Endo-/Epicardial Catheter Ablation of Atrial Fibrillation

Feasibility, Outcome, and Insights Into Arrhythmia Mechanisms

BACKGROUND: Until today, catheter interventional mapping and ablation of atrial fibrillation (AF) has been limited to the right and left atrial endocardium. We report feasibility, electrophysiological findings, and clinical outcome using a combined endo-/epicardial catheter approach for mapping and ablation of AF.

METHODS AND RESULTS: Fifty-nine patients with permanence of pulmonary vein isolation and further symptomatic recurrences of paroxysmal AF, persistent AF, or atrial tachycardia underwent reablation using biatrial endo-/epicardial mapping and ablation. Identification of arrhythmia substrates and selection of ablation strategy were based on sinus rhythm voltage mapping. Using continuous monitoring and a 3-month blanking period, freedom from AF/atrial tachycardia ≥2 minutes was defined as primary end point. In all patients, endo-/epicardial mapping and ablation was feasible using standard technologies of catheter access, 3-dimensional mapping, and radiofrequency ablation. Epicardial mapping and ablation did not add procedural risks. Exclusively epicardial low voltage substrates were found in 14% of the patients. For the first time, novel epicardial conduction abnormalities located in the epicardial fiber network were described in human AF patients (19% of the cohort). Epicardial ablation was needed in 80% of the patients. Over 23±10 months of follow-up freedom from arrhythmia recurrences measured 73%.

CONCLUSIONS: Catheter-based endo-/epicardial mapping and ablation of AF was feasible and safe. Epicardial mapping provided new insights into AF mechanisms. Epicardial ablation increased transmurality of ablation lesions. Clinical outcome in this cohort of complex AF patients was favorable, indicating potential further development of current AF treatment.

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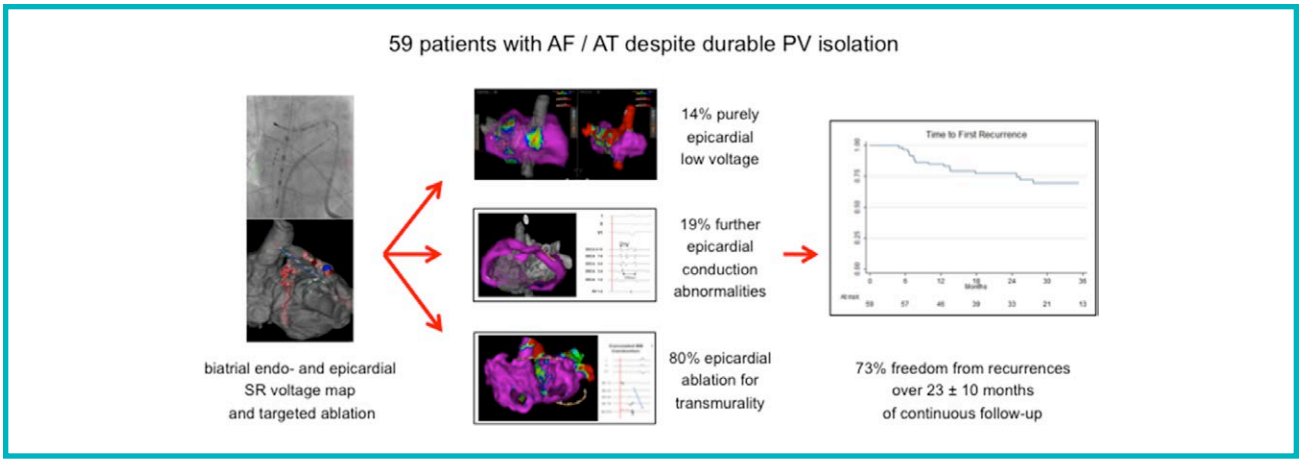

WHAT IS KNOWN?

- Arrhythmia mechanisms located outside the pulmonary vein antrum are commonly suspected, especially in patients with recurrences of atrial fibrillation and atrial tachycardia despite durable pulmonary vein isolation.
- Until today, mapping and targeted ablation of such arrhythmia sources has been limited to right and left atrial endocardium.

WHAT THE STUDY ADDS?

- Simultaneous catheter-based endo- and epicardial mapping and ablation of complex atrial arrhythmias was technically feasible with low risks of complications.
- Epicardial access (1) enabled detection of exclusive epicardial low voltage substrates, (2) improved transmurality of linear ablation lesions, and (3) revealed conduction abnormalities located in the epicardial fiber network and their role in the development of atrial fibrillation reentry mechanisms.

Catheter ablation is standard of care for patients experiencing symptomatic drug-refractory atrial fibrillation (AF).[1] Performing wide circumferential pulmonary vein (PV) isolation is recognized as ablation cornerstone.[2,3]

Additional arrhythmia sources outside the PV antrum are commonly suspected, especially in patients with recurrences despite PV isolation. Targeting these sources has lead to several ablation concepts on top of PV isolation.[4–8] The limited success rate—especially in persistent AF—drives further research to better understand arrhythmia mechanisms and improve ablation concepts.

Although percutaneous epicardial ablation of other arrhythmias such as ventricular tachycardia or accessory pathways has been performed,[9] until now catheter ablation of AF has been limited to the right atrial (RA) and left atrial (LA) endocardium. Only surgical approaches have used epicardial AF ablation access.[10–12] Besides the invasiveness of surgical access, these procedures have focused on continuity of empirical ablation lines without mapping additional arrhythmia mechanisms.

This study reports feasibility, electrophysiological findings, and clinical outcome using a combined catheter interventional endo-/epicardial mapping and ablation approach in patients with arrhythmia recurrences despite permanent PV isolation.

METHODS

An investigator-initiated, prospective, nonrandomized study was conducted at Heart Center Dresden, Germany, between January 2014 and March 2017. The study was approved by the institutional ethical review board (EK 28409202), and all participants provided written informed consent. Data were collected, managed, and analyzed at Heart Center Dresden and the Steinbeis Research Institute-Electrophysiology and Cardiac Devices. The data, analytic methods, and study materials will be made available to other researchers for purposes of reproducing the results or replicating the procedure.

