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Electrophysiological characteristics of epicardial atrial tachycardias and endocardial breakthrough site targeting for ablation: a single center experience

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Abstract

Background Despite being increasingly observed in daily practice, epicardial atrial tachycardias (Epi AT) have not been extensively characterized. In the present study, we retrospectively characterize electrophysiological properties, electroanatomic ablation targeting, and outcomes of this ablation strategy.

Methods Patients who underwent scar-based macro-reentrant left atrial tachycardia mapping and ablation patients with at least one Epi AT, which had a complete endocardial map, were selected for the inclusion. Based on current electroanatomical knowledge, Epi ATs were classified based by utilization of following epicardial structures: Bachmann's bundle, septopulmonary bundle, vein of Marshall. Endocardial breakthrough (EB) sites were analyzed as well as entrainment parameters. EB site was targeted for initial ablation.

Results Among seventy-eight patients undergoing scar-based macro-reentrant left atrial tachycardia ablation, fourteen (17.8%) patients met the inclusion criteria for Epi AT and were included in the study. Sixteen Epi ATs were mapped, four utilizing Bachmann's bundle, five utilizing septopulmonary bundle, and seven utilizing vein of Marshall. Fractionated, low amplitude signals were present at EB sites. Rf terminated the tachycardia in ten patients; activation changed in five patients and in one patient atrial fibrillation ensued. During the follow-up, there were three recurrences.

Conclusions Epicardial left atrial tachycardias are a distinct type of macro-reentrant tachycardias that can be characterized by activation and entrainment mapping, without need for epicardial access. Endocardial breakthrough site ablation reliably terminates these tachycardias with good long-term success.

Keywords Atrial tachycardia · Bachmann's bundle · Epicardial bridging · Septopulmonary bundle · Vein of Marshall

1 Introduction

The advent of high-density mapping catheters has improved the ability to understand mechanisms of atrial tachycardias [1]. The classical model of scar-based atrial tachycardia defines the tachycardia circuit as a two-dimensional structure, commonly having an anatomical obstacle such as a valve annulus or scar tissue around which atrial activation continuously propagates [2]. The continuous activation is dependent on the tachycardia isthmus, which is usually a slow conduction zone. Recently, a concept of three-dimensional tachycardia circuit has been delineated for ventricular substrate, demonstrating that the circuit components may reside at different layers of the myocardium; and therefore, mapping the endocardial surface may yield an incomplete tachycardia circuit [3]. Although the atrial tissue is generally much thinner, certain sites display increased thickness due to epicardial myofibers (such as septopulmonary bundle (SPB) and Bachmann's bundle (BB)) [4, 5]. A disease process predominately affects endocardial fibers while relatively sparing epicardial fibers, as well as ablation attempts that may lead to non-transmural lesions create the anatomical basis for the epicardial atrial tachycardia (Epi AT) substrate.

There is a scarcity of data regarding the characteristics and ablation targets of Epi ATs. In this study, we

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characterize electrophysiological properties, electroanatomic ablation targeting, and the outcomes of this ablation strategy retrospectively.

2 Methods

This was a retrospective observational study performed in a single university center. Local ethical committee has approved and supervised conduction of this study. The study was conducted in accordance with the Declaration of Helsinki.

2.1 Patient selection

All patients undergoing scar-based macro-reentrant left atrial tachycardia ablation (by three operators: EB, ATA, and OA) between 1st of January 2020 and 1st of March 2022 were screened for enrollment to the study. Patients with at least one epi AT, which had a complete endocardial map, were included. Patients whose maps were not interpretable, or entrainment maneuvers could/were not performed were excluded from the study. All patients provided a written informed consent for participation in this study.

