Electrophysiological findings during atrial fibrillation reablation: Extending from pulmonary vein reconnection to sequential bipolar voltage map information

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Disclosures: None.

Abstract

Background: Left atrial substrate modification targeting low voltage zones (LVZ) is an ablation strategy that—in addition to pulmonary vein (PV) isolation—tries to eliminate arrhythmogenic mechanisms harbored in such tissue. Electrophysiological findings at reablation include (a) PV reconnection, (b) reconnection over previous substrate ablation, and (c) de-novo LVZ.

Objective: To study, prevalence and contribution of these arrhythmogenic electrophysiological entities in patients with atrial fibrillation (AF) recurrences.

Methods: Consecutive patients with highly symptomatic AF undergoing index and reablation were included (n = 113). In all patients’ PV reconnection, reconnection over previous substrate ablation and spontaneous de-novo LVZ were quantitatively assessed and integrated into an individual reablation strategy. Follow-up was based on continuous device monitoring.

Results: At re-do procedure, 45 out of 113 (39.8%) patients showed PV reconnection as the only electrophysiological abnormality. Reconnection over previous lines was the only electrophysiological abnormality in 8 out of 113 (7.1%) patients. Spontaneous de-novo LVZ was the only electrophysiological abnormality in 12 out of 113 (10.6%) patients. Combined findings of PV reconnection, line reconnection, and/or spontaneous de-novo LVZ were seen in 40 out of 113 (35.4%) patients. No detectable electrophysiological abnormality was observed in 8 out of 113 (7.1%) patients. In univariate analysis, none of the tested electrophysiological characteristics independently predicted the outcome after re-do.

Conclusions: In patients undergoing reablation, we could show that reconnection over previous substrate ablation as well as the development of new low voltage...
Presence of low voltage zones (LVZ) in the left atrium (LA) during high-density bipolar voltage mapping (HD-BVM) indicates fibrofatty infiltration which is an important determinant for initiation and maintenance of atrial fibrillation (AF).1-3 Spontaneous fibrofatty transformation is thought to be a progressive process that increases over time and is associated with age and comorbidity such as hypertension, diabetes, and heart failure.6

Individualized LA substrate modification (LASM) targeting LVZ in addition to pulmonary vein isolation (PVI) is an ablation strategy that tries to eliminate arrhythmogenic mechanisms harbored in such tissue areas.5-6 Although this approach is superior against sole PVI, recurrences do occur.7,8 Electrophysiological findings at reablation include (a) pulmonary vein (PV) reconnection, (b) reconduction over previously ablated LVZ, and (c) spontaneous de-novo LVZ. The overall contribution of each of these three arrhythmia-generating entities to the actual recurrence of AF is unclear. That limits strategies for reablation procedures but also impacts the strategic focus during the index ablation.

The aims of the present study were (a) to systemically analyze all electrophysiological findings at reablation, (b) to compare voltage map characteristics between the index and re-do procedures, and (c) to explore long-term outcomes according to those electrophysiological characteristics.

2 | METHODS

2.1 | Subjects

We included consecutive patients with highly symptomatic AF who underwent consecutive index (1st) and re-do (2nd) HD-BVM guided AF ablations at Heart Center Dresden between January 2014 and January 2017. Patients were followed intensively with continuous monitoring as reported previously.8

All participants had (a) paroxysmal or persistent symptomatic AF, (b) previously ineffective antiarrhythmic drug therapy (at least one antiarrhythmic drug), and (c) LA diameter of less than 60 mm (transthoracic echocardiography, parasternal long axis). We excluded patients with (a) the previous ablation affecting the LA, (b) previous thoracic surgery, or (c) previous thoracic radiotherapy or chemotherapy.

The study was approved by the institutional ethical review board (EK 284092012) and conforms to the principles outlined in the Declaration of Helsinki. All data were collected, managed and analyzed at Heart Centre Dresden and the Steinbeis Research Institute, Electrophysiology and Cardiac Devices.

2.2 | Mapping procedure

Mapping was performed as previously reported.4,8 In brief, a HD-BVM map (LVZ <0.5 mV) was created simultaneously with LA surface reconstruction, guided by a three-dimensional electroanatomical mapping system (CARTO3, Biosense Webster, or Ensite Precision, Abbott) using a circular mapping catheter (Lasso, 4 mm interelectrode spacing, Biosense Webster, or Advisor FL, 3 mm interelectrode spacing, Abbott).