Inclusion and Exclusion Criteria

Patients with ≥2 prior attempts of endocardial catheter ablation who continued to experience symptomatic paroxysmal AF, persistent AF, or atrial tachycardia despite permanent PV isolation were included.

Patients were excluded if they had suspected pericardial adhesions caused by prior cardiac surgery or other pericardial disease (eg, constructive pericarditis).

Primary and Secondary End Points

Freedom from any episode of AF/atrial tachycardia ≥2 minutes occurring >3 months after the procedure was defined as the primary end point.

Feasibility to complete the epicardial map, procedural duration, fluoroscopy exposure, and complications were secondary end points.

Electrophysiological Analysis

Identification of AF substrates was based on voltage mapping in sinus rhythm (SR) as described previously.[13–15] The following 3 questions were assessed: (1) presence of endo- and epicardially detected low voltage areas (LVAs), (2) presence of conduction abnormalities beyond LVAs, and (3) need for epicardial ablation to create transmural lesions.

Procedural Setup

Patients were studied in deep sedation. Epicardial and LA access was obtained by established techniques of pericardial and transseptal puncture.[16,17] Steerable sheath technology was used for both access routes.

Endocardial surface reconstruction was acquired using a circular mapping catheter (LASSO-NAV; Biosense Webster) for fast anatomic mapping (CARTO-UNIVU; Biosense Webster). Epicardial surface reconstruction was acquired using a linear multipolar mapping catheter (DECA-NAV; Biosense Webster) for point-by-point mapping.

Ablation was performed with radiofrequency energy using standard irrigated-tip ablation catheter (ThermoCool SF-ST; Biosense Webster). The standard ablation settings included a preselected power of 40 W and a flow rate of 15 mL/min. At the posterior LA wall, power delivery was limited to 10 g, 30 W, and 20 s and was terminated in case of intraesophageal temperature increases >39°C. During epicardial ablation at the posterior LA wall, 80 cc saline was installed into the pericardial cavity, and the force vector-directed catheter tip orientation pointed away from the parietal pericardium and the esophagus.

Ablation Line Concept and Procedural End Point

Patients presenting in AF were cardioverted, and patients presenting in stable atrial tachycardia were mapped and ablated using established techniques of activation and entrainment mapping.

In SR, biatrial endo-/epicardial voltage maps were reconstructed in all patients. Bipolar peak-to-peak electrogram amplitude <0.5 mV was defined as diseased low voltage signal. Lack of local pace capture (10 V, 2 ms) was used to identify scar.[13–15]

The ablation strategy was individualized based on the voltage maps. Low voltage substrates were targeted with (1) homogenization of small LVAs, (2) linear lesions connecting LVAs to anatomic obstacles (eg, PVs, mitral annulus, superior vena cava [SVC], or inferior vena cava [IVC]), and (3) linear lesions isolating large LVAs (eg, isolation of the posterior LA wall).[13–15] LVAs were ablated to abolish potential slow conduction zones. Linear lesions were performed to prevent macroreentrant circuits around LVA. In LVAs covering >60% of the surface of a specific wall segment (eg, posterior wall), the entire wall segment was isolated. Ablation end points were (1) lack of local pace capture and (2) bidirectional conduction block over linear lesions. Ablation end points were assessed from endo- and epicardial.

In patients without low voltage substrates, the posterior LA wall was isolated, and linear ablation from the right upper PV to the mitral annulus was performed.

All patients received isolation of the SVC. Ablation of the RA isthmus was only performed in case of documented/induced typical atrial flutter.

At the end of the procedure, burst/ramp stimulation was performed (decreasing cycle lengths from 300 ms until atrial refractoriness) without isoproterenol. Noninducibility was the procedural end point.

Specific Aspects of Epicardial Atrial Mapping

Mapping of the epicardial atrium is closely related to the locations of the pericardial folds, which mark the transition from visceral to the parietal layer of the pericardium. They are typically located around the PV antra, horizontally along the roof of the posterior LA, vertically from the posterior SVC to the posterior IVC, and around the proximal aspects of the aorta and the pulmonary artery. Because these pericardial folds cannot be crossed from within the pericardial space, they determine catheter manipulation.

Best mapping conditions were found with an inferior pericardial access. The posterior LA wall was freely accessible from below the left lower PV. Over the ligament of Marshall, access was gained into the LA roof. A registered computed tomography model provided information on the localization of the ostia of the coronary arteries. Mapping of the LA roof was possible within the boundaries of the horizontal pericardial fold (toward the posterior LA) and the aorta (toward the mitral annulus). The latter one limited epicardial access to the annular portion of an anterior ablation line (right upper PV to mitral annulus), which could only be ablated from the inside. The LA roof remote from the annulus, the overlaying fibers of Bachmann's bundle (BB), and the epicardial interatrial groove, however, were freely accessible from the transverse sinus. Underneath the aorta and in front of the SVC, the sinus also provided catheter access toward the RA surface. The vertical pericardial fold (SVC—posterior RA—IVC) represented the anatomic boundary from the posterior RA back to the posterior LA.

Care was taken to orientate the epicardial catheter curve away from the parietal pericardium and toward the outer atrial surface. Permanent suction on the epicardial sheath evacuated fluid out of the pericardium instantaneously. All patients received an intravenous single shot antibiotic coverage (cefazolin, 2 g) before the pericardial puncture. After the experience of late inflammatory pericardial effusions, all patients received intrapericardial steroid injection (prednisolone, 2 mg/kg) at the end of the procedure.

Postprocedural Care and Follow-Up

Heparinization was reversed using equivalent doses of protamine. All sheaths were withdrawn on the table. Hourly echocardiography was repeated the first 6 hours. All patients received an esophagoscopy to rule out thermal esophageal lesions.

Antiarrhythmic medication was discontinued, and patients remained on β-blocker. In case of arrhythmia recurrences, antiarrhythmic drugs were reinitiated on an individual decision.