2.2 General approach

All patients underwent the mapping and ablation procedures in a sedated state or under general anesthesia. All patients received appropriate doses of unfractionated heparin to achieve an active clotting time of 300–350 seconds. A decapolar catheter was introduced into the coronary sinus via the right femoral vein, and a double transseptal puncture was performed. Steerable long sheaths were advanced into the left atrium to stabilize catheters. Mapping was performed either with Pentaray (Biosense-Webster, Diamond Bar, California, USA) or Orion (Boston Scientific, Boston Scientific Way, MA, USA) catheters and Carto 3 (Biosense-Webster, Diamond Bar, California, USA) or Rhythmia (Boston Scientific, Boston Scientific Way, MA, USA) electroanatomic mapping systems, respectively. Left atria were mapped during AT (if necessary, ATs were induced with pacing or drugs such as isoproterenol). Patients with a history of AF and without prior ablation, as well as those undergoing posterior wall isolation underwent PVI. Ablation for AT was performed thereafter. In the case of tachycardia termination during ablation, an aggressive re-induction protocol, including up to three programmed extrastimuli and burst pacing, was employed.

2.3 Definition of the Epi atrial tachycardia and epicardial bypass fibers

Following are characteristics of Epi ATs:

1) These are macro-reentrant tachycardias confirmed by entrainment maneuvers
2) On electroanatomic map, an endocardial activation gap occurs during a certain period of the tachycardia cycle length (TCL) manifesting as an absence of electrical activity (or presence of a bystander slow activation that is not a crucial part of the circuit)
3) Epicardial "jump" of the activation: endocardial breakthrough (EB) must be anatomically related with epicardial structures. Based on previous anatomical and electrophysiological studies, we have observed three epicardial fibers as potential bypass structures for macro-reentrant circuits: (1) BB, (2) SBP, and (3) VOM (Figs. 1–3)
4) Focal automaticity or small-area reentry circuits must be excluded: breakthrough site electrocardiogram (EGM) s must be analyzed to exclude small-area reentry that may have been annotated incorrectly or may be difficult to annotate. Additionally, entrainment maneuvers should performed and concealed fusion should be observed at EB site

Tachycardias consistent with these criteria were defined as Epi ATs. Epicardial mapping was not performed in any of the patients, and VOM was not cannulated due to the absence of a dedicated small caliber (≤3F) catheter.

After completing the activation mapping, endocardial breakthrough and assumed epicardial exit sites were analyzed. Entrainment maneuvers were performed at both sites. Since both sites are assumed to be inside the circuit, the post-pacing interval was expected to be within 20 ms of the TCL.

2.4 Selection of the ablation targets

Ablation targets were selected by analyzing (1) entrainment data and (2) anatomical and electrophysiological characteristics of EB sites on the electroanatomic map. Ablation targets would ideally have a post-pacing interval within 20 ms of the TCL. Target site myo-architecture should be relatively thinner so that intramural ablation can be achieved.

Ablation was performed with either Smarttouch SF/Navistar (Biosense-Webster, Diamond Bar, California, USA) or IntellaNav Mifi (Boston Scientific, Boston Scientific Way, MA, USA) ablation catheters with power set at 35–45 W for 10–30 seconds and a contact force of 7–30 g, when available. Although tachycardia termination and non-inducibility were the procedural endpoint, ablation lesions were connected to electrically inert areas such as pulmonary

