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Association of Left Atrial Function with Incident Atypical Atrial Flutter Following Atrial Fibrillation Ablation

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Abstract

Background—Symptomatic left atrial (LA) flutter (LAFL) is common following atrial fibrillation (AF) ablation.

Objective—To examine the association of baseline LA function with incident LAFL following AF ablation.

Methods—The source cohort included 216 patients with cardiac magnetic resonance (CMR) prior to initial AF ablation between 2010 and 2013. Patients who underwent cryoballoon or laser ablation, patients with AF during CMR, and those with suboptimal CMR, or missing follow-up data were excluded. Baseline LA volume and function were assessed by feature-tracking CMR analysis.

Results—The final cohort included 119 patients (age 58.9±11 years, 76.5% male, 70.6% paroxysmal AF). During a median follow-up of 421 (interquartile range 235–751) days, 22 (18.5%) patients developed LAFL. Baseline LA volume was similar between the two groups. In contrast, baseline reservoir, conduit and contractile function of the LA were significantly impaired in patients with incident LAFL. Baseline global peak longitudinal atrial strain (PLAS) < 22.65% predicted incident LAFL with 86% sensitivity and 68% specificity (C-statistic 0.76). In a multivariable model adjusting for age, heart failure, and LA volume, PLAS (HR 0.9 per % increase in PLAS, p=0.003) and LA linear lesions (HR 2.94, p=0.020) were independently

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associated with incident LAFL. Coexistence of PLAS<22.65% and linear lesions was associated with 9-fold increased hazard of incident LAFL.

Conclusion—Baseline LA function and linear lesions were independently associated with incident LAFL after AF ablation. Linear lesions should be limited to selected cases, especially in patients with impaired LA function.

Keywords

Atrial fibrillation; atrial flutter; cardiac magnetic resonance; left atrium; pulmonary vein isolation

Introduction

Atrial fibrillation (AF) is the most common arrhythmia and is associated with significant symptoms, and mortality (1). Catheter-based AF ablation has become a well-established management strategy for symptomatic AF (2). Despite technological advances in AF ablation, evolving strategies and experience, arrhythmia recurrences remain relatively high (3, 4). Left sided atrial flutter (LAFL) accounts for a substantial proportion of symptomatic sustained recurrences after AF ablation (2, 5–8). Previous studies have reported clinical and procedure-related risk factors of post-ablation LAFL, including linear lesions, incomplete pulmonary vein isolation (PVI), persistent AF, and structural heart disease (2, 7–10).

In recent years, feature-tracking cardiac magnetic resonance (CMR) has emerged as a sophisticated tool for detailed chamber specific functional analysis (11). It has been shown that left atrial (LA) function predicts recurrences after AF ablation (12). In this study, we aimed to define feature tracking CMR-derived LA functional characteristics of patients who developed LAFL after AF catheter ablation.

Methods

Study Population

The source cohort included 216 consecutive AF patients who underwent CMR prior to initial AF ablation between 2010 and 2013. Of these, 37 patients were excluded due to cryoballoon or laser ablation. Patients with AF at the time of CMR (n=46), suboptimal/missing cine images (n=8), and missing follow-up data (n=6) were also excluded. Clinical characteristics of the final cohort, which included 119 patients, were extracted by review of electronic medical records. The Johns Hopkins Institutional Review Board approved the study and all subjects provided written informed consent.

CMR Protocol

Pre-procedural (average 4 days before ablation) CMR was performed using a 1.5-Tesla MR scanner (Avanto and Aera, Siemens, Erlangen, Germany) equipped with a phased array cardiac coil. Vertical and horizontal long-axis cine CMR was performed using a steady-state free precession sequence (minimal TR/TE, 8 mm slice thickness, 2 mm spacing, 78° flip angle, 36–40 cm field of view) with 1.5 x 1.5 mm in-plane resolution.