Oral anticoagulation was permanently continued. Patients with LA appendage (LAA) isolation additionally received aspirin (100 mg/d) and were rescheduled for LAA occlusion.

Postinterventional rhythm assessment was based on implantable device monitoring and quarterly 4-day Holter recordings.

Statistical Analysis

Categorical variables were reported as count and percentages and continuous variables as mean and SD if normally distributed otherwise as median with interquartile range. Arrhythmia recurrence rate was reported with Kaplan–Meier plot using a 3-month blanking period. All statistical analyses were performed using SPSS Statistics (version 21).

RESULTS

The study enrolled 59 patients (mean age, 67±9 years; 38 male) who previously had undergone median 3 (3–4) endocardial ablation procedures. Clinical arrhythmia presentation was paroxysmal AF in 17, persistent AF in 28, and atrial tachycardia in 14 patients. Arrhythmia symptom burden measured a median EHRA (European Heart Rhythm Association) score of 2 (2–3). Detailed patient characteristics are shown in Table.

Procedural Data

The biatrial endo-/epicardial surface reconstruction including a high-resolution voltage map was feasible in all patients. Procedure duration measured 174±55 minutes, and fluoroscopy time was 36±12 minutes. Radiofrequency energy was delivered over 27±14 minutes.

At the end of the procedure 49 (83%), patients were noninducible during burst and ramp stimulation.

As major complications, we observed 1 cardiac tamponade occurring during endocardial ablation after an incidence of steam pop and 1 femoral access complication. We did not observe any esophageal fistula, phrenic nerve palsy, or coronary artery injury.

No complication was associated with the epicardial access route.

Ablation resulted in electric LAA isolation in 13 (22%) patients. All of them underwent uncomplicated LAA closure later on.

Primary End Point

Using a 3-month blanking period, 43 (73%) patients remained free from arrhythmia during 23±10 months follow-up. Kaplan–Meier plot of time to first arrhythmia recurrence is shown in Figure 1. In 49 (83%) patients,outcome assessment was based on implantable devices (Table).

Freedom from arrhythmia occurred in 40 (77%) patients off antiarrhythmic medication and in 3 (43%) patients on antiarrhythmic medication (3 on Flecainide and 4 on Amiodarone). Furthermore, 4 patients had prolonged healing up to 6 months—with subsequent arrhythmia freedom. And additional 4 patients developed late recurrences after 2 years without arrhythmias. When adding those 8 patients to the 43 patients free from arrhythmia, 51 (86%) patients had clinically benefited from the ablation.

Table. Baseline Characteristics

Baseline Characteristics	All (n=59)
Age, y	67±9
Male sex	38 (64%)
Body mass index	28.6±5
Hypertension	47 (80%)
Diabetes mellitus	10 (17%)
Coronary artery disease	7 (12%)
Renal failure	2 (3%)
Ischemic cardiomyopathy	2 (3%)
Dilated cardiomyopathy	2 (3%)
History of stroke	2 (3%)
Left ventricular ejection fraction (%)	55±9
Left atrial diameter, cm	44±5
Dual chamber pacemaker	5 (8%)
Dual chamber defibrillator	1 (2%)
Triple-chamber defibrillator	1 (2%)
Implantable loop recorder	42 (71%)
No. of previous AF ablations	3 (IQR, 3–4)
CHA_2DS_2VASc score	2 (IQR, 1–3)
EHRA score	2 (IQR, 2–3)
Presenting arrhythmia	
Paroxysmal AF	17 (29%)
Persistent AF	28 (47%)
Regular AT	14 (24%)

AF indicates atrial fibrillation; AT, atrial tachycardia; EHRA, European Heart Rhythm Association; and IQR, interquartile range.

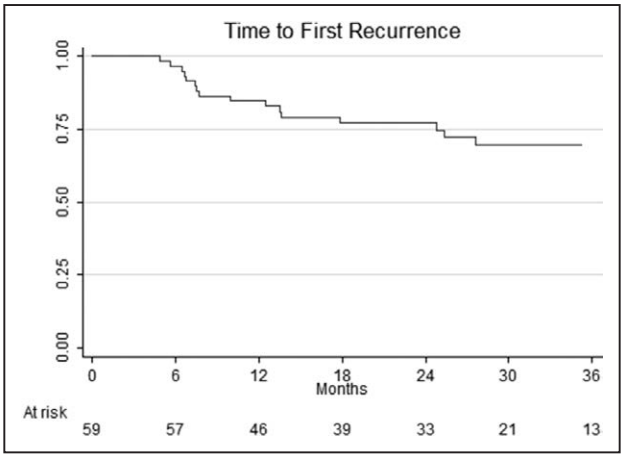

Figure 1. Kaplan–Meier recurrence analysis: time to first arrhythmia recurrence after a 3-month blanking period.

Electrophysiological Findings: SR Voltage Analysis

In 44 (75%) patients, mapping revealed regions with impaired voltage amplitudes outside the PVs—13 (30%) patients without prior substrate ablation and 31 (70%) patients with prior substrate ablation. Overall, 29 patients had LVAs at sites without previous ablation indicating intrinsic disease.

Endo-/epicardial map analysis showed a transmural representation of all LVA substrates in 33 of 44 (75%) patients. Exclusive epicardially located LVAs were found in 6 of 44 (14%) patients—typically at the posterior RA wall between SVC and IVC (Figure 2). Conversely, 6 of 44 (14%) patients showed endocardial LVAs that were not detectable epicardially (Figure 3).

Electrophysiological Findings: Further Conduction Abnormalities

In 11 (19%) patients, novel and exclusively epicardial conduction abnormalities were detected.

As a pattern, SR electrograms with double or triple atrial signal components separated by a significant conduction delay with multipolar activation sequences pointing into opposite wave front directions indicated regions with nonuniform spread of electric activation (Figure 4). These conduction abnormalities represented a localized finding covering a distance of 3 to 4 cm. They were unrelated to the actual voltage amplitude and not detectable from endocardial.