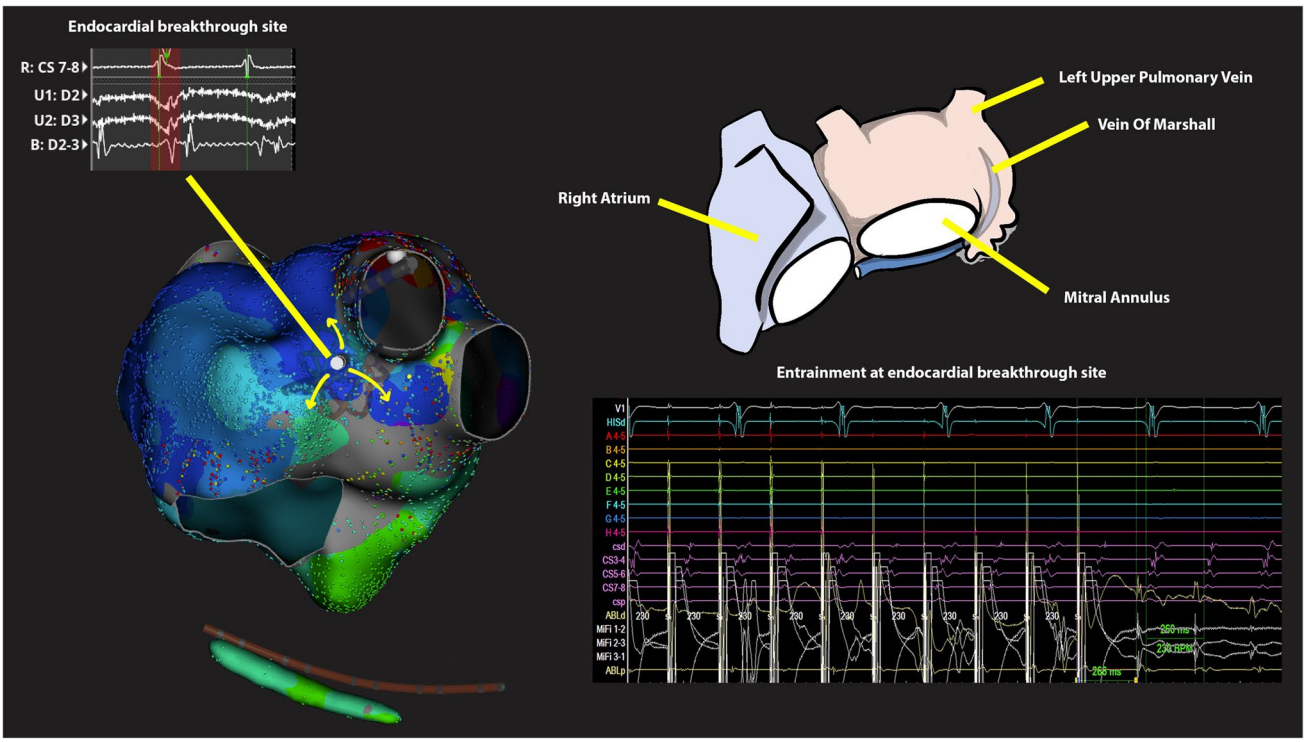

Fig. 1 A trial tachycardia presumably utilizing vein of Marshall bypass tract. Endocardial breakthrough occurs at the left atrial appendage (removed from 3D map for better visualization) ridge. Local unipolar and bipolar signals are provided on the upper left inset. This typical example shows a far-field initial component followed by a near field component, which may be attributed to the far-field epicardial component, followed by a near-field endocardial component. Entrainment maneuvers at this site (lower right) revealed concealed fusion with a post-pacing interval of 268 ms, while tachycardia cycle length is 260 ms. Ten seconds after initiation of 45 W radiofrequency led to activation change (abrupt tachycardia cycle length change along with coronary sinus activation patter change, which was later mapped to a CTI-dependent flutter)

veins or mitral annulus. All tachycardias were targeted focally at the EB sites. Additionally, ablation lesions for epicardial circuits utilizing BB were incorporated into an anterior mitral line (these patients typically have extensive anterior wall low voltage area), and lesions for circuits utilizing SPB were incorporated into posterior wall isolation lesions, despite termination and/or non-inducibility of the tachycardia. At target sites, all far-field appearing low-frequency electrocardiograms were ablated (Fig. 4).

2.5 Follow-up

Patients were followed up via clinical visits, electrocardiograms, and 24-hour ambulatory electrocardiogram recordings, when deemed clinically necessary.

2.6 Statistical analysis

IBM SPSS Statistics (version 23, for Windows) was used for the statistical analyses. Continuous variables are expressed as the group mean ± 1 SD, and categorical variables were expressed as a count and a percentage.