All mapping points were taken in sinus rhythm. For each mapping point, stable contact between the local atrial tissue and each pair of electrodes of the circular mapping catheter was required. Extra care was taken while collecting voltage points on the border of LVZ.4

Sufficient quality of the acquired voltage points was verified by the following criteria5: (a) P-wave morphology, (b) coronary sinus (CS) activation sequence, (c) cycle length, (d) local bipolar electrogram morphology, (e) local activation time, and (f) reproducibility.

2.3 | Bipolar voltage map analysis

Quantitative analysis of voltage maps was performed as described previously.4 The LA was divided into five regions, that is, septum, anterior, posterior, inferior and lateral walls, omitting the LA appendage. Each region was further divided into nine equally sized blocks. The median voltage value within each block was recorded and used for further offline analysis. Contiguous areas of bipolar voltage less than 0.5 mV were considered as an area of LVZ. Within each region size and localization of LVZ were analyzed.

Eventually, the LVZs found at reablation were further categorized as (a) spontaneous de-novo LVZ, and (b) ablation associated LVZ.

The bipolar voltage maps at the index procedures served as a reference and compared to voltage maps at re-do procedures. The spontaneous de-novo LVZ at re-do procedures was defined as a contiguous area with bipolar voltage less than 0.5 mV, which located in a predefined segment without previous substrate modifications. The enlarged LVZs in the segment, which contained previous substrate modification, were excluded from further analysis.
2.4 | Ablation line concept and procedural endpoint

Ablation was performed as described previously.\(^3,8\) In brief, all patients received wide encircling PVI with proven entrance and exit block. Additional extra PV ablation was individualized based on the voltage maps where LVZ substrates were targeted with (a) homogenization of small LVZs, (b) linear lesions connecting LVZs to anatomical obstacles (eg, PVs, or mitral annulus [MA]), and (c) linear lesions isolating large LVZs (eg, isolation of the posterior LA wall). Ablation endpoints were (a) lack of local pace capture and (b) bidirectional conduction block over linear lesions.\(^4,5,8\)

A standard induction protocol was carried out at the end of the procedure using a burst stimulation (300, 250, and 200 ms, each for 10 seconds) and a ramp stimulation (300-200 ms for 10 seconds). Further induced regular atrial tachycardias (ATs) were also mapped and ablated. A duration of induced AF longer than 30 seconds was defined as "AF inducible." However, no further ablations were conducted if PVI or PVI + LASM due to LVZ were achieved.

Ablation of the right atrial isthmus was only performed in case of documented/induced typical atrial flutter. Premature atrial contractions (PACs) were mapped and ablated after the achievement of re-PVI and/or LASM if necessary.

2.5 | Follow-up postprocedural management

Antiarrhythmic medications were discontinued, and patients remained on \(\beta\)-blocker. In the case of arrhythmia recurrences, antiarrhythmic drugs were reintitated upon an individual decision. Reablation for symptomatic drug-refractory recurrences of AF and AT was scheduled after at least 3 months from the index procedure.

TABLE 1  Baseline characteristics

|                          | All patients, n = 113 | with LVZ progression, n = 36 | without LVZ progression, n = 77 | \(P\) value |
|--------------------------|-----------------------|-----------------------------|--------------------------------|------------|
| Age                      | 67 ± 9                | 70 ± 6                      | 65 ± 10                         | .009       |
| Male gender              | 67 (59%)              | 21 (58%)                    | 46 (60%)                        | .89        |
| Body mass index          | 29.4 ± 6              | 29.1 ± 6                    | 29.6 ± 4                        | .64        |
| Sleep apnea              | 9 (8%)                | 2 (6%)                      | 7 (9%)                          | .52        |
| Hypertension             | 91 (81%)              | 32 (89%)                    | 59 (77%)                        | .13        |
| Diabetes                 | 24 (21%)              | 8 (22%)                     | 16 (21%)                        | .86        |
| Coronary artery disease  | 29 (26%)              | 13 (36%)                    | 16 (21%)                        | .08        |
| Heart failure            | 15 (13%)              | 7 (19%)                     | 8 (10%)                         | .19        |
| Renal failure            | 6 (5%)                | 3 (4%)                      | 3 (8%)                          | .32        |
| History of stroke        | 4 (4%)                | 0 (0%)                      | 4 (5%)                          | .16        |
| Pacemaker or ICD         | 13 (12%)              | 4 (11%)                     | 9 (12%)                         | .93        |
| CHA2DS2VASc score        | 2.5 ± 1.3             | 2.7 ± 1.1                   | 2.4 ± 1.3                       | .19        |
| LVEF (%)                 | 54 ± 12               | 50 ± 14                     | 56 ± 10                         | .03        |
| Left atrial diameter, mm | 45 ± 6                | 45 ± 5                      | 46 ± 6                          | .86        |

Abbreviation: LVEF, left ventricular ejection reaction; LVZ, low voltage zones.

