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ACUTE TERMINATION OF HUMAN ATRIAL FIBRILLATION BY IDENTIFICATION AND CATHETER ABLATION OF LOCALIZED ROTORS AND SOURCES:

First Multicenter Experience of Focal Impulse and Rotor Modulation (FIRM) Ablation

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Abstract

Catheter ablation of atrial fibrillation (AF) currently relies on eliminating triggers, and no reliable method exists to map the arrhythmia itself to identify ablation targets. The aim of this multicenter study was to define the use of Focal Impulse and Rotor Modulation (FIRM) for identifying ablation targets.

METHODS—We prospectively enrolled the first (n=14, 11 males) consecutive patients undergoing FIRM guided ablation for persistent (n=11) and paroxysmal AF at 5 centers. A 64 pole basket catheter was used for panoramic right and left atrial mapping during AF. AF electrograms were analyzed using a novel system to identify sustained rotors (spiral waves), or focal beats (centrifugal activation to surrounding atrium). Ablation was performed first at identified sources. The primary endpoints were acute AF termination or organization (>10 % cycle length prolongation). Conventional ablation was performed only after FIRM guided ablation.

RESULTS—12/14 cases were mapped. AF sources were demonstrated in all patients (average of 1.9±0.8 per patient). Sources were left atrial in 18 cases, and right atrial in 5 cases, and 21/23 were rotors. FIRM guided ablation achieved the acute endpoint in all patients, consisting of AF termination in n=8 (4.9±3.9 min at the primary source), and organization in n=4. Total FIRM time for all patients was 12.3±8.6 min.

CONCLUSIONS—FIRM guided ablation revealed localized AF rotors/focal sources in patients with paroxysmal, persistent and longstanding persistent AF. Brief targeted FIRM guided ablation at a priori identified sites terminated or substantially organized AF in all cases prior to any other ablation.

Keywords

catheter ablation; atrial fibrillation; rotors; FIRM; spiral waves

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INTRODUCTION

Despite several advances, our mechanistic understanding of human atrial fibrillation (AF) remains limited. Haïssaguerre et al ¹ first discovered that ectopic beats from the pulmonary (PV) and thoracic veins may trigger AF. However, it is undefined how paroxysmal or persistent AF, once triggered, are sustained ^{2,3}. Therefore, unlike most arrhythmias, it is difficult to identify targets that could acutely terminate AF during catheter ablation and provide evidence of potential therapeutic benefit.

Mechanisms for the maintenance of human AF are unclear. The *multiwavelet hypothesis* proposes that spatially meandering electrical waves cause AF ^{4,5}. However, this hypothesis does not easily explain why AF exhibits consistent spatial non-uniformities in rate and activation vector ⁶⁻⁸, how ablation may terminate AF relatively early in some cases before compartmentalization of meandering wavelets ^{9,10}, or why extensive ablation often has little acute impact ^{9,11}. The alternative *localized source hypothesis* is based on experimental models in which spiral waves (rotors) ^{12,13} or focal impulses ³ cause disorganized AF when rapid impulses cannot be uniformly propagated. Elegant human studies have used spectral dominant frequency as a surrogate for localized AF sources ^{14,15}, although ablation at such sites has not been shown to acutely terminate AF. However, recent human studies have directly demonstrated, for the first time, that a majority of AF patients exhibit rotors and focal beat sources ¹⁶ where targeted ablation alone can acutely eliminate AF ¹⁷.

We performed a multicenter study to test the feasibility of a novel mapping system to demonstrate rotors and focal beat sources in patients during paroxysmal and persistent AF by multiple operators in various Institutional settings. In addition, we tested the hypothesis that the successive elimination of identifiable sources by Focal Impulse and Rotor Modulation (FIRM) would have a substantial effect on AF (AF termination to sinus rhythm or an organized atrial tachycardia, rendering AF non-inducible, or consistent AF organization).