Feature Tracking CMR

The Multimodality Tissue Tracking software (MTT; version 6.0, Toshiba, Japan) was used to measure phasic LA volumes, strain and strain rate on vertical and horizontal long-axis cine images. After manual contouring of end systolic endocardial and epicardial LA borders, the software automatically tracked pixel features within the tissue throughout the cardiac cycle as previously described (13). In all cases, pulmonary veins and LA appendage were excluded from the contours. Manual adjustments were performed when tracking was suboptimal. The software generated a volume curve for each phase by using the biplane area-length method (Figure 1). Maximum, pre-contraction, and minimum LA volumes were obtained from the phasic volume curve to calculate the following functional parameters:

- Total LA emptying fraction: $100 \times (\text{Maximum LA volume} - \text{minimum LA volume}) / \text{Maximum LA volume}$
- Passive LA emptying fraction: $100 \times (\text{Maximum LA volume} - \text{pre-contraction LA volume}) / \text{Maximum LA volume}$
- Active LA emptying fraction: $100 \times (\text{Pre-contraction LA volume} - \text{minimum LA volume}) / \text{Pre-contraction LA volume}$

Global LA longitudinal strain and strain rate curves were calculated as the mean longitudinal strain and strain rate of all segments (Figure 1). Peak longitudinal LA strain (PLAS), peak systolic LA strain rate (SR-s), peak early diastolic LA strain rate (SR-ed) and peak late diastolic LA strain rate (SR-ld) were measured. The SR-ed and SR-ld were presented as absolute values. Reservoir function was represented by PLAS, SR-s and total LA emptying fraction; conduit function was represented by passive LA emptying fraction and SR-ed; and contractile function was represented by SR-ld and active LA emptying fraction. We previously reported on the reproducibility of CMR derived LA functional parameters using MTT software (14).

Catheter Ablation

All patients underwent radiofrequency ablation as previously described (15). After obtaining double atrial transseptal puncture, electroanatomic mapping (CARTO, Biosense-Webster, Diamond Bar, CA) of the LA endocardium was performed with registration onto a previously acquired CMR image. An irrigated 3.5 mm tip catheter (Thermocool, Biosense-Webster, Diamond Bar, CA) was used for mapping and ablation. A circular multipolar electrode-mapping catheter (Lasso, Biosense, Diamond Bar, CA) was used to confirm PVI by presence of entrance and exit block. Additional linear lesion sets, connecting the right and left pulmonary venous antra across the LA roof, floor or posterior wall, and complex fractionated atrial electrogram (CFAÉ) ablations were performed per operator discretion. High-frequency stimulation for identification of ganglionated plexi was not performed. However, transient >50% prolongation of the RR interval and sudden >20 mmHg transient decreases in systolic radial artery pressure were noted as vagal responses consistent with ganglionated plexus ablation. Repeat PVI was performed during follow-up depending on symptoms and clinician's preference. During the repeat procedure the diagnosis and characteristics of LAFL were confirmed based on activation and entrainment mapping. Sites with fractionated potentials that participated in the LAFL circuit were targeted for ablation.

Follow-up

After the procedure, patients were observed for 24 hours. In the absence of AF recurrence, pre-procedurally ineffective antiarrhythmic medications were discontinued after 3 months. Symptom prompted 24-hour Holter monitoring and scheduled electrocardiography at 3, 6, and 12 months were performed. Incident LAFL was defined as symptomatic or asymptomatic documented LAFL > 30 seconds in duration. In the absence of electrophysiology study, an LAFL diagnosis was made on the basis of agreement between two expert reviewers, masked to clinical and imaging information. The readers diagnosed LAFL if a) high frequency (180–260 ms) F waves with continuous oscillation without a flat baseline were noted, and b) the F wave morphology was not suggestive of typical cavotricuspid isthmus dependent AFL (sawtooth pattern in inferior leads, positive F-waves in lead V1, isoelectric or negative in leads V5–V6). Patients without LAFL were censored at the time of last available follow-up.

Statistical Analysis

The distribution of continuous variables was evaluated using the Shapiro-Wilk test. Continuous variables were expressed as mean $\pm$ standard deviation or median (interquartile range [IQR]) and compared using the Student's *t*-test or Mann Whitney U test as appropriate. Categorical variables were expressed as number (percentages) and compared using the χ^2 or Fisher exact tests. The association between LA volume and LA functional parameters was evaluated using Spearman correlation and graphically assessed using a scatter matrix plot. The optimal threshold for PLAS to estimate LAFL free survival was selected by maximizing the area under the receiver-operating characteristic (ROC) curve. Kaplan-Meier survival curves were used to assess LAFL free survival in patient subgroups based upon the optimal PLAS threshold. Univariable and multivariable Cox proportional hazards models were used to examine the association of clinical and functional independent variables with time to incident LAFL. Model proportionality assumptions were verified using Schoenfeld residuals. Statistical analyses were performed using SPSS software version 22.0 (IBM Corp., Armonk, NY).

