who underwent elective transvenous defibrillation. These atrial foci typically occurred around the pulmonary vein or the crista terminalis as described by Jais et al. (22). Apart from a consistent coupling cycle length of the first ectopic beat, we observed that these left atrial foci are recognized by a distinct pattern of atrial activation with advancement of the CS atrial electrograms relative to the RA electrograms when compared to the activation during sinus rhythm. This pattern of initiation was helpful as an indicator for left-sided mapping.

We further observed that the electrograms in the area of the focus typically showed a fragmented pattern of electrical activity with shortening of its cycle length and decremental conduction to the rest of the atria before AF was initiated. As described in preliminary communications (20,30), the electrogram at the pulmonary venous orifices is multicomponent, with an initial lower frequency far-field atrial component and a high-frequency component during sinus rhythm, with advancement of the high-frequency spike compared with the lower frequency electrogram at the onset of tachycardia. In our study we observed a similar complex electrogram in five of seven patients. An anatomical basis for such atrial foci to occur in the pulmonary vein had been suggested by Nathan and Eliakim (31), who demonstrated the presence of atrial myocardial sleeves extending 1 to 2 cm into the pulmonary veins.

Precursor rhythms for AF have been previously described, where the degeneration of the precursor rhythm leads to AF (32–37). These precursor rhythms include the atrioventricular reentrant arrhythmias of the Wolff-Parkinson-White syndrome (32,33), atrial flutter (34), and, more recently, atrioventricular nodal reentrant tachycardia (35). Elimination of these precursor arrhythmias may lead to either a cure or amelioration of AF (34,36,37). We found that an atrial tachyarrhythmia emanating from an isolated focus was a precursor arrhythmia for AF in our patients with reproducible, spontaneous reinitiation of AF following TADF. Furthermore, during the brief runs of atrial activations from the focus, decremental conduction occurred to the rest of the atrium owing to the short cycle length of the tachycar-