METHODS

Patient Enrollment

We analyzed data from 14 consecutive patients referred for AF ablation to five busy and experienced ablation centers (University of California Los Angeles, Medical College of Virginia, Ohio State University, Indiana University and Valley Health System/Columbia University College of Physicians & Surgeons). These represent the first cases of FIRM mapping and ablation at each of our Institutions, enrolled from 11/29/2011 to 02/22/2012, at which time we held an Investigator meeting to plan this first report. We enrolled patients with paroxysmal, persistent and longstanding persistent AF referred for standard indications. ⁹ Patients were required to have left atrial diameter < 55 mm (i.e., the maximum size of mapping catheters) on preprocedural imaging to optimize atrial mapping. Nevertheless, as noted below, atria diameters were > 55 mm on intraprocedural measurements in several patients. All patients provided written informed consent for the clinical procedure (utilizing the FDA approved clinical mapping software), and each local IRB allowed review of this data. The clinical workflow for FIRM mapping and ablation is depicted in figure 1.

Procedural Details

Ablation was performed > 5 half-lives after discontinuing all antiarrhythmic medications except amiodarone (Table 1). Via femoral venous access, a linear multipolar catheter was placed in the coronary sinus, and a 64 pole basket catheter (Constellation, Boston Scientific

Inc, Natick, MA) was advanced to the first to the right atrium and then to the left atrium for data acquisition¹⁶ (Figure 2). Baskets were manipulated to optimize electrode contact with the atrium as judged by fluoroscopy (Figure 2), by electrograms and by intracardiac echocardiography¹⁶. Intravenous heparin was infused to maintain activated clotting time > 300 seconds throughout each case.

Baskets provides average interelectrode spacing of 4–8 mm, sufficient to resolve small reentry circuits in human atria with minimum circumference (wavelength¹⁸) of ≈ 4.4 cm (diameter ≈ 1.4 cm) based on shortest action potential durations ≈ 110 ms^{19, 20} and slowest conduction velocity ≈ 40 cm/s²¹ in human atria. Moreover, AF sources should control atrial tissue over a wider area than the minimum wavelength (by definition), and thus may be resolvable with lower resolution.

Patients were studied in native AF whenever possible. Those in sinus rhythm were paced into AF using rapid atrial pacing, with a decremental protocol at cycle lengths (CL) 500 ms, 450 ms, 400 ms, 350 ms, 300 ms, then in 10 ms steps to AF. In some cases, isoproterenol was needed to facilitate initiation. Sustained AF was induced in all patients, and analyzed after 10 minutes. Unipolar intracardiac signals obtained from the basket catheter were filtered at 0.05 to 500 Hz for digital export for analysis from each lab's electrophysiological recorders (Bard Pro, Billerica, MA or Cardiolab, GE Medical, Milwaukee, WI; NavX export, St Jude Medical, Minneapolis, MN).

Computational Mapping of AF

Electrograms were acquired from a wide panorama over both atria, then analyzed using a novel mapping system (RhythmView™, Topera Inc, San Diego, California) that employs a recently described physiologically-guided computational method.¹⁶ Briefly, RhythmView™ is an FDA approved system that first preprocesses electrograms to remove QRS signals and improve signal-to-noise ratio. The system then analyzes AF cycles at each electrode using reported human atrial tissue physiology^{19, 2021} to reconstruct propagation movies (FIRM maps) of electrogram amplitude at each electrode over successive timepoints, projected onto a grid (see Supplementary Movies). This grid, as in figures 2–5, portrays the right atrium opened vertically through the tricuspid annulus, with the lateral tricuspid valve half-opened laterally and the septal half opened medially. The left atrium is opened horizontally along the mitral annulus, with the superior mitral half folded up and the inferior folded down. In this manuscript, single propagation cycles have been extracted from the original movies and are illustrated as isochronal snapshots, color-coded from red (early) to blue (late) (Figures 2–5).

Definition of Localized AF Sources

Rotors (spiral waves) were identified during AF only if activation completed multiple clockwise or counterclockwise rotations (see Supplemental Movies). For each sustained rotor, the center of rotation showed slight movement (precession) bounded by 1–2 electrodes in each axis (1–2 cm², or ≈ 1 –5% of atrial surface area). Focal beat sources in AF were identified as sites with centrifugal activation to surrounding atrium, and their origins identified at the electrodes where activation initiated (again bracketed by 1–2 electrodes). We diagnosed rotors or focal beats only if sustained over multiple 4–8 second recording epochs (> 50 cycles, typically 100s of cycles)¹⁶.

Activation movies were the tool used for FIRM mapping and ablation in each case (Supplemental Movies). These FIRM movies show activation around (rotor) or from (focal) AF sources, and also illustrate the slight movement (precession) of sources within and between cycles. Isochronal snapshots of single AF cycles, generated after each case and

shown in this manuscript (figures 3–6), are illustrative of each case but provide only a summary of these complex AF dynamics.