Results

The final cohort included 119 patients. Mean age was 58.9 ± 11 years, 76.5% were male, and 70.6% had paroxysmal AF. The detailed baseline characteristics are shown in Table 1. During a median follow-up of 421 days, 22 (18.5%) patients developed incident LAFL. Median time to LAFL incidence was 198 (IQR 42 – 409) days. History of heart failure was more common in patients with LAFL. Among the entire cohort of 119 patients included in this report, 28 patients (23.5%) received linear ablation lines at the initial AF ablation. The most common linear lesion was a roof line, followed by a posterior line, both of which connected the left to right sided PVI lesion sets. Of the 28 patients with linear lesion sets, 11 (39%) developed LAFL during follow-up. LAFL was less commonly observed among patients who did not have linear lesions created during the initial ablation (11/91, 12%, $p=0.001$). Overall, 2 patients had CFAÉ ablation, and both patients were LAFL free during follow-up. Significant vagal pause consistent with ganglionated plexus ablation was noted in 13 patients. There was no difference in occurrence of LAFL in these patients (Table 1).

Baseline LA volume and functional parameters are shown in Table 2. LA volume was similar in patients with and without incident LAFL. In contrast, all baseline LA functional parameters were lower in patients with LAFL, indicating impaired LA function in all phases (reservoir, conduit and contractile functions). The association of LA volume and LA functional parameters is displayed in Figure 2. Due to significant co-linearity among all phasic LA functional parameters, PLAS was used as a surrogate of LA function. In the multivariable model adjusted for age, heart failure, and LA volume; PLAS (HR 0.9 per % increase in PLAS, CI 0.84–0.96) and linear ablation lesions (HR 2.94, CI 1.23–7) were the only predictors of LAFL.

Management of the patients with incident LAFL is summarized in Table 3. Of the 22 patients with LAFL, 14 had repeat ablation. Of the 14 that underwent repeat ablation, 4 only underwent repeat PVI, whereas 10 underwent activation and/or entrainment mapping with demonstration of macro-reentry. The most common circuit was peri-mitral macro-reentry (60%). The remaining 8 patients underwent cardioversion and have been maintained on antiarrhythmic drug therapy.

Baseline PLAS < 22.65% predicted LAFL with 86% sensitivity and 68% specificity (C-statistic 0.76; Figure 3). Patients were divided into 2 groups based on this threshold. Median time to LAFL was: 152 (IQR 97–206) days in patients with PLAS ≥ 22.65% and linear lesions, 190 (IQR 40–298) days in patients with PLAS < 22.65% and linear lesions; and 308 (IQR 73–700) days in patients with PLAS < 22.65% and without linear lesions. Figure 4 demonstrates Kaplan Meier curves of LAFL free survival among PLAS < 22.65% and PLAS ≥ 22.65% patient subgroups further stratified by the presence or absence of linear lesion sets. Coexistence of PLAS < 22.65% and linear lesions was associated with 9-fold increased hazard of LAFL incidence.

Discussion

Major Findings

To the best of our knowledge, this is the first study to examine the association of baseline LA functional properties with incident LAFL after AF ablation. We showed that 1) baseline LA functional parameters were lower in patients with LAFL, indicating impaired function in all phases; 2) LA function was an independent predictor of LAFL along with linear lesions; and 3) the co-existence of linear lesions and impaired LA function was associated with the highest risk of LAFL.

LAFL following AF ablation

LAFL may comprise up to 50% of all recurrences after ablation (2). The risk has been reported to be low after cryoballoon ablation; however, the incidence may rise to 25% after radiofrequency ablation (7, 8). Patients with LAFL often present with more severe symptoms, including the development of a rate-related cardiomyopathy, reflecting the challenges of effective rate control in these patients (5, 6).

Post-ablation LAFL has been mainly attributed to sequelae of LA linear lesions; but may also results from ablation-induced conduction slowing, gaps in the ablation lines or

organization of AF into a reentry circuit (5, 8, 16). According to our results, LA function is associated with incident post-ablation LAFL independent of additional lesion sets. Fibrosis and LA remodeling clearly play a role in the pathogenesis of AF recurrences (17). Our data highlight the association of LA remodeling and LAFL. Fibrosis likely serves as a substrate for slow conduction and creates anatomical obstacles that enable the development of a critical isthmus. In a previous study, a significant association was found between lower PLAS and the presence of low voltage regions (18). Similarly, LA contractile function is associated with the extent of low voltage (< 0.5 mV) (19). Therefore, non-invasive assessment of LA function prior to AF ablation procedure may be used as an indicator of the extent of fibrosis.