3 Results

3.1 Patient characteristics

Seventy-eight patients underwent scar-based macro-reentrant left atrial tachycardia ablation by three operators (EB, ATA, and OA). Among these patients, ten had uninterpretable ATs, and a total of fourteen (17.9%) patients met the inclusion criteria for epi AT and were included in the study. Mean age of the study population was 65.7 ± 11 years, and 9 (64.3%) patients were females (Table 1). Thirteen patients (92.8%) had a history of atrial fibrillation. Six patients (42.9%) had prior history of ablation, specifically catheter ablation. Four patients (28.6%) had a history of mechanical mitral valve replacement. All procedures were performed high-resolution mapping with Carto 3 ($n = 10$) and Rhythmia ($n = 4$) 3D mapping and ablation systems and their dedicated high-resolution mapping catheters.

3.2 Procedural characteristics

Twenty-two ATs were mapped in fourteen patients, sixteen (72.7%) of which were presumed to utilize with Epi ATs (Table 2). Mean procedure duration was 152 ± 20.1 minutes, and Rf duration was 18.5 ± 10 minutes. Among presumed Epi ATs, mean TCL was 288 ms ± 104, and 83.3 ms ± 26.1 of the cycle was missing which corresponded to 28.9% of TCLs (Table 3). At the end of the procedures, all patients had isolated PVs (only patient without history of atrial fibrillation underwent PVI after posterior wall isolation). No participant of the study population underwent a second procedure.

3.3 Epicardial atrial tachycardias utilizing vein of Marshall bypass

There were seven instances of Epi ATs presumed to utilize VOM bypass tract (Tables 3 and 4 and Fig. 1). The mean cycle length of these tachycardias was 271 ms ± 78, and the mean gap was 79 ms ± 30, corresponding to 29.1% of the TCL. In all seven instances, EB was invariably at left atrial appendage ridge; thereafter, pseudo-focal activation could be observed. Example of an endocardial activation is provided in the Supplemental Video 1. EB site ablation terminated the tachycardia in four patients, two patients had an activation change, and the tachycardia degenerated into AF in one patient. During the follow-up, there was only one recurrence as AT (albeit the mechanism is unknown).

3.4 Epicardial atrial tachycardias utilizing septopulmonary band bypass

There were five instances of Epi ATs presumed to utilize SPB bypass (Tables 3 and 4, Fig. 2, and Supplemental Video 1). The mean cycle length of these tachycardias was 338 ms ± 150, and the mean gap was 79 ms ± 30, corresponding to 28.9% of the TCL. These Epi ATs usually showed a pseudo-focal activation with EB usually occurring in the vicinity of right upper pulmonary vein—posterior wall junction (insertion site of SPB). Targeting EB site terminated tachycardia in two patients, and activation changed in three patients. Posterior wall was isolated in all patients regardless of tachycardia termination. During the follow-up, there was only one recurrence (both as AF and AT).

3.5 Epicardial atrial tachycardias utilizing Bachmann's bundle bypass

There were four instances of Epi ATs presumed to utilize BB bypass (Tables 3 and 4, Fig. 3, and Supplemental Video 1). The mean TCL was 280 ms ± 41, and on average, 77 ms ± 12 was missing on 3D maps. All patients had anteroseptal low voltage area, and activation suggested an epicardial jump at the low voltage area borders. Targeting epicardial jump borders terminated the arrhythmia in all patients, and there was one recurrence (as AT) during follow-up (Fig. 4).

3.6 Electrocardiogram characteristics at endocardial breakthrough sites

Endocardial breakthrough sites typically displayed fractionated long electrocardiograms. Mean duration was 52 ms ± 20 (maximal duration 112 ms and minimal duration 27 ms). Mean electrocardiogram voltage was 0.2 mV ± 0.11. We have observed a dual-component electrocardiogram with far-field component presumed to be of epicardial origin, and a near-field component presumed to be of endocardial origin (Fig. 1, inset), although this was not uniform.

3.7 Clinical follow-up

Mean follow-up was 10 months ± 5, with the shortest duration being 4 months and the longest being 18 months (Table 2). No patients were lost at the follow-up. Recurrences occurred as AF and AT in 1 patient and as AT in two patients. The mechanism of recurrent ATs has not been established.