The conduction abnormalities were found along the epicardial fiber network: at the junction between Ligament of Marshall and BB (n=7), at BB insertions into the septal LAA (n=1), inside BB itself (n=3), at BB insertions into the anterior SVC (n=2), and within the lower interatrial connections (n=3).

Underlining their arrhythmogenic role, spontaneous or induced localized reentrant tachycardia originating from within these regions could be mapped in 6 out of 11 patients (Figure 5).

Selective elimination of 1 limb of the interatrial conduction (eg, ablation of BB) abolished the conduction abnormalities in areas remote to the ablation (Figure 4).

Electrophysiological Findings: Lesion Transmurality

Overall, 47 of 59 (80%) patients received epicardial radiofrequency energy delivery.

A total of 165 linear ablation lines were completed or newly placed. Epicardial ablation was needed to achieve continuity in 63 of 165 (38%) of these lines (Figure 6).

The need for epicardial line completion varied between anatomic regions of the atria. The anterior LA line (including blockage of BB) required epicardial ablation in 36 of 46 (78%) cases, the superior box lesion in 14 of 42 (33%) cases, and the inferior box lesion in 4 of 42 (9%) cases. All 9 (100%) classical mitral isthmus lines needed epicardial closure, and no epicardial ablation was performed for paraseptal lines, RA isthmus lines, and intercaval lines.

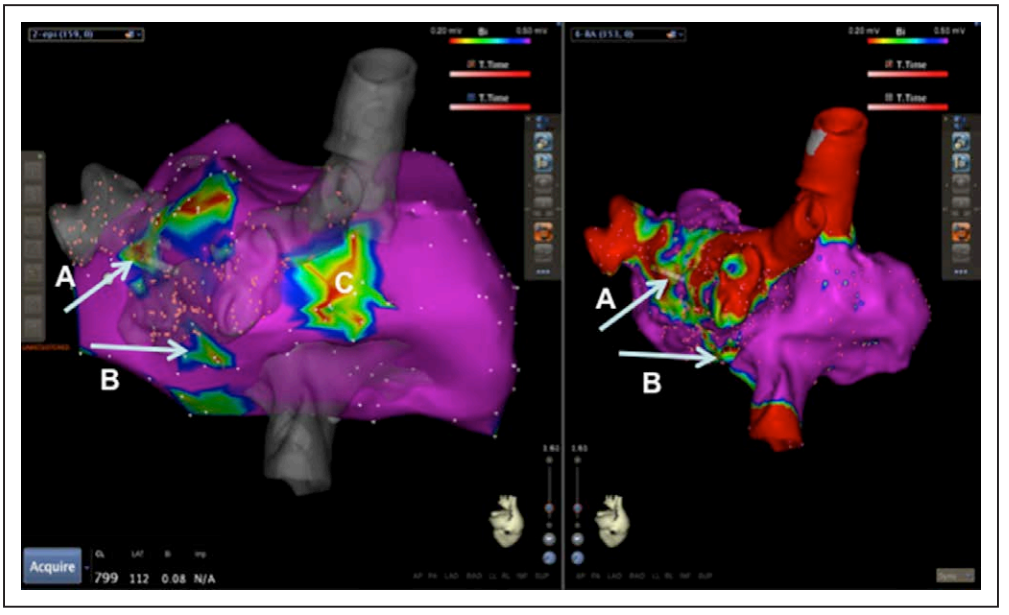

Figure 2. Biatrial endo- and epicardial voltage map.
Left, Epicardial map. **Right**, Endocardial map. Case example of a patient with transmural low voltage substrates (LVA) at the posterior left atrium (A) and the paraseptal wall (B) and exclusive epicardial LVA at the posterior right atrium between superior and inferior caval vein (C). The ablation concept included isolation of the posterior left atrial wall, a paraseptal ablation line (from right lower pulmonary vein to mitral annulus), and a right atrial intercaval ablation line.