Focal Impulse and Rotor Modulation (FIRM) Ablation Procedure

Ablation commenced with FIRM at localized sources identified in propagation movies in all patients, i.e., prior to PV isolation. This report focuses on the acute impact of FIRM ablation. Radiofrequency energy was delivered using a 3.5 mm tip irrigated catheter (Thermocool™, Biosense-Webster, Diamond Bar, CA or Safire B™, St Jude Medical, Minnetonka, MN) at 25–40 W depending on location (lower power in the posterior left atrial wall). Radiofrequency energy at each source was applied per local standard protocol, e.g., moving the catheter within the area indicated by FIRM maps to represent the center of rotation or focal impulse origin (typically 1–2 cm²) until AF terminated or time at that source reached ≈ 10 minutes, whichever came first. Typically, only 3–5 minutes of ablation was required per source. Power was regulated to ensure temperature did not exceed 42°C. The coronary sinus bipolar electrogram with the most discrete electrograms was used to measure AF cycle length (CL)²² as the average over 10-cycles, prior to, then after each step of ablation confirmed by 2 observers.

The primary endpoint of this report was the acute impact of FIRM ablation on AF, represented by the composite of AF termination or AF organization defined as CL prolongation > 10 %. AF organization was included since, when substantial, CL prolongation has been reported to indicate functional change in AF. Our 10% cutpoint for AF CL prolongation by ablation at a single location is more conservative than previous reports²² that used >3–4%, or 6 ms prolongation at single locations. If AF terminated, attempts were made to reinitiate AF and, if AF was reinduced, FIRM maps were regenerated and FIRM ablation repeated until each source area had been ablated or until AF was not reinducible, whichever came first (figure 1).

Conventional ablation⁹ was then performed *after* FIRM ablation, and comprised pulmonary vein isolation verified using a circular catheter (Lasso™, Biosense-Webster, Diamond Bar, CA or Optima, St Jude Medical, Minnetonka, MN). Ablation power, temperatures and duration were as noted above. Based on operator preference, other clinically indicated ablation performed after the measurements in this report included a left atrial roof line (in persistent AF) or ablation of atrial tachycardia or flutter if observed. Defragmentation²³ was not performed. If AF persisted after completion of the ablation protocol, the procedure concluded with cardioversion.

Statistical Analysis

Continuous data are represented as mean ± standard deviation (SD). The Student t-test was used to compare continuous variables between 2 groups, such as times to termination, ablation delivery, AF cycle lengths and other parameters. Paired continuous variables were compared using linear regression and the paired *t*-test. The chi-square test was applied to contingency tables for categorical variables. A p-value of < 0.05 was considered statistically significant.

RESULTS

Patient characteristics are summarized in Table I. As shown in Table 2, technical issues in 2 of these first 14 cases (unavailability of the appropriate basket size, and connection difficulties with the electroanatomic mapping system, not the FIRM system) prevented FIRM mapping in these 2 cases.

Localized Sources for AF Were Present in All Patients

As indicated in Table 2, localized sources were demonstrated in all mapped patients in our series for an average of 1.9 ± 0.8 sources per patient. Of 23 AF sources in this series, (21 rotors, 2 focal sources), 18 lay in left atrium and 5 in the right atrium (figure 7). All sources were stable for at least 20–30 minutes during mapping and remapping.

Figure 3 shows AF in a 55-year-old man (patient 5), characterized by a single clockwise rotor (red to blue) in the mid-left atrial roof. The single rotor is consistent with relatively organized AF electrograms (CL 160–180 ms). The supplemental movie indicates that the center of rotation showed limited movement (spatial precession). This explains why electrograms at any electrode, such as near the rotor center, may vary due to rotor precession through and away from this site.

Rotors from individual patients are illustrated in figures 3–6, and described below in the context of FIRM ablation.