Previous studies reported early presentation of LAFL after AF ablation (8). Due to the relatively long follow-up in our study, a subset of patients with very late recurrences after AF ablation, defined as >12 months, were noted. In this subset, the prevalence of pulmonary vein reconnection was significantly lower, and impaired baseline left ventricular systolic and diastolic function was more common, suggesting that patient-related rather than procedure-related factors may primarily account for late LAFL (20).

The 2012 HRS/EHRA/ECAS Consensus Document on AF ablation states that for patients with persistent AF, additional substrate ablation either targeting of CFAE or additional linear lesion sets may be appropriate (2). These recommendations are likely to evolve following the recent STAR AF II trial, which revealed fewer recurrences in the PVI versus the PVI plus substrate modification group (21). In this trial, the investigators concluded that extensive ablation may cause arrhythmogenic areas and that persistent AF patients do not derive benefit from linear lesions. Our findings are consistent with the results of the STAR AF II trial and suggest that linear lesion sets are particularly arrhythmogenic in patients with pre-existing LA dysfunction.

Association of LA fibrosis with LA function

Reservoir function represents distensibility and is closely associated with the extent of interstitial fibrosis. A previous study evaluated patients prior to mitral valve surgery with speckle-tracking echocardiography and examined LA tissue samples obtained at cardiac surgery (22). Histopathological analysis of tissue fibrosis was compared with pre-surgical PLAS. In patients with moderate ($PLAS < 20\%$) and severe ($PLAS < 10\%$) LA dysfunction, the extent of the fibrosis was more than 50% and 70%, respectively. Likewise, there was an inverse relationship between LA fibrosis as estimated on late gadolinium enhancement (LGE) CMR with LA strain and strain rate (23). Our group has shown similar associations between the extent of LA LGE and LA function, suggesting that both of the CMR-derived assessments can be used as non-invasive surrogates of LA fibrosis (14). The association of LGE extent with LAFL incidence post ablation warrants future study. However, in contrast to LGE-CMR, which is not available in many centers and requires extensive technical expertise for image acquisition and analysis, LA functional analysis is easy to perform and widely available via echocardiography, MRI, and phasic computed tomography.

LA function and outcome

In recent years, LA deformation studies have revealed strong associations between clinical outcomes and LA function in the general as well as AF populations (13, 24–27). In AF patients, AF recurrence is closely associated with baseline LA function (28, 29). In routine practice, LA volume is frequently used to predict LA dysfunction and remodeling, however, LA deformation indices are more sensitive and their impairment can be detected before dilatation occurs. According to our results, unlike LA volume, baseline LA function analysis (particularly reservoir and contractile function) provides useful adjunctive information and may help to optimize management to prevent post-ablation LAFL. In our study, we only included patients that were in sinus rhythm at the time of CMR. Recent data suggest that LA strain can be measured via speckle tracking echocardiography in patients with non-sinus rhythm, albeit with analyses limited to the reservoir phase (30). The presence of AF prior to ablation, however, may confound not only the measurement of LA strain, but also the association of that measurement with outcomes following ablation. Therefore, further studies are necessary to examine the association reservoir function during AF with outcomes following ablation.

Limitations

This is a single center study and patients with rhythm other than sinus at the time of baseline CMR were excluded; therefore, our results may not be generalizable to all patients with AF and study power was limited for multivariable analyses. In 10 patients, activation mapping and entrainment confirmed LAFL. In others, review of ECG by two experienced reviewers suggested LAFL. We cannot exclude the possibility that the surface ECG may have been misleading in a minority of cases. Finally, differences in LAFL incidence existed compared to some prior studies (5–8), and may reflect variability in patient characteristics, length of follow up, methods for LAFL detection, and procedural techniques.

Conclusions

Baseline LA function and creation of linear lesions is associated with incident LAFL after an initial AF ablation. These findings, combined with the recent results of the STAR AF II Trial, suggest that careful consideration should be given prior to creation of linear ablation lesions to treat AF.