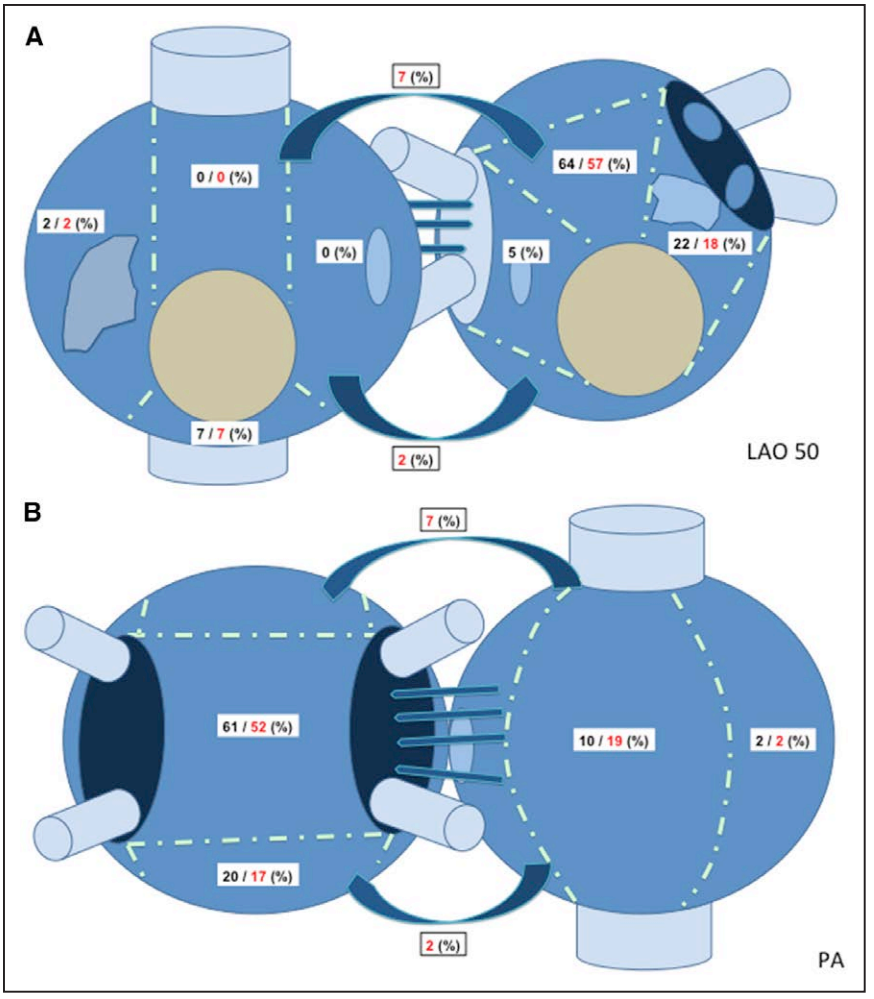

Figure 3. Schema showing biatrial endo- and epicardial distribution of low voltage substrates. Two different projections (**A**, left anterior oblique projection [LAO]; and **B**, posterior-anterior projection [PA]) in which the atria are shown for the analysis. For each atrial wall segment and the interatrial connection pathways, percentage of patients with endocardial (black number) and epicardial (red number) low voltage area (LVA) is shown.

DISCUSSION

Main Findings of the Study

This is the first study using a combined percutaneous endo-/epicardial mapping and ablation strategy to treat patients with AF. The procedure was technically feasible with low risk of complications.

Besides insights into the distribution of endo-/epicardial low voltage substrates and data on ablation line transmurality, for the first time, this study showed novel conduction abnormalities—associated to the epicardial fiber network—and their potential role in the development of AF reentry mechanisms in selected patients.

Using biatrial endo-/epicardial mapping information to individualize the ablation strategy and treat any detectable conduction abnormality, freedom from arrhythmia recurrence was 73% after 2 years of intense follow-up.

Epicardial Arrhythmia Sources: Voltage Analysis

Our patients represent a selected cohort to study AF mechanisms. In all patients, the PVs were already widely isolated and could not serve as a source of arrhythmia recurrence anymore. That makes the data interesting from a pathophysiological point of view.

The understanding of the AF pathophysiology has advanced significantly. Today structural remodeling in histoarchitecture is considered to alter cellular coupling with subsequent conduction impairment.[18–20] Affected myocardial regions are substrates for reentry—a keystone in AF development.[20,21]

Similar to patients with ventricular tachycardia such arrhythmia substrates do not necessarily locate at the endocardium.[22] After this analogy, we have studied AF substrates in a combined biatrial endo-/epicardial voltage mapping approach.

Different to patients with nonischemic ventricular tachycardia, isolated epicardial low voltage substrates were less commonly seen in our AF cohort. Seventy-nine percent of the regions with impaired voltage amplitudes showed a simultaneous endo- and epicardial mapping representation.

Nevertheless, epicardial LVA regions with normal endocardium did exist and had therapeutic implications. Fourteen percent of the patients had exclusive epicardial low voltage at the posterior RA between