Oral anticoagulation was continued for at least 3 months and thereafter according to CHA2DS2-VASc-Score with deviations upon patient and physician’s discretion.

Postinterventional rhythm assessment was based on implantable device monitoring (Reveal LINQ, Medtronic, Inc) and 4-day Holter every 3 months.\(^8\)

2.6 | Data management and statistical analysis

Continuous variables were tested for normal distribution using the Shapiro-Wilk test. Data with normal distribution are presented as mean ± SD and data without normal distribution are presented as the median and interquartile range. Categorical variables are expressed with a number and percentage of patients.

Differences between continuous normally distributed data were tested with the Student \(t\) test and differences between categorical data were tested with a \(\chi^2\) test. A \(P \leq .05\) was considered significant. The data were collected and managed by the investigators (MK and YH) and all statistics were performed using Stata version 12 (Stata Corporation, College Station, TX).

3 | RESULTS

3.1 | Patient characteristics

During the study period, 1758 patients underwent HD-BVM guided AF ablation at our institution. Of these, 129 patients underwent both index and re-do procedures. In 16 patients, the HD-BVM was insufficient at one of the procedures and a total of 113 patients was included in the study
(mean age 67 ± 9, 59% male, 40% with paroxysmal AF). Meantime between the index and re-do procedures was 16 ± 10 months. Baseline characteristics of patients are shown in Table 1. Patients with spontaneous de-novo LVZ after the index procedure were significantly older (65 ± 10 vs 70 ± 6 years, P = .009) and had a lower left ventricular ejection fraction (LVEF) (56% ± 10% vs 50% ± 14%, P = .03) as compared with patients without spontaneous de-novo LVZ, otherwise baseline characteristics were comparable. Among the study patients, 82% (n = 93) were followed with continuous monitoring from the implantable device and 18% (n = 20) with periodic 4-day Holter (Figure 1).

### 3.2 Characteristics of documented atrial arrhythmias between the index and re-do procedures and ablated regular ATs at the re-do procedure

Out of 113 patients, 33 (29%) paroxysmal AF, 66 (58%) persistent AF, and 14 (12%) regular AT were documented between the index and re-do procedures. There were no significant differences in the regular ATs recurrences between patients with and without spontaneous de-novo LVZ (n = 6/36 vs n = 8/77, P = .35). However, in the patients with spontaneous de-novo LVZ at re-do procedures, the incidence of persistent AF was significantly greater than in patients without de-novo LVZ (n = 28/36 vs n = 38/77, P = .001; Table 2).

### TABLE 2 Type of clinical documented atrial arrhythmias between the index and re-do procedures

| Spontaneous de-novo LVZ | Type of atrial arrhythmias before re-do procedure |
|-------------------------|-----------------------------------------------|
| No                      | Paroxysmal AF | Persistent AF | AT/AFL | Total |
| No                      | 31             | 38             | 8      | 77    |
| Yes                     | 2              | 28             | 6      | 36    |
| Total                   | 33             | 66             | 14     | 113   |

Abbreviations: AF, atrial fibrillation; AFL, atrial flutter; LVZ, low voltage zones.

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**FIGURE 1** Flowchart. Abbreviation: AL, anterior line; LVZ, low voltage zone; MIL, mitral isthmus line; PL, posterior line; PSL, paraseptal line; PVI, pulmonary vein isolation; RL, roof line; SL, septal line.
A total of 7 patients with a regular AT at the beginning of the re-do procedures and further 5 were fully mapped and ablated successfully, including 2 perimital ATs, 2 roof-dependent ATs, and 1 localized reentrant AT originating at LA anterior wall. The rest 2 ATs were terminated during mapping. At the end of the re-do procedures, 3 perimital ATs originating at LA were induced due to acute re-conductions over anterior ablation lines (large LVZ-LA anterior wall) and further bidirectional blocks were re-achieved.