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Abbreviations

AF	Atrial fibrillation
LAFL	Left sided atrial flutter

PVI	pulmonary vein isolation
CMR	cardiac magnetic resonance
LA	left atrial
PLAS	Peak longitudinal LA strain
SR-s	systolic LA strain rate
SR-ed	peak early diastolic LA strain rate
SR-ls	peak late diastolic LA strain rate
CFAE	complex fractionated atrial electrograms
ROC	receiver-operating characteristic
LGE	late gadolinium enhancement

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Clinical Perspectives

This manuscript examines the association of baseline left atrial functional properties with incident left atrial flutter following after atrial fibrillation ablation. Baseline left atrial functional was impaired in all phases in patients with incident left atrial flutter. After adjusting for potential confounders and linear lesions left atrial function remained independently associated with incident left atrial flutter. Importantly, the co-existence of linear lesions and impaired left atrial function was associated with the highest risk of incident left atrial flutter. Our findings are consistent with the results of the STAR AF II trial, which revealed fewer recurrences in the pulmonary vein isolation versus the pulmonary vein isolation plus substrate modification group, and suggest that linear lesion sets are particularly arrhythmogenic in patients with pre-existing left atrial dysfunction.

Figure 1.

Quantitative CMR-derived functional analysis of the LA in a healthy volunteer. Panel A: Tissue tracking at end systolic 4-chamber cine image, pulmonary veins are excluded. Panel B: Tissue tracking at end systolic 2-chamber cine image, the appendage and pulmonary veins are excluded. C: Volume curve of the LA, blue dotted line represents 4-chamber view; green dotted line represents 2-chamber view and pink dotted line represents bi-plane LA volume. The peak volumes during ventricular systole and pre-atrial contraction represent LAV-max and pre-contraction volumes, respectively. B: Global LA longitudinal strain curve, the peak of the curve during ventricular systole corresponds to PLAS. C: Global LA longitudinal strain rate curve, peaks at ventricular systole, early diastole and late diastole correspond to SR-s, SR-ed and SR-ld, respectively.

Figure 2.

Scatter matrix plots illustrate associations between LA volume and LA functional parameters and results of the spearman correlation analysis.

Figure 3.
The receiver operating characteristic curve of baseline PLAS for prediction of post-ablation LAFL.

Figure 4.

The Kaplan Meier survival curves demonstrate the association of LA function and linear lesions with LAFL incidence.

Table 1

Baseline characteristics of patients with (LAFL) and without (No LAFL) left atrial flutter at follow-up.

	LAFL (n=22)	No LAFL (n=97)	P
<i>Clinical characteristics</i>			
Age (years)	59.46±7.9	58.9±11.6	0.8
Male gender	86.4% (19)	74.2% (72)	0.23
BMI (kg/m ²)	29.5 (26.4–32.9)	27.7 (24–31.1)	0.13
Hypertension	59.1% (13)	46.4% (45)	0.28
Diabetes mellitus	18.2% (4)	7.2% (7)	0.12
Coronary artery disease	9.1% (2)	11.3% (11)	0.99
Heart failure	22.7% (5)	7.2% (7)	0.045
Cerebrovascular event	18.2% (4)	8.2% (8)	0.23
Sleep apnea	31.8% (7)	17.5% (17)	0.15
CHA ₂ DS ₂ VASC 2	50% (11)	40.2% (39)	0.4
Persistent AF	27.3% (6)	29.9% (29)	0.81
<i>Procedural characteristics</i>			
Total RF time (minutes)	48.2±15.2	45.2±15.6	0.43
CFAÉ ablation	0% (0)	2.1% (2)	0.66
Vagal Response consistent with GP ablation	13.6% (3)	10.3% (10)	0.71
Linear lesions	50% (11)	17.5% (17)	0.001

AF: Atrial fibrillation, AFL: Atrial flutter, BMI: Body mass index, CFAÉ: Complex fractionated atrial electrograms, RF: Radiofrequency

Table 2

Baseline LA volume and LA functional characteristics

	LAFL (n=22)	No LAFL (n=97)	P
LVEF (%)	55.4±7.8	57.7±5.5	0.21
LAV max (ml)	98.6 (81.7–125.6)	95.5 (76.2–116.4)	0.46
Indexed LAV max (ml/m²)	48.7±13.9	47.9±13.7	0.81
PLAS (%)	18.2 (13.3–22)	25.5 (20.4–32.4)	<0.001
LA SR-s sn⁻¹	0.74 (0.5–1.1)	0.99 (0.8–1.31)	0.004
LA SR-ed sn⁻¹	0.71 (0.52–0.98)	0.95 (0.65–1.34)	0.01
LA SR-ld sn⁻¹	0.81 (0.4–1.14)	1.22 (0.88–1.63)	<0.001
LATEF (%)	37.2±12.8	46±9.6	<0.001
LAPEF (%)	16.7±6	19.3±6	0.08
LAAEF (%)	25.1±12.2	33.3±9.2	<0.001