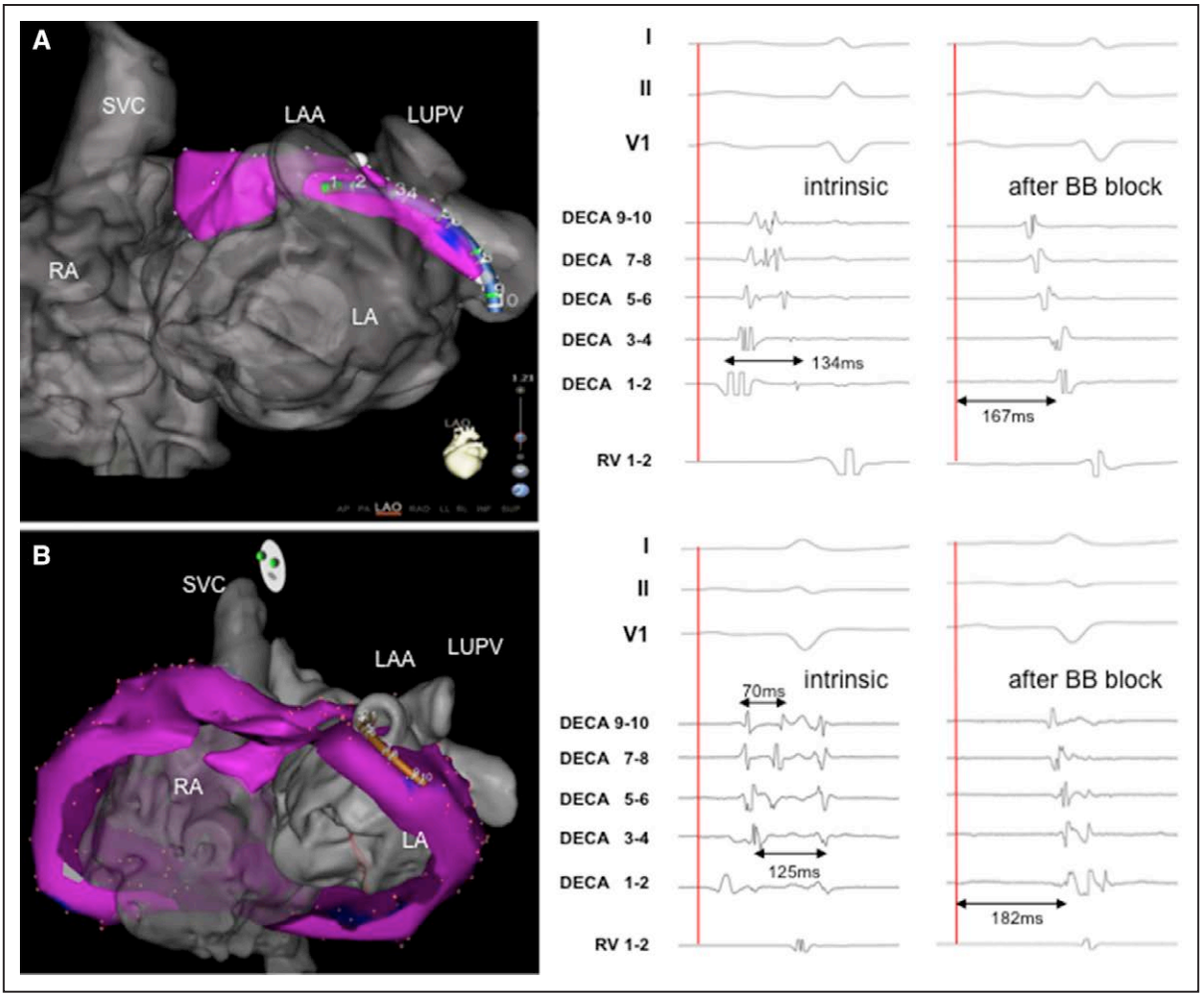

Figure 4. Case examples of patients with epicardial conduction abnormalities in sinus rhythm (SR)—associated to the epicardial fiber network.
A, The mapping catheter (DECA) is located along Ligament of Marshall at the junction to Bachmann's bundle (BB). SR electrograms are characterized by double atrial signal components with up to 134 ms conduction delay. Conduction abnormalities disappeared after ablation of BB with an anterior ablation line (BB block). **B**, The mapping catheter (DECA) is looped around the septal base of the left atrial appendage (LAA). SR electrograms are characterized by triple atrial signal components with up to 125 ms conduction delay. Conduction abnormalities disappeared after ablation of BB with an anterior ablation line (BB block). LA indicates left atrium; LUPV, left upper pulmonary vein; RA, right atrium; RV, signal from catheter in right ventricle; and SVC, superior vena cava.

SVC and IVC. These patients received an intercaval ablation line.

Epicardial Arrhythmia Sources: Fiber Network

Identification of histoarchitectural disarrangements needs to extend beyond LVA assessment. It has to consider the unique structure of the normal atrial histoarchitecture.[23] Next to the RA and LA endocardium, an epicardial fiber network provides electric interatrial connections through various anatomic routes. The bundle of Bachmann is the most well-known component along the RA and LA roof. Other pathways connect inferior and posterior. The fibers extend around the entire epicardial surface, intersect with each other, sleeve around PVs/LAA/SVC/IVC, and punctually connect to the underlying endocardium. Animal studies, computer models, and human autopsies have highlighted their potential relevance for the development of AF.[24,25] Fibrosis, anisotropic fiber orientation, slow/preferential conduction, and supernormal excitability have been reported for BB in vitro and in animals—all of which can facilitate epicardial reentry.[26] Underlining the arrhythmogenic independency of the network, electric dissociation between endo- and epicardium has been postulated in persistent AF.[27,28]

For the first time, our mapping data show potential clinical electrophysiological surrogates of the aforementioned conduction abnormalities in the epicardial fiber network in selected patients. Arrhythmias originating from localized reentry within such substrates could be mapped in 6 patients. Selective ablation of 1 limb of the interatrial

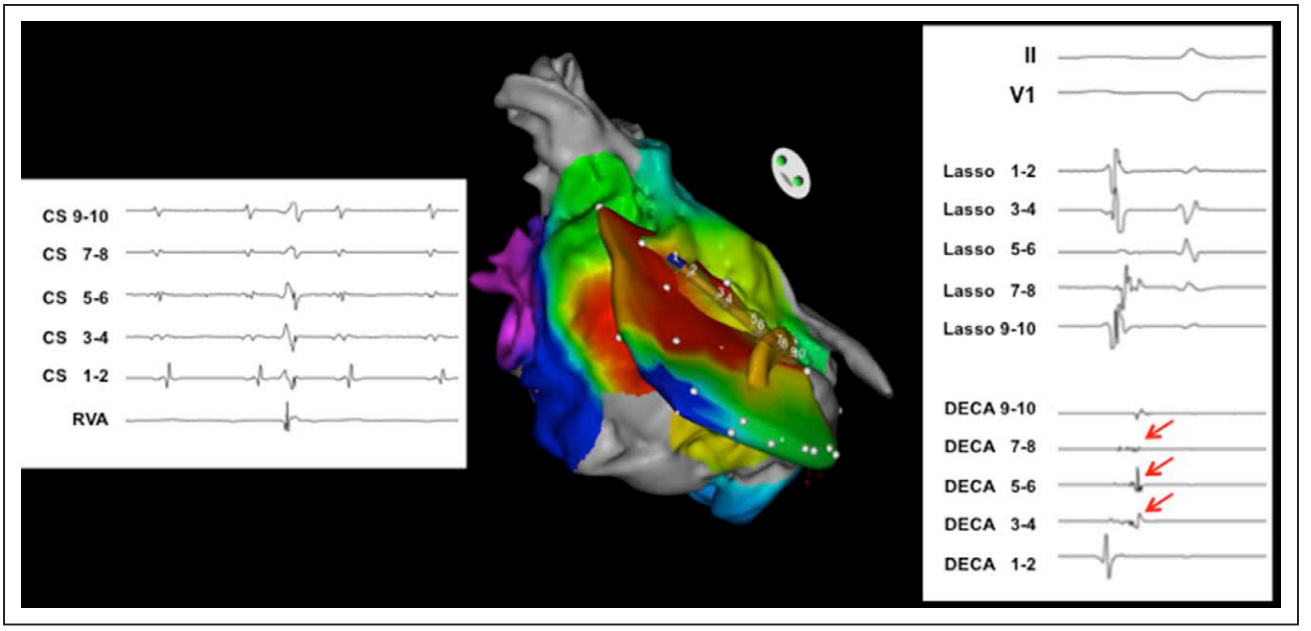

Figure 5. Case example of a patient with a localized reentry tachycardia originating from an epicardial conduction abnormality.