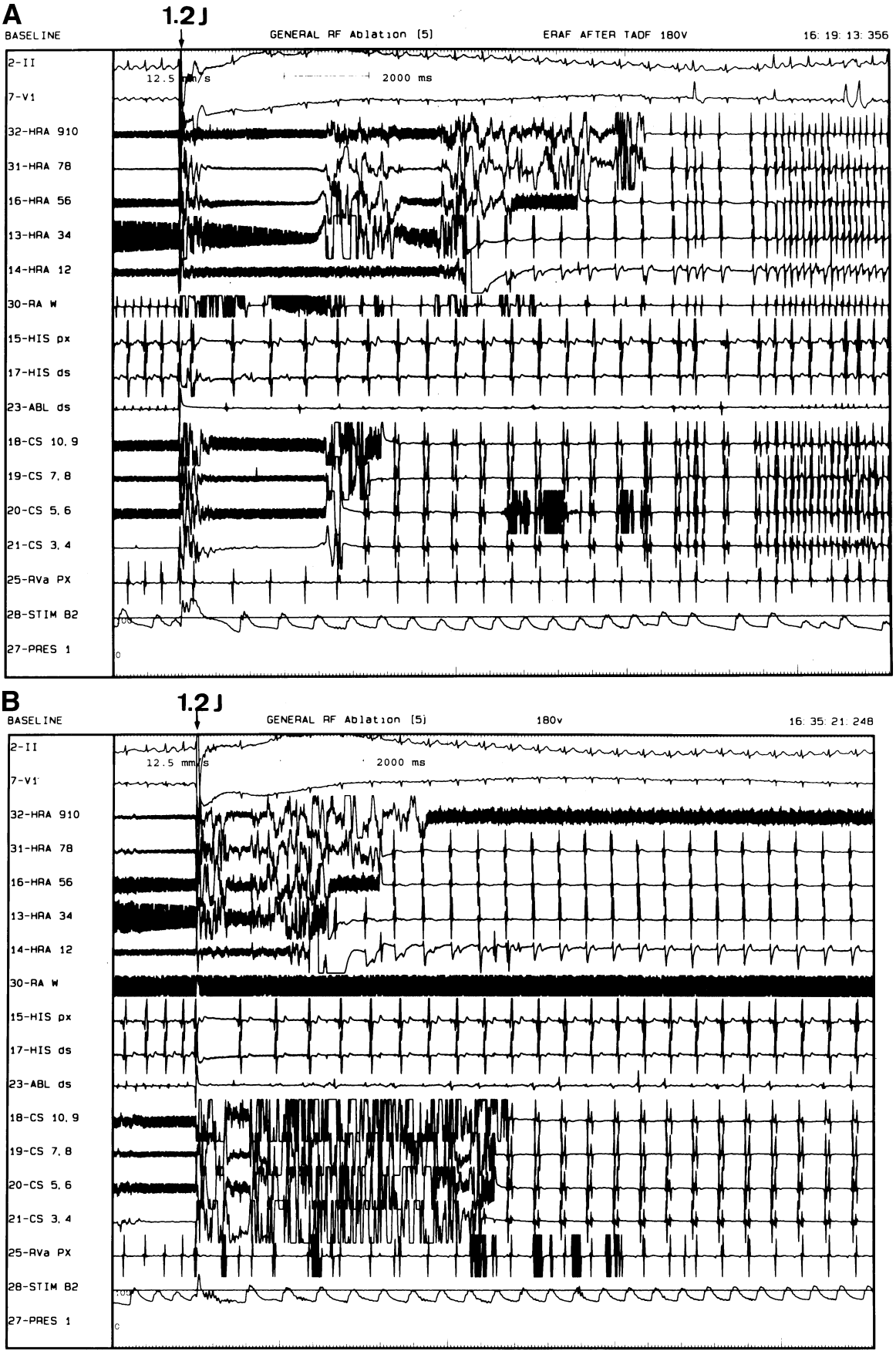

Figure 3. Suppression of early reinitiation of AF after RF ablation of a focus in the right superior pulmonary venous orifice (patient 5). **(A)** Before ablation, AF recurred consistently within 10 s after a 1.2-J transvenous atrial defibrillation shock. **(B)** After ablation, sinus rhythm remained after restoration of induced AF. Recording artifact occurred during switching from the defibrillation to the recording mode. Abbreviations as in Figure 1.

dia, which may lead to reentry and establishment of AF wavelets. Finally, we noted that the organized electrical activity emanating from the focus was suppressed once AF was reestablished, thereby requiring repeated TADF to map the focus accurately. Suppression of early reinitiation of AF by RF ablation led to an improved clinical outcome. Thus, by unmasking a focus that initiates AF with repeated TADF, our technique allows mapping and ablation of this focus in patients who are either in sinus rhythm or in AF.

Interestingly, despite eventual arrhythmia control during the follow-up period, AF remained inducible by burst atrial pacing at the successful RF site and at the high lateral RA immediately after the ablation in all patients. We postulate that atrial remodeling had occurred in these patients as a result of the rapidly discharging focus in a manner similar to an atrial fibrillation pacemaker in goats (38). This continued overdrive of the atria leads to a shortening of atrial refractoriness and loss of its rate adaptation. Reverse remodeling may take some time to occur, and the atrium may remain susceptible to AF reinduction even after successful elimination of the focus (38). Furthermore, elimination of the triggering precursor arrhythmia does not preclude the possibility of recurrent AF due to other mechanisms (39).

Defibrillation-guided mapping of focus-initiated AF. We describe a technique in which TADF was used to identify a potentially curable cause of AF. By repeated TADF and observing early reinitiation of AF, mapping of the putative focus can be achieved and RF catheter ablation of the focus accomplished. Furthermore, suppression of early reinitiation of AF can be used as an end point of RF ablation, as acute AF inducibility may not preclude long-term improvement in clinical outcome. Repeated TADF using the described method has been shown to be safe and without significant proarrhythmia complications and to be well tolerated by patients (29,40–43), provided that R-wave-synchronized shocks are delivered with a preceding cycle length that is longer than 500 ms, without a long-short sequence. We have not observed any proarrhythmia nor mechanical complications following repeated TADF in any patients.