Acute Results of FIRM Ablation

FIRM ablation achieved the acute endpoint of AF termination ($n=8$, 67% of mapped patients) or organization ($>10\%$ slowing) ($n=4$) in all patients (table 2). AF termination was achieved with 4.9 ± 3.9 minutes of ablation at the primary rotor (i.e., site of termination), and for a total FIRM ablation time at all sources in all patients was 12.25 ± 8.59 minutes (all sources/patient). In the patient shown in figure 3, FIRM ablation was delivered based on propagation movies (of which one frame is summarized by the isochronal map in figure 3B). FIRM ablation delivered for 4 minutes at 35–40 W at the LA roof terminated AF directly to sinus rhythm (figure 3C). Although AF had previously sustained for >90 minutes, AF was now noninducible despite aggressive burst pacing to CL 140 ms and high dose isoproterenol infusion. As part of the clinical protocol, the pulmonary veins were isolated (in sinus rhythm).

Figure 4 illustrates paroxysmal AF in a 60-year-old man (patient 9), characterized by a single electrical rotor in the left atrium with clockwise activation from red (early) to blue (late) at CL 160 ms. As organized activation breaks down to fibrillation, distal regions activate continuously and asynchronously (not 1:1), and thus may time ‘early’ or ‘late’ with continuously varying wavebreak away and/or collision towards the rotor (double white lines). This isochronal snapshot thus indicates local activation from the rotor, with wavebreak/collision in peripheral atrial regions. FIRM ablation commenced at the lateral left atrial rotor prior to any other ablation. FIRM ablation terminated AF directly to sinus rhythm, without any intervening atrial tachycardia, and rendered AF noninducible despite aggressive burst pacing (CL 150 msec) and high dose isoproterenol for over an hour after the FIRM ablation. The nearby left superior pulmonary vein as well as the other pulmonary veins were subsequently isolated.

Figure 5 illustrates extremely rapid AF (CL 135 ms) in a 42-year-old man (patient 13) characterized by asynchronous left atrial rotors. FIRM ablation for 5 minutes at left superior and lateral rotors slowed AF by 11% from CL 136 to 151 ms. FIRM ablation at 40 W power at the septal rotor (just posteroinferior to the right inferior pulmonary vein) abruptly terminated AF from CL 151 to sinus rhythm in less than 1 minute. AF had been sustained for >2 hours prior to FIRM mapping.

Figure 6 illustrates a complex spatial activation during AF in a 74-year-old man (patient 14) with continuous AF for over 9 months and left atrial diameter 75 mm on intracardiac imaging (despite smaller preprocedural estimates). Supplemental movie 2 shows these complex dynamics during AF for multiple cycles. This isochronal snapshot indicates high

and low posteroseptal rotors, with asynchronous peripheral activation. FIRM ablation of both rotors slowed AF from CL 178 ms to 220 ms in 7 minutes. FIRM ablation at the septal LA focal source (3rd source), terminated AF to an organized atrial tachycardia (CL 240 ms, fig 5B) in less than 2 minutes, for a total FIRM ablation time of < 9 minutes.

Anatomical Locations of AF Sources

As seen in table 2, AF sources lay at unique patient-specific locations throughout both atria. Figure 7 shows that left atrial sources (n=18) lay adjacent to the pulmonary vein antrum (7/18, 39%), the posterior wall/coronary sinus (5/18, 28%), roof/anterior location (6/18, 33%). Right atrial sources (n=5) lay both lateral and septal. In some cases lateral right atrial sources were adjacent to the phrenic nerve which precluded complete ablation (Table 2). Of note, FIRM ablation achieved the acute endpoint in all patients, supporting the functional role of these sources.

Lack of Acute Endpoint and Left Atrial Size

There was an observed relationship between the absence of an acute endpoint by FIRM ablation and marked atrial dilatation (with incomplete atrial mapping). Of the 2 patients who did not achieve the endpoint both had very dilated left atria on intraprocedural imaging (patient 2, 86 mm and patient 10, 61 mm.). Although sources were found in each, a primary driver may have been present in unmapped atrial regions.

DISCUSSION

This is the first prospective multicenter experience of FIRM-guided mapping and ablation of AF in patients with very diverse presentations. The major findings of this study are: (i) Computational FIRM mapping readily identified localized rotors and focal beat sources for AF in all patients thus providing a clear ablation target; (ii) FIRM ablation for an average of about 12 minutes terminated or substantially slowed AF in all mapped patients, prior to any other ablation. The sources responsible for AF lay in a variety of patient-specific locations in the left and right atria without any characteristic distributions (Figure 7). While some locations may have been incidentally targeted by ‘empirical’ anatomical lesions sets, many would not. This emphasizes the importance of patient-specific AF mapping. Although we are in the process of accumulating long-term outcome results, these results suggest that FIRM ablation may be an effective technical strategy for patient-tailored AF ablation.