AF: Atrial fibrillation, EF: Emptying fraction, LA: Left atrium, LVEF: Left ventricular ejection fraction, LAAEF: Active left atrial emptying fraction, LAPEF: Passive left atrial emptying fraction, LATEF: Total left atrial emptying fraction LAV max: Maximum left atrial volume, PLAS: Peak left atrial longitudinal strain, SR-s: Systolic strain rate, SR-ed: Early diastolic strain rate, SR-ld: Late diastolic strain rate

Table 3

Baseline characteristics and management of each case with LAFL

Cases	Clinical Characteristics	PLAS (%)	iLAV (ml/m ²)	Linear Lesion	Time to LAFL (Days)	Management
1	61 y/o F, Parox AF	20.9	41.9	Roof	64	Cardioversion
2	55 y/o M, HTN, Parox AF	21.6	37.9	None	582	Medical management
3	58 y/o F, h/o CVA, OSA Parox AF	9.8	71.3	Roof	44	Cardioversion, repeat PVI
4	56 y/o M, HTN, DM, OSA, Parox AF	18.6	34.1	None	966	Repeat PVI, posterior mitral annulus ablation
5	69 y/o M, HTN, CVA, Parox AF	17.7	37.1	Roof	671	Repeat PVI, mitral isthmus flutter ablation
6	55 y/o M, HTN, CHF, Parox AF	15.9	58.7	Posterior	258	Cardioversion
7	62 y/o M, HTN, DM, Parox AF	17.8	59.9	None	37	Repeat PVI, perimitral flutter ablation
8	58 y/o M, HTN, Parox AF	21.7	36.2	Roof	338	Repeat PVI, perimitral ablation
9	46 y/o M, OSA, Pers AF	19.3	63.7	None	1044	Cardioversion
10	69 y/o M, DM, CHF, Pers AF	21.9	54.3	Roof	36	Cardioversion
11	64 y/o M, Pers AF	11.9	60.2	None	103	Cardioversion
12	55 y/o M, HTN, CVA, Parox AF	22.4	44.9	None	351	Repeat PVI, roof line terminated flutter
13	77 y/o M, HTN, CAD, CHF, OSA, Pers AF	10.7	48.8	None	264	Repeat PVI, peri LPV and mitral isthmus flutter
14	52 y/o M, HTN, Parox AF	16.3	49.7	None	611	Repeat PVI, LSPV ablation terminated flutter
15	52 y/o M, DM, CAD, CHF, OSA, Parox AF	16	32.9	None	20	Cardioversion, repeat PVI
16	65 y/o M, OSA, Parox AF, MVr, AVR	34.2	44.8	None	21	Repeat PVI, roof line terminated flutter
17	68 y/o M, HTN, Parox AF	12.8	81.3	None	85	Repeat PVI, roof and perimitral ablation
18	48 y/o F, HTN, Parox AF	28.1	60.8	Posterior	97	Repeat PVI, cardioverted before mapping, hypotensive
19	53 y/o M, HTN, CHF, CVA, OSA, Pers AF	13.5	23.3	Roof	207	Cardioversion
20	57 y/o M, HTN, Parox AF	9.4	40.1	Roof	190	Cardioversion, repeat PVI
21	61 y/o M, Pers AF	41	46.3	Posterior	206	Cardioversion
22	69 y/o M, Parox AF	22.6	43.2	Roof	31	Repeat PVI, reentry originating from RSPV

AF: Atrial fibrillation, AVR: Aortic valve replacement, CAD: Coronary artery disease, CHF: Congestive heart failure, DM: Diabetes mellitus, F: Female, HTN: Hypertension, iLAV: Indexed left atrial volume, LPV: Left sided pulmonary veins, LSPV: Left superior pulmonary vein, M: Male, MVr: Mitral valve repair, OSA: Sleep apnea, Parox: Paroxysmal, Pers: Persistent, PLAS: Peak longitudinal strain, PVI: Pulmonary vein isolation, RSPV: Right superior pulmonary vein, y/o: year old