### 3.3 Voltage map and ablation characteristics during the index procedure

At the index procedure, the mean surface area of LA was $93 \pm 24$ cm$^2$ and the mean voltage amplitude of LA was $2 \pm 1$ mV. In 39 out of 113 (35%) patients LVZ was documented with a mean extension of $27 \pm 19$ cm$^2$ at the index procedure.

A PVI-only ablation strategy was performed in all patients without LVZ and in 5 patients with sole LVZ location at the PV antrum. Overall 79 (70%) patients received PVI only. The remaining 34 (30%) patients additionally received a median of 3 (range: 1-5) ablation lines for LASM (Figures 2-4).

### 3.4 Voltage map characteristics during re-do procedures

A total of 36 (32%) out of 113 patients developed spontaneous de-novo LVZ at the re-do procedure. Out of the 74 patients without LVZ at the index procedure, 23 (31%) patients displayed spontaneous de-novo LVZ at the reablation. Regions with de-novo LVZ included posterior, anterior, and septal LA segments.

Out of the 39 patients with LVZs at the index procedure, 13 (33%) patients displayed spontaneous de-novo LVZ at the reablation. In those patients, only the posterior region had a significant increase in LVZ (Figures 2).

In patients (n = 51) with no LVZ at the index and re-do procedures, overall bipolar LA voltage declined significantly from the index to re-do procedures ($2.6 \pm 0.9$ vs $2.2 \pm 0.7$ mV, $P = .013$). On a regional level, a decline in voltage was only seen in the anterior and septal regions (Table 3).

### 3.5 Permanence of lesions assessed during the re-do procedure

At the re-do procedure, persistent PV isolation in all 4 PVs was found in 35 out of 113 (31%) patients. In 17 (15%) patients 1 PV had

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**FIGURE 2** Voltage maps at the index and re-do procedures. Upper row: voltage maps were carried out before any ablation at the index procedure. The map shows that a small LVZ surrounding LPV on the posterior wall of LA. The yellow lines represent the PVI lesion set. Lower row: voltage maps were carried out before any ablation at the re-do procedure. The map shows that the presence of de-novo LVZ covers the entire posterior wall of LA and patchy de-novo LVZ on the anterior segment of LA. AP, anterior-posterior projection; LA, left atrium; LPV, left pulmonary vein; LVZ, low voltage zone; PA, posterior-anterior projection; SUP, superior projection.
reconnected, in 30 (27%) patients 2 PVs, in 5 (4%) patients 3 PVs, and in 26 (23%) patients all 4 PVs.

Persistent bidirectional conduction block overall previous ablation lines placed for LVZ substrate modification was seen in 7 out of 34 (21%) patients. As such reconduction over the posterior segment (e.g., roof and posterior lines) was present in 9 out of 14 (64%) affected patients, and over the anterior segment (e.g., roof, anterior and/or septal lines) in 20 out of 31 (65%) affected patients. In total 56 (56%) of all 100 index lines had recovered.

3.6 | Prevalence of different arrhythmogenic entities at re-do procedures

The 3 identifiable electrophysiological conduction abnormalities at the re-do procedure; (type I) PV reconnection, (type II) reconduction over previous ablation lines, and (type III) spontaneous de-novo LVZ were prevalent as follows:

a. 45 (39.8%) Patients displayed type I abnormality only and received reisolation of the PVs.

b. In 8 (7.1%) patients type II abnormality was the only electrophysiological abnormality and reenforcing line continuity was the equivalent ablation concept.

c. In 12 (10.6%) patients type III abnormality was the only electrophysiological abnormality and tailored linear ablation was performed.

d. 40 (35.4%) Patients had a combination of type I, II, or III and required a combination of PV reisolation and linear ablation.

e. In 8 (7.1%) patients no detectable electrophysiological conduction abnormality was observed.

3.7 | Ablation of PACs outside of PVs

In 11 (9.7%) out of 113 patients, PACs were present after the Re-PVI and LASM. Further, these PACs (n = 17) were mapped and ablated at the end of the procedure (four originating at CS ostium, 5 from CT, 1 from MA 5 o’clock, 3 from LA anterior wall—eg, Bachmann’s incision, and 4 from LA posterior wall).

3.8 | Complications, follow-up, and predictors of outcome

Perioperative complications for overall 226 interventions included 1 transitory ischemic attack, 1 tamponade, and 5 pseudoaneurysms.
Overall 12-month freedom from arrhythmia was 58% (66/113) after reablation. Out of the patients with continuous device monitoring 56% (52/93) patients had an arrhythmia burden of less than 0.1% during 12 months of follow-up.