Localized Sources for Human AF

These results support a recent report¹⁶ that biatrial computational mapping of AF reveals rotors and focal beat sources for human AF. As also shown in a recent video case study²⁴, detection of localized rotors with subsequent targeted FIRM ablation was able to acutely terminate AF and render it noninducible. Sources in the first multicenter experience were sustained for multiple cycles and showed limited movement (precession) but remained localized within a restricted domain (1–2 cm², or ≈2–3% of the atrial surface), as demonstrated by multiple maps over tens of minutes during mapping. The primary role of sources in causing AF is supported by the remarkable finding of acute AF termination and noninducibility by brief FIRM ablation. The etiological role of sources is supported by activation maps (figures 3–6) in which sources ‘controlled’ local activation with patient-specific and variable breakdown of wavefronts leading to AF. These results provide the first human validation of elegant experimental studies using dye-fluorescence imaging of AF in isolated sheep^{25–27} or canine²⁸ atria to identify rotors that had similar characteristics.

Ablation at Sources Terminated Human AF

In our study, FIRM ablation during AF at specific locations specified *a priori* from FIRM maps, prior to any other ablation, acutely terminated (8/12) or organized (3/12) AF in all patients. These results substantially extend prior animal studies by demonstrating that brief FIRM ablation to eliminate sources can terminate or organize human AF. Chou et al.²⁸ recently ablated rotors in canine AF, demonstrating that such ablation suppressed AF inducibility.

Further work is required to define the precise mechanistic basis of acute AF termination by FIRM. One likely possibility is that FIRM truncates rotor precession, organizing AF towards an atrial tachycardia that is then eliminated by continued FIRM ablation. Although it is unclear why some patients exhibited only AF organization (>10% slowing) without termination, these patients had larger atria and it is likely from our experience that additional sources lay in incompletely mapped atrial regions. Other cases of AF slowing may reflect the fact that in some cases, sources were incompletely ablated due to esophageal heating or proximity to the phrenic nerve (table 2).

Patients in our series are still undergoing follow-up, yet these results support data that FIRM ablation could be a promising approach for patient-tailored AF ablation¹⁷. Other notable studies have suggested that AF termination by conventional ablation (although rarely subsequently tested for non-reinducibility), portends freedom from AF on long-term follow-up.²⁹ AF termination occurred rapidly and predominantly to sinus rhythm by FIRM ablation contrasting with the long ablation times and termination predominantly to atrial tachycardia via traditional ablation.^{10, 29} Slowing of AF is an interesting outcome and it has been hypothesized this may be due to elimination of secondary sources of AF that are still relevant for long term clinical benefit.¹⁷ The CONFIRM trial showed 31% of patients in the FIRM guided arm had slowing of AF as a procedural endpoint did well on follow-up.¹⁷ In these patients it is likely that there were rotor and sources in unmapped regions of the atria. Since AF typically initiates from sinus rhythm rather than from sustained atrial tachycardia, AF termination to sinus rhythm likely indicates a different mechanism of termination that requires further study. By limiting the extent of RF application to the atria and to limited atrial territory ($\approx 2\text{--}3\%$ of the atrium), FIRM mapping intuitively may help limit development of post ablation macroreentrant tachycardias that follows more aggressive ablation techniques.

Limitations

Despite pre-procedural atrial diameter <55 mm, many patients had atria larger than the largest available basket (60 mm diameter) that limited mapping. Two patients could not be mapped due to technical connectivity or basket availability issues that should be resolved with continued experience. In 2 patients (numbers 10 and 11), AF slowed by FIRM ablation after 10 and 15 minutes at the primary source (i.e., where the effect was noted), respectively, total FIRM time is analogous to that seen in other non-PV ablation approaches. Thus, it is possible that additional sources in unmapped (septal left atrial) regions or other mechanisms may have caused slowing in these cases. Current basket catheters may significantly limit optimal mapping, but RhythmViewTM reduces the impact of its spatial resolution (4 mm along splines, larger between splines) by substantial noise reduction and interpolation. This is likely be less relevant for ablation targeting than mechanistic discussions since the size of each ablation lesion ($\approx 5\text{--}7$ mm) defines the practical spatial resolution required for targeting. Varying interspline distances are handled using algorithms based upon physiological atrial conduction velocities²¹ to define plausible propagation paths.¹⁶ Although this is a small series, it may be argued that each case of AF termination by localized ablation at an *a priori* predicted site is a remarkable event. It may also be argued