4 Discussion

The major points of this study are (Fig. 5):

- Epi ATs are defined as (1) macro-reentrant ATs displaying an activation gap on endocardial map, (2) the activation of the AT disappears at one of the anatomical epicardial structures (BB, VOM, and SPB) to reappear at one of the other insertions of this structure, (3) at both sites PPI-TCL is within 20 ms, and (4) endocardial recordings where activation "jump" is occurring usually show no near field signals
- Epi ATs can be successfully targeted by ablation of the EB site

4.1 Pathophysiology of epicardial left atrial tachycardias

Anatomical studies have revealed that epicardial fibers that run across the left atrium increase the thickness of certain portions of the atrial walls [6]. A fibrotic disease process or iatrogenic ablation lesions may lead to non-transmural lesions with slow conduction that provide a nidus for reentrant ATs. Low bipolar voltage on the anterior and posterior left atrial walls should by no means imply that a transmural scar is present since the correlation between low bipolar

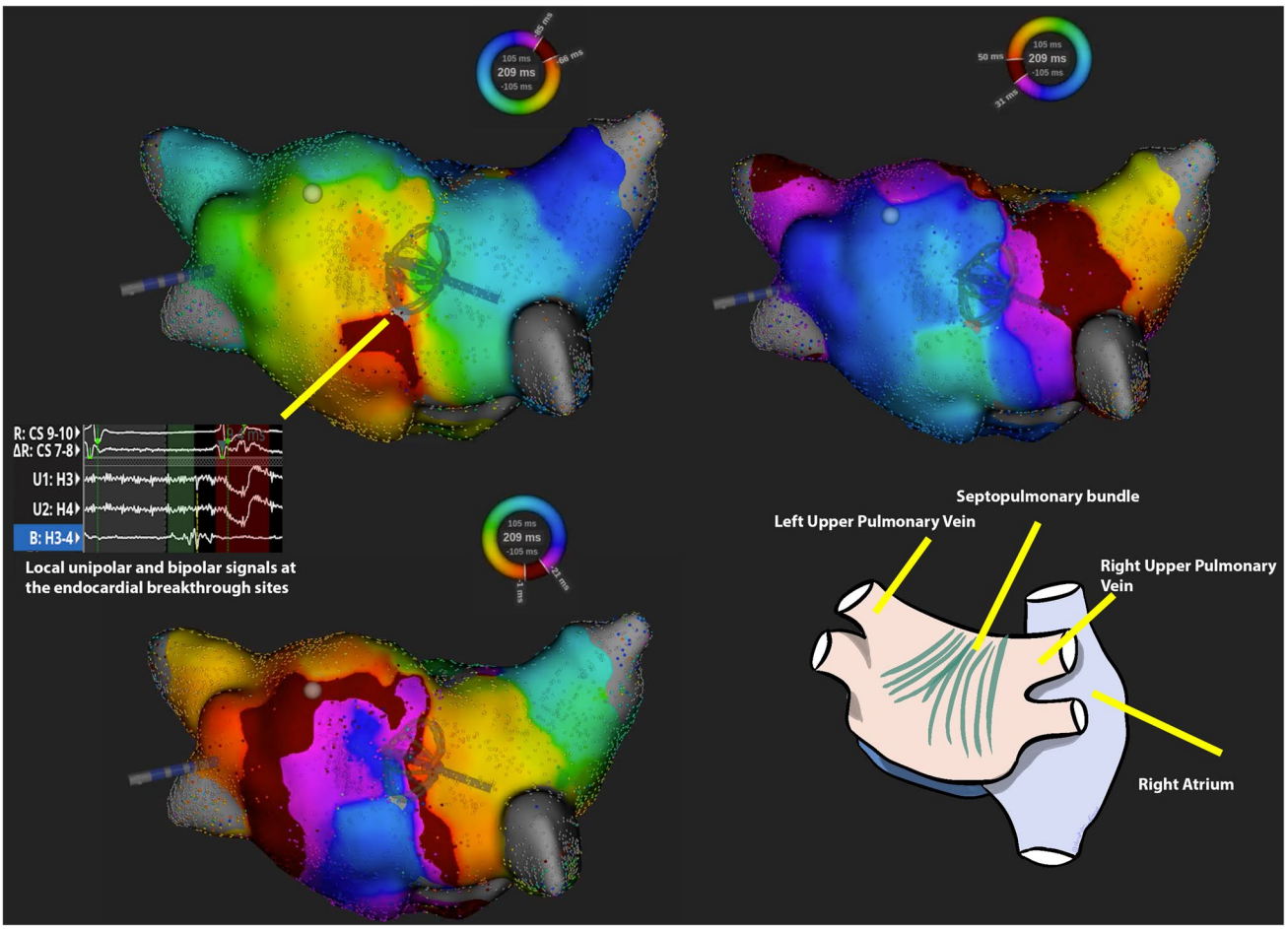

Fig. 2 Atrial tachycardia presumably utilizing septopulmonary bundle. Left and right upper figures display left atrial activation map in posterior view. Endocardial breakthrough occurs in the vicinity of inferior pulmonary veins, and activation of the left atrium continues in pseudo-focal fashion (lower left, then upper right). Probable epicardial activation utilizing septopulmonary bundle is depicted with an arrow. Complete endocardial propagation is available on Supplemental Video 1. Unipolar and bipolar endocardial electrograms are provided in the inset. Ablation at the endocardial breakthrough site led to activation change; nevertheless, the posterior wall was isolated at the discretion of the operators, and isolation was confirmed at the end of the procedure by absence of capture during high output pacing