During univariate analysis, age, gender, CHA2DS2-VASc score, type of AF, isolated pulmonary veins, line reconduction, and presence of LVZ were not significantly associated with AF recurrence after reablation (Figure 5).

4 | DISCUSSION

4.1 | Main findings

Our study extends the existing knowledge of causes of AF recurrences after an index ablation beyond the finding of PV reconnection. Developments of new LVZ, as well as reconnection over previous substrate modification, are frequent electrophysiological findings during the re-do procedure. Multiple of these arrhythmia-generating entities were often found together in a single patient.

These data suggest that singular strategic approaches—such as a focus on permanently durable PV isolation alone—may not be able to suppress all AF recurrences. Decreasing bipolar voltages in the majority of patients at re-do procedures, especially the high amount of de-novo low voltage substrates, indicates a significant role of disease progression and supports the need for alternative concepts.

4.2 | What makes AF recurrences?

PV electrophysiology has recognized a cornerstone of the AF pathophysiology. Therefore, PV isolation is an accepted ablation strategy and endpoint. Subsequently PV reconnection has been identified as a frequent finding during reablation and is considered the main reason for AF recurrences. Our data are in line with these findings of PV-reconnection.
In the attempt to improve the long-term efficacy of AF ablation a focus has been put on improving durable PV isolation. Outcome data in large-scale clinical trials, however, are conflicting. Therefore, the question needs to be raised, whether alternative arrhythmia-generating mechanisms are involved in the development of AF recurrences. Under that light, the evolution in the understanding of AF pathophysiology has to be considered. Today structural remodeling in histarchitecture is recognized to alter cellular coupling with subsequent conduction impairment. Affected myocardial regions are substrates for reentry—a keystone in AF development. Voltage mapping during sinus rhythm provides electrophysiological surrogates for such tissue pathologies. Our study used sequential bipolar voltage map information to describe the evolution of these tissue and conduction abnormalities over longer time periods, which for the first time allows insights into mechanisms for AF recurrences beyond PV reconnection.

4.3 | Disease progression

Table 3: Voltage map characteristics of the patients undergoing reablation

| Voltage map characteristics | 1st Procedure | 2nd Procedure | P      |
|----------------------------|---------------|---------------|--------|
| Voltage, mV, in patients without LVZ, n = 51 |               |               |        |
| Left atrium                | 2.6 ± 0.9     | 2.2 ± 0.7     | .013   |
| Anterior wall              | 2.5 ± 1.1     | 2.0 ± 0.9     | <.001  |
| Septal wall                | 2.1 ± 0.8     | 1.8 ± 0.7     | .018   |
| Inferior wall              | 2.8 ± 1.4     | 2.4 ± 1.1     | .08    |
| Lateral wall               | 2.8 ± 1.2     | 2.6 ± 1.2     | .45    |
| Posterior wall             | 2.7 ± 1.4     | 2.4 ± 1.3     | .37    |
| Area, cm², LVZ in patients with new LVZ, n = 23 |               |               |        |
| Left atrium                | 0 ± 0         | 9.7 ± 9       | <.001  |
| Anterior wall              | 0 ± 0         | 3.8 ± 6       | .02    |
| Septal wall                | 0 ± 0         | 1.6 ± 2.7     | .02    |
| Inferior wall              | 0 ± 0         | 0.6 ± 1.4     | .06    |
| Lateral wall               | 0 ± 0         | 0.4 ± 0.7     | .011   |
| Posterior wall             | 0 ± 0         | 3.2 ± 4.4     | .004   |
| Area, cm², LVZ in patients with existing LVZ, n = 39 |               |               |        |
| Left atrium                | 27 ± 19       | 38 ± 22       | .004   |
| Anterior wall              | 11.3 ± 7      | 12.6 ± 7      | .34    |
| Septal wall                | 6.6 ± 7       | 8.5 ± 8       | .21    |
| Inferior wall              | 1.8 ± 4       | 3.2 ± 7       | .24    |
| Lateral wall               | 0.6 ± 2       | 1.2 ± 3       | .36    |
| Posterior wall             | 6.6 ± 8       | 11.8 ± 9      | .006   |

Abbreviation: LVZ, low voltage zones.

In the attempt to improve the long-term efficacy of AF ablation a focus has been put on improving durable PV isolation. Outcome data in large-scale clinical trials, however, are conflicting. Therefore, the question needs to be raised, whether alternative arrhythmia-generating mechanisms are involved in the development of AF recurrences.