Our technique may be an alternative approach to those reported by Swartz et al. (17) and Haissaguerre et al. (20). Namely, our technique uses induction of AF and subsequent TADF for focus manifestation. The other techniques use a number of stepwise linear ablations and compartmentalization of the atrium to smaller electrical masses in an attempt to prevent AF. Recently, it was reported that in some patients with paroxysmal AF, after a number of linear ablations, AF was converted to an organized rhythm (20), and sustained monomorphic atrial tachycardias or repetitive extrasystoles triggering episodes of AF were observed. These tachycardias required separate ablations. It is likely that these AF-initiating foci were similar to those we observed after TADF. The method we described would allow one to elucidate an underlying focal origin of AF without first going through linear ablation procedure with its associated morbidity. Furthermore, by limiting ablation to the focus rather than around the atria, or to several pulmonary veins, may account for why we have not observed major morbidity such as pulmonary hypertension in our patients after ablation. Although our method cannot be performed in patients who did not have a reproducible recurrence of AF after TADF, it may be useful as a first procedure in approximately 20% of patients with lone AF before contemplating catheter maze procedure.

Our technique may complement the technique of focal ablation described by Jais et al. (22). These investigators mapped a putative focus of AF by relying on either self-terminating runs of an atrial tachycardia that simulate an ECG appearance of AF, or by ablating frequently occurring atrial ectopics that were considered to initiate AF. However, their technique would not be useful for patients who had persistent AF or in patients who develop sustained AF during the procedure in that as we have reported, the focus appears to be passively activated by fibrillatory conduction under these conditions. In addition, inducibility of AF by atrial pacing was suppressed in their study after successful ablation of the focus. This finding is at odds with our observation of postprocedure inducibility of AF, which may be due to the presence of sustained AF in our population (with three of seven patients in persistent AF at the time of the study) such that more extensive atrial remodeling had taken place that favored AF maintenance after induction by any means. However, we required a larger number of ablations to achieve suppression of the focus that was responsible for early AF reinitiation. This may be due to a difference in end points used or the different population studied. As the atrial tissue extending inside the pulmonary vein orifices is significantly larger than the ablation catheter, accurate mapping and placement of the catheters at the exact site of the focus was difficult. Alternatively, the focal source may be larger than can be encompassed by a 4-mm electrode tip. The use of larger-tip catheters or alternative catheter designs may reduce the number of RF applications required.

Study limitations. Although all patients had an acute procedural success, one patient with persistent AF had AF recurrence soon after successful ablation. As the patient refused any further attempts in restoring sinus rhythm, we cannot comment on whether the recurrence was due to an inadequately ablated focus or the residual propensity of developing AF by the remodeled atria. Additionally, a rate of 33% has been reported for AF recurrence after ablation of paroxysmal supraventricular tachycardia as a precursor rhythm for AF (36). Further study is needed to determine whether failed primary therapy with ablation or a secondary mechanism (i.e., atrial stretch or alterations in autonomic tone) is the cause of these AF recurrences.

Finally, of keen interest to the medical community is the concept of reverse remodeling. As our study consisted of

only clinical follow-up and additional electrophysiologic evaluation was beyond the scope of this study, we cannot address the electrophysiologic changes that may have occurred in our patients as a result of their sustained sinus rhythm postprocedure. Additionally, there exists no clinically validated methods to evaluate true inducibility of AF as one must consider factors such as site specificity of pacing to test this factor. For us to repeat our tests of inducibility after follow-up, we would need to perform an additional transseptal puncture with little benefit to the patient, and for these reasons we chose not to reassess inducibility as a measure of atrial reverse remodeling.

Conclusions. Consistent reinitiation of AF after TADF identifies a subset of patients with AF secondary to an atrial focus. A technique using atrial defibrillation-guided mapping followed by catheter ablation of the focus may lead to arrhythmia cure in a significant proportion of patients with lone AF.

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