After electric cardioversion of persistent atrial fibrillation, the patient develops incessant regular atrial tachycardia (AT)—as seen on the coronary sinus (CS) catheter. Three-dimensional endo- and epicardial entrainment mapping reveals best return cycles epicardially—at the ridge between left upper pulmonary vein (PV) and left atrial appendage (LAA). Return cycles centrifugally deteriorate indicating a focal/localized arrhythmia origin. Mechanical manipulation of the epicardial mapping catheter (DECA) terminates AT at the junction between Ligament of Marshall and Bachmann's bundle. In sinus rhythm (SR) small, locally confined conduction abnormalities are found at the anatomic sites of best entrainment results (red arrows, DECA 3–8). These conduction abnormalities are not seen on the opposite endocardial surface (Lasso catheter positioned at LAA ostium). RVA indicates signal from catheter in right ventricular apex.

conduction (eg, BB) abolished conduction abnormalities in regions remote from the ablation. That observation raises the hypothesis of colliding SR wave fronts, which—through such areas of nonuniform conduction—may also develop large-loop epicardial reentrant circuits.[29]

Challenge of Lesion Transmurality

Traditionally, the atrial wall is considered thin enough to achieve ablation line transmurality. Only the classical mitral isthmus has been reported to be challenging.[30]

Our data illustrate the problem of ablation line transmurality also for other target regions of the LA. Overall, 38% of all lines required epicardial completion. Next to the classical mitral isthmus, the roof of the LA was a region with frequent difficulties. Thirty-three percent of the superior box lesions and 78% of the anterior LA lines needed epicardial ablation. For the latter one, endocardial line assessment through differential pacing was an additional problem. It could provide electrophysiological characteristics of completeness of block, although epicardial mapping revealed remaining conduction over BB (Figure 7).

These results are important when interpreting studies on linear lesion sets for AF catheter ablation.[8] The inability of an endocardial approach to create and control continuous and transmural linear lesions limits the outcome evaluation of such lesion sets.

Alternative Treatment Concepts

From a clinical perspective, there is no established standard to treat AF patients with permanent PV isolation.

Few smaller studies have reported treatment success after purely endocardial mapping/ablation in around 50% of such patients, respectively.[31,32] Given the discontinuous monitoring in those studies, the outcome of a pure endocardial mapping/ablation approach seems to be lower compared with our data.

On the other hand, surgical ablation has gained relevance over the years.[33] Published data result from untreated patients or patients with incomplete PVI.[10–12] Therefore, a primary goal of ablation surgery is also PV isolation. That, however, is irrelevant to our cohort. Surgical extra PV ablations are generically placed—without mapping of underlying pathology. In the light of our data, these lesions may be able to solve the issue of transmurality. They, however, are unable to treat individual disease scenarios and conduction abnormalities. Besides such principal differences, ablation surgery is more invasive.[34]

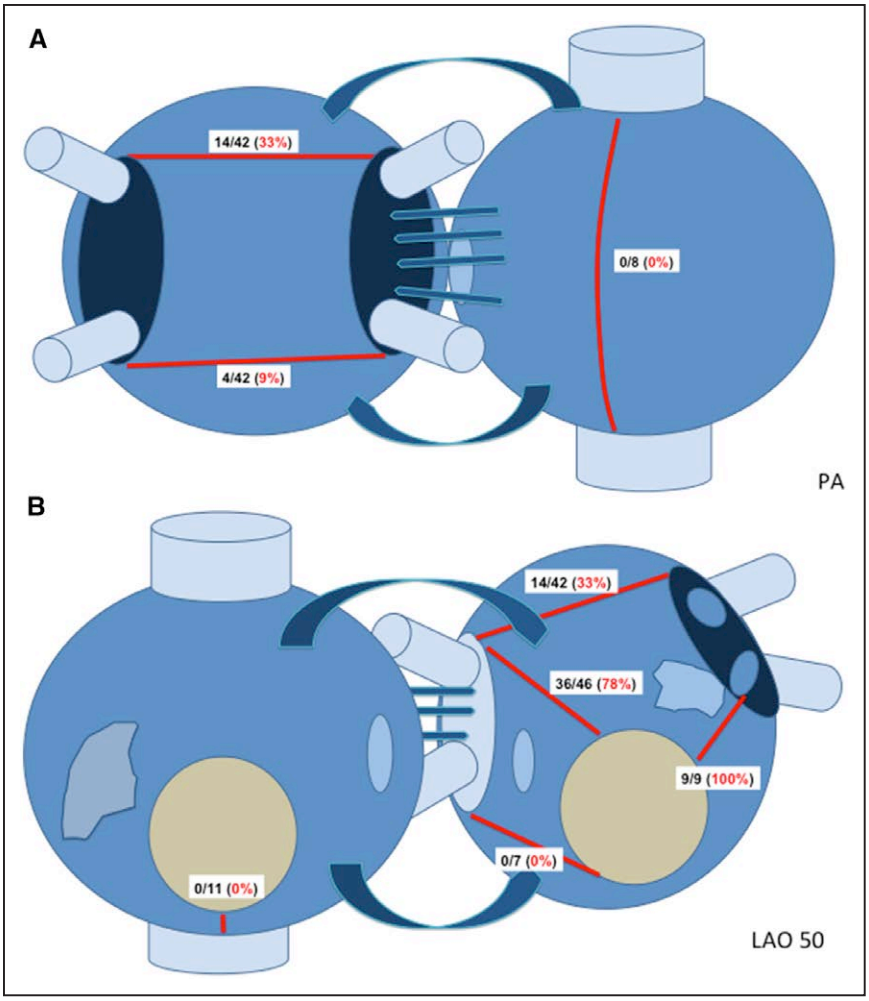

Figure 6. Schema showing the count of all biatrial ablation lines and the percentage requiring epicardial line completion. Two different projections (**A**, left anterior oblique projection [LAO]; and **B**, posterior-anterior projection [PA]) in which the atria are shown for the analysis.

Limitations

This study is an observational study. The patient cohort represents a negative selection after failed AF ablation with inherent limitations. (1) It is unclear whether a cohort of untreated AF patients would have similar difficulties with line completeness. (2) Previous ablation also produced low voltage, which may mask underlying disease processes or alter conduction in previously healthy environments. Eventually, as a next step, it is scientifically necessary to extend epicardial AF mapping into untreated patients and to study clinical outcome in a randomized design.