voltage and late gadolinium enhancement is limited to 2 mm tissue depth [7]. Therefore, epicardial bridging is possible if activation and entrainment mapping suggest a possible circuit. In our study population, we have observed three typical patterns of presumed Epi ATs which we have pragmatically classified as BB, VOM, and SPB utilizing tachycardias, based on previous reports [8–10]. This report certainly does not exclude other possible epicardial bypass pathways or tachycardia circuits. Left atrial walls have complex architecture with variable epicardial layers and additional connections such as septoatrial bundle [8]. Additionally, structures such as left lateral ridge have extensions of BB fibers, VOM, SBP, and septoatrial bundle [9]. Therefore, our classification, although clinically relevant, is a simplification of a very complex atrial architecture.

Certain features are present on electroanatomic map that suggests a possibility of an Epi AT. First, the missing TCL portion on the map histogram in appropriately annotated macro-reentrant AT suggests that either (a) the tachycardia is not macro-reentrant, (b) the tachycardia is not of a left atrial, (c) the tachycardia circuit is not completely endocardial, or (d) incomplete mapping and/or small near-field signals that cannot be detected by current mapping catheter technology. Since we have excluded patients with incomplete/uninterpretable maps and confirmed the macro-reentrant mechanism and left atrial origin of ATs in patients that were included in the study, the circuit not being fully endocardial is the only plausible answer. Second, we have observed a pseudo-focal activation pattern frequently on BB, VOM, and SPB dependent atrial tachycardias. Nakatani et al. have similarly observed this activation pattern in patients with epicardial bypass ATs [10]. Pseudo-focal activation pattern can be commonly observed in focal (including micro-reentrant) ATs. In this