Under that light, the evolution in the understanding of AF pathophysiology has to be considered. Today structural remodeling in histarchitecture is recognized to alter cellular coupling with subsequent conduction impairment. Affected myocardial regions are substrates for reentry—a keystone in AF development. Voltage mapping during sinus rhythm provides electrophysiological surrogates for such tissue pathologies.

Our study used sequential bipolar voltage map information to describe the evolution of these tissue and conduction abnormalities over longer time periods, which for the first time allows insights into mechanisms for AF recurrences beyond PV reconnection.

Even in patients with no LVZ at the index and re-do procedures, we found a significant attenuation of the overall left atrial bipolar voltage amplitudes with predominance in anterior and septal LA wall regions during reablation. Teh AW et al. reported a significant decrease in mean RA bipolar voltage in patients with lone AF that underwent PVI after a mean follow-up period of 10 ± 13 months, which is in line with our findings. In a recently published study by Marrouche et al., they found that an increase of 1% "new fibrosis" increased the chance of postablation AF recurrence by 3%, which also suggests that disease progression on the level of atrial myocardium does occur and may cause AF recurrences.

These findings may be interpreted as surrogates of the progression of the underlying degenerative process in the atrial myocardium. The time variable of this process is not well understood. Our data, however, imply that at least in a subset of AF patients, that process may occur within a year. We speculate, that next to environmental influence and cardiovascular risk profiles, genetic factors may contribute. Except the presence of spontaneous de-novo LVZ most likely exists in elder patients and patients with lower LVEF, which is in line with previous publications, there are no further correlations observed on previously studied factors, which related to fibrofatty infiltration and voltage reduction, such as obesity, sleep apnea, etc. It might relate to the characteristics of the study cohort, in which only patients with AF recurrences involved. Unfortunately, our patient numbers are too small to provide further solid insights into that phenomenon.
4.4 | Durability of RF ablation lesions

The detailed analysis of the durability of ablation lesions from the index procedure was a further important aspect of our study. Next to the finding of PV reconnection our data illustrate the problem of ablation line durability also for other target regions of the LA. The high rate of substrate line reconduction raises questions on technology, technique, and acute endpoint of RF catheter induced left atrial lesions. These data are consistent with recent reports on the need for epicardial catheter access to achieve and assess true atrial lesion transmurality.

4.5 | Clinical outcome

Given the intense follow-up with continuous device monitoring the reported clinical outcome after the re-do procedure was acceptable. More than half of the patients did not have a single arrhythmia episode within the first 12 months.

In univariate analysis reconnection of PVs, reconduction over previous lines, presence of LVZ, type of AF, and various clinical characteristics were not independently associated with the success of reablation. These findings suggest that none of these factors alone can be considered as the main reason for recurrences. They may also hint to other so far unrecognized arrhythmia mechanisms—not included in the analysis—to play a role for recurrences. In that context, it is interesting to mention recent data on exclusive epicardial arrhythmia substrates responsible for AF in re-do patients.

4.6 | Limitations

The present study could suffer from the inherent limitations of a nonrandomized study including selection bias, confounding and lack of control group. The sample size was moderate, and a minor proportion of the patients was excluded due to insufficient voltage maps from one of the two procedures. Furthermore, we did not perform any routine imaging or histology to support the presence of fibrofatty tissue underlying LVZs. On the other hand, this is the first study reporting findings from sequential voltage maps over long time periods in patients undergoing ablation for AF.

The fact, that we only mapped patients with AF recurrences limits our ability to judge the individual contribution of the various electrophysiological abnormalities to the actual arrhythmia recurrence. The fact that PACs were mapped and ablated after Re-PVI
and/or substrate modifications may underestimate the influences of triggers outside of PVs.

5 | CONCLUSION

In patients undergoing reablation we could show that reconduction over previous substrate ablation as well as the development of new low voltage areas are frequent findings besides classical PV reconnection—without a clear leading cause for recurrences. These findings impact reablation strategies as well as the strategic focus during index procedures.

CONFLICT OF INTERESTS

The authors have no conflicts of interest pertaining to this research. However, CP has received modest lecture honoraria from Abbott, Biotronik, Siemens, Medtronic, Biosense; has received research support from Abbott, Biotronik, Biosense, Medtronic; and serves as an advisory board member of Abbott, Siemens, Biosense, Biotronik, Imricor. The other authors have no conflicts of interest to disclose.

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