All procedures were performed with standard catheters for endocardial mapping and ablation. Dedicated catheter designs may improve handling, accuracy, and resolution of epicardially acquired atrial chamber maps. Similarly, technological developments facilitating interventional epicardial access could be helpful for wider clinical adoption of epicardial mapping concepts.

The substrate analysis was based on SR voltage mapping. Although this is a common method, there are limitations related to voltage threshold and epicardial fat. The latter one seems to be less relevant because typical electrogram characteristics of fat induced low voltage were not observed. The overall healthier epicardial surface about count and size of LVAs also argues against a significant impact of fat on mapping results. Voltage threshold, however, is a remaining limitation. A uniform value does not exist. Besides wall thickness and tissue properties, it is likely to be influenced by electrode spacing and mapping resolution. Apart from better understanding on such optimal values, other modalities of tissue and conduction analysis (eg, conduction velocity/direction) may improve endo- and epicardial substrate mapping in the future—during SR or even during AF.

In a minority of patients, ablation resulted in LAA isolation with following device occlusion, which increases both overall risk and cost of this procedure.

Conclusions

This is the first report on feasibility, electrophysiological findings, and clinical outcome using a combined endo-/epicardial catheter approach for mapping/ablation of AF.

In 59 patients, we showed feasibility using standard technologies of interventional catheter access, electroanatomic mapping, and radiofrequency ablation. The procedures were safe.

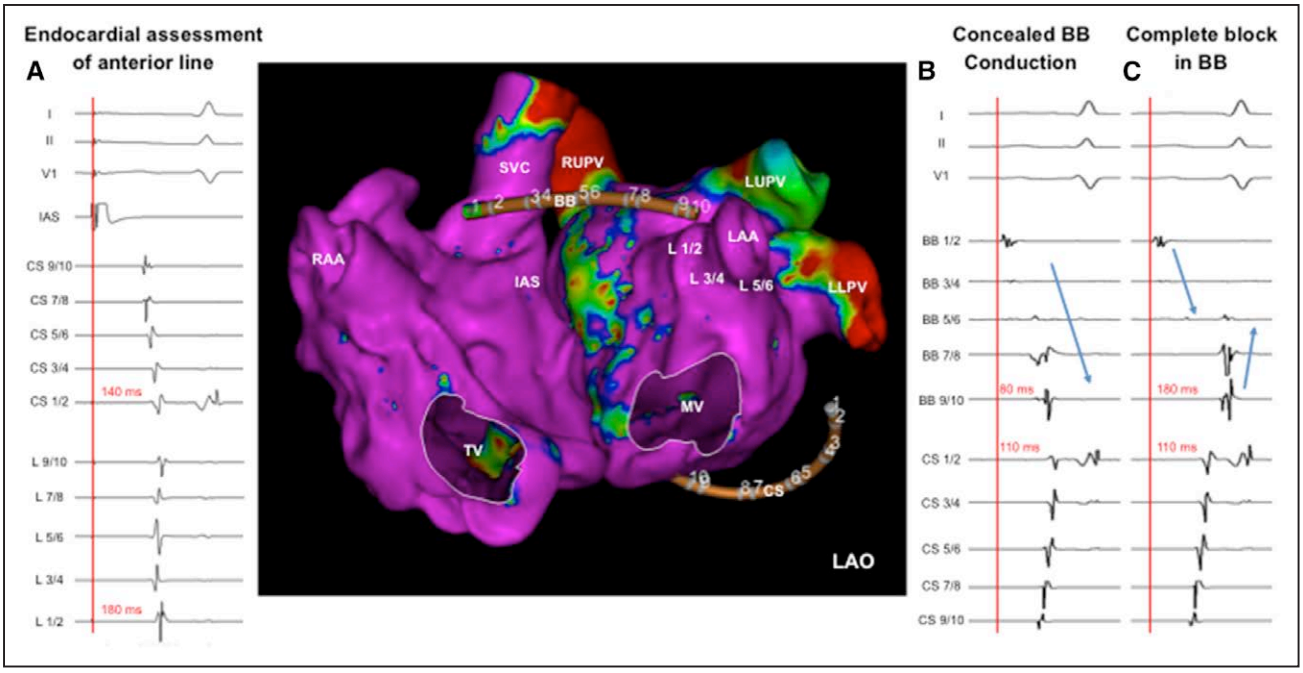

Figure 7. Case example of a patient with previous placement of an anterior line from the right upper pulmonary vein (RUPV) to the mitral valve (MV).
A, Endocardial line assessment is performed while pacing from the high right atrial septum (IAS). Activation sequence and timing on the coronary sinus catheter (CS, 1–10) and the Lasso catheter in left atrial appendage (LAA; L 1–10) are compatible with complete block. **B**, Epicardial mapping, however, reveals remaining conduction over Bachmann's bundle (BB) with only 80 ms conduction delay between right and left atrial BB insertions. **C**, Endo-/epicardial ablation achieves true line completeness with reversal of activation sequence on BB catheter, wide double potentials on BB 5/6, and an increase of conduction time to the left atrial BB insertions up to 180 ms. Timing and activation sequence of the CS catheter is not affected from presence or absence of BB block. LLPV indicates left lower PV; LUPV, left upper PV; RAA, right atrial appendage; SVC, superior vena cava; and TV, tricuspid valve.

In selected patients, epicardial mapping data gave new insights into arrhythmia mechanisms. Epicardial ablation increased transmurality of RF lesions. The approach indicates potential further development of current AF treatment. Clinical outcome in a difficult cohort of AF patients was favorable.

DISCLOSURES

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AFFILIATIONS

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FOOTNOTES

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Endo-/Epicardial Catheter Ablation of Atrial Fibrillation: Feasibility, Outcome, and Insights Into Arrhythmia Mechanisms

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