that any AF may terminate spontaneously, yet AF in our patients was sustained for hours prior to acute termination by FIRM. These results argue for wider testing of FIRM-guided AF ablation. Our patients are early in their follow-up, but this study was designed to address the characteristics of AF sources and the acute impact of FIRM guided ablation at these sources.

Conclusions

FIRM guided ablation revealed localized rotors/focal sources in all patients during AF, where brief targeted Focal Impulse and Rotor (FIRM) ablation at *a priori* identified sites during AF terminated or organized AF in all cases prior to any other ablation. Many of these FIRM-identified sites would not have been targeted using empiric anatomical lesion sets. Notably, this report represents the first FIRM-guided cases at each of our respective Institutions, and results may improve with further experience. FIRM guided mapping and ablation of AF may have substantial value in shortening procedural times, providing patient-tailored lesions and limiting ablation volume, and thus could potentially improve the long-term success of AF ablation procedures. Clearly, further larger studies with follow-up are required, as well as randomized clinical trials versus conventional techniques.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
FIRM Guided Ablation Work Flow (in lab)

Figure 2. Fluoroscopic Views of Basket Data Acquisition in
(A) Right Atrium, RAO 30° projection with circular catheter in left atrium, multipolar catheters in the coronary sinus and the His position. **(B) Left atrium, LAO 30° projection**

Figure 3. FIRM at Left Atrial Rotor Terminates Paroxysmal AF To Sinus Rhythm in 4 minutes (patient 5)

(A) AF with CL 180 ms, illustrating sequential activation at electrode locations spanning the rotor center. (B) Left atrial roof rotor represented in this isochronal snapshot by clockwise activation sequence (red to blue). Clinical mapping was guided by the propagation movie (Supplemental movie 1) that shows this spiral wave with slight movement (precession) of its center of rotation bounded by 1–2 electrodes. **(C) FIRM at Rotor alone terminates AF to Sinus Rhythm in 4 minutes, prior to any other ablation.** AF was now noninducible despite burst pacing and high dose isoproterenol.

Figure 4. Left Atrial Rotor During Human Atrial Fibrillation, Where FIRM Ablation Terminates AF to Sinus Rhythm (patient 9)

(A) Isochrones show AF Rotor with activation sequence from red to blue (CL 160 ms, clockwise). Note wavebreak and collision within the left atrium indicating breakdown of 1:1 activation from rotor (double white lines). **(B) AF Termination to sinus rhythm by FIRM ablation** (10 minutes). Panels **C** (Ablation catheter and basket in left atrium) and **D** (circular PV catheter and ablation catheter) LAO view

Figure 5. FIRM Terminates AF to sinus rhythm (patient 13)

(A) Isochrones show 2 small rotors in the left atrium during AF, both with small space constants in the superior and inferior septal left atrium. **(B) FIRM Ablation at the inferoseptal LA rotor, posteroinferior to the right inferior pulmonary vein, at 40W** rapidly terminated AF to sinus rhythm in less than one minute. AF was now non-inducible.

Figure 6. FIRM Terminates AF (CL 178 ms) in this patient with persistent AF and LA diameter 75 mm (patient 14)

(A) Isochrones show 2 rotors in the left atrium and one focal source (indicated).

Supplemental movie 2 illustrates these complex dynamics, and was used to guide FIRM ablation. FIRM at each rotor prolonged AF CL from 178 to 220 ms. **(B) FIRM Ablation at the focal beat** terminated AF to atrial tachycardia (CL 240 ms). Total FIRM time was 10 minutes, prior to pulmonary vein or any other ablation.

Figure 7. Schematic showing locations of Rotors and Focal sources in 12 patients

Key: RA, LA, right and left atria; MV, mitral valve; TV, tricuspid valve; RAA, LAA, right and left atrial appendages; LSPV, LIPV, left superior and inferior pulmonary veins; RSPV, RIPV, right superior and inferior pulmonary veins; SVC, superior vena cava; IVC, inferior vena cava. Rotors were defined as areas where activation completed multiple clockwise or counterclockwise rotations. Focal beats were identified as sites with centrifugal activation to surrounding atrium, and their origins identified at the electrodes where activation initiated. For both definitions the pattern needed to be sustained over multiple 4–8 second epochs.

Table 1

Clinical Characteristics of Population

Pt ID	Age	Gender	AF type	Largest LA size/mm	Prior Ablation
1	53	M	LSPeAF	50	Y
2	60	M	LSPeAF	86	Y
3	60	M	PeAF	60	N
4	72	F	PeAF	56	Y
5	55	M	PAF	40	N
6	39	M	PeAF	43	N
7	60	M	PeAF	80	N
8	75	F	LsPeAF	59	Y
9	60	F	PAF	55	N
10	62	M	PeAF	61	N
11	39	M	PeAF	39	Y
12	59	M	PAF	52	Y
13	42	M	PAF	54	N
14	74	M	peAF	75	Y
Average	58±12	3F/11M	3 PAF 11 PeAF (including 3 LSPeAF)	58±14	

Key: PAF, Paroxysmal AF; PeAF, Persistent AF; LSPeAF, Longstanding Persistent AF Pts 8, 12 and 14 were on amiodarone

Table 2

AF Sources and Acute Response to FIRM Ablation

Pt	AF CL, ms	No. of Sources	Source Chamber	Source Location	FIRM Time, min	Impact of FIRM	Time @ Final site, min	Endpoint
1	162	2	RA	Posterior	5	Term to flutter	5*	TERM
			LA	Post/Lat LSPV	5	Term to sinus		
2	185	1	LA	Posterior roof	<10	11% AF Slowing, CL 180>200	10	SLOW
3	204	2	RA	Anterolateral	<10	13% AF Slow, CL 204>230	0.5*	TERM
			LA	Post/Roof LSPV	0.5	Term to sinus		
4	231	2	LA	LA Postseptal (RSPV)	5	8% Slow, 231>250	7*	TERM
			RA	Septal	7	Term to sinus		
5	176	1	LA	LA Mid Roof	4	Term to Sinus	4*	TERM
6	BASKET MISMATCH – NOT FIRM MAPPED							
7	205	2	RA	Mid Posterior	-	Not Ablated – Phrenic	8	SLOW
			LA	Lateral Roof/LSPV	8	15% AF Slowing, CL 205>235		
8	172	1	LA	Low Posterolateral focal source (near LIPV)	<10	Term to sinus	10*	TERM
9	163	1	LA	Lateral roof (near LSPV)	<10	Term to Sinus	10*	TERM
10	178	3	LA	Lateral roof	<10	No change	10	SLOW
			LA	Septal Roof	<10	No change		
			LA	Septo Inferior	<10	10% Slow, 178>195		
11	178	2	RA	Midposterior	15	No change	15	SLOW
			LA	Post/Septal (near RIPV)	15	13% Slow, 178–205		
12	CONNECTION ISSUES – NOT FIRM MAPPED							
13	136	3	LA	Posterior Roof	1	11% Slow, 136>151	0.5*	TERM
			LA	Antero Mitral	4	No change		
			LA	Inferoseptal near RIPV	0.5	Term to sinus		
14	178	3	LA	Ant/lateral rotor	5	24% Slow 178>220	2*	TERM
			LA	Post/lat rotor	2	8% Slow, 220–240		
			LA	Mid posterior focal source	2	Term to AT (CL 250)		

Pt	AF CL, ms	No. of Sources	Source Chamber	Source Location	FIRM Time, min	Impact of FIRM	Time @ Final site, min	Endpoint
	181±24	1.9±0.8	RA RA 5 LA 18	Posterolateral	5 12.3±8.6	Term to AT	*4.9±3.9	12 (100%) ENDPOINT 8 TERM 4 SLOWING

Key: LA=left atrium, RA= right atrium, LIPV=left inferior pulmonary vein, LSPV=left superior pulmonary vein AF=atrial fibrillation, CL=cycle length, AT=atrial tachycardia, TERM=termination, Among patients who had previous ablations pts 1,11 and 14 showed no PV reconnection and had FIRM only ablation, pts 2,4 and 8 showed PV reconnection and underwent PVI post-FIRM,

* average time at sites of AF termination