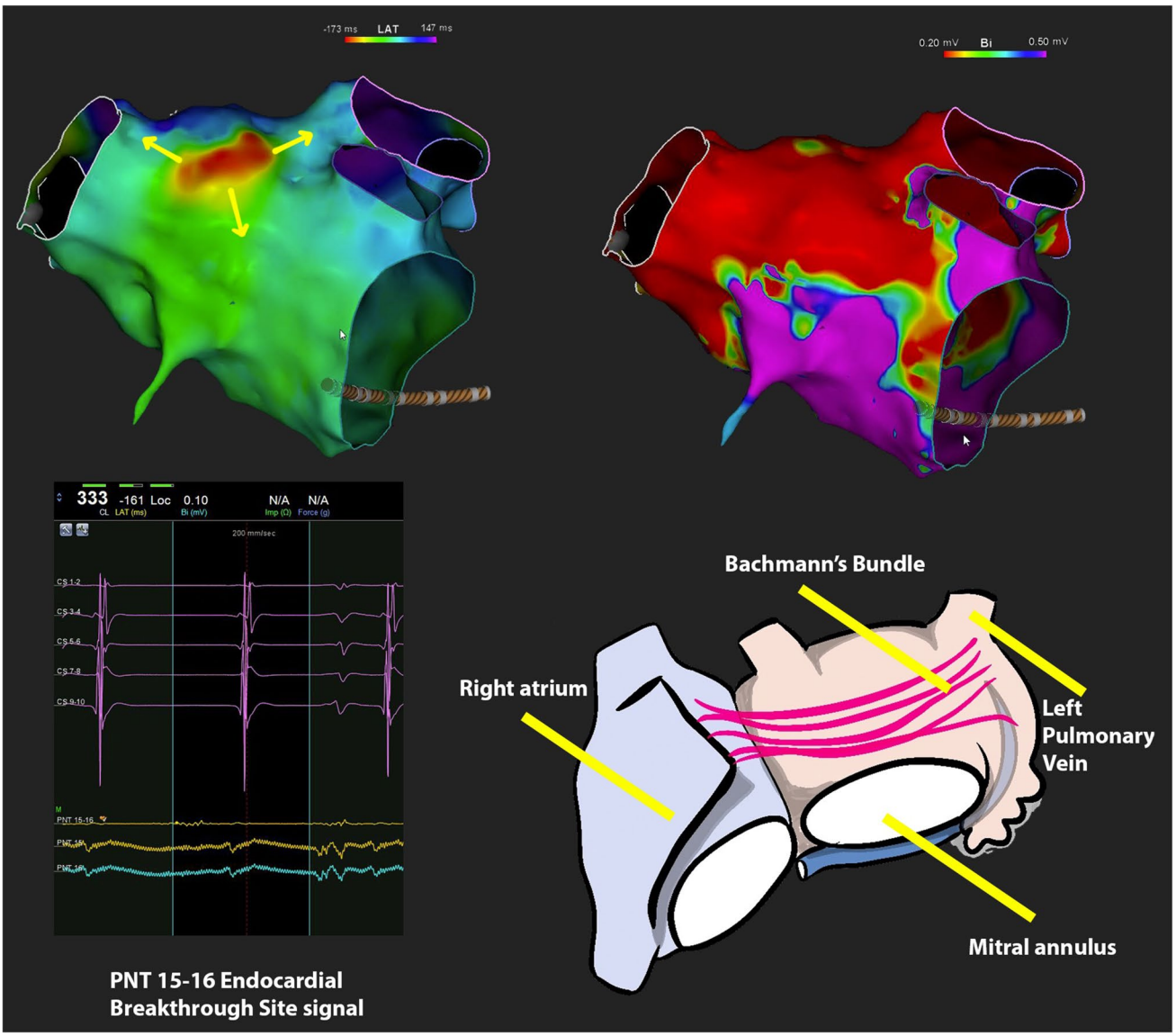

Fig. 3 Atrial tachycardia utilizing Bachmann's bundle bypass. Left upper figure depict earliest activation (endocardial breakthrough) site occurring near the left atrial appendage. Left atrium activates in the pseudo-focal nature (arrows). After the whole left atrium has been activated, activation re-appears at the endocardial breakthrough site (Bachmann's bundle bypass explains this activation). Complete endocardial propagation is available at the Supplemental Video 1. Right upper figure depicts low voltage area at the anterior left atrial wall. The left lower figure depicts endocardial breakthrough site electrocardiograms. Schematic representation of Bachmann's bundle is provided at the lower right figure. Ablation at endocardial breakthrough site terminated the tachycardia

study, we report that, like Nakatani et al., this activation pattern is consistent with Epi AT, but only after the macro-reentrant mechanism is proven by entrainment mapping. The electrophysiologic discrimination of focal-appearing activation on electroanatomical maps has been elegantly described by Takigawa et al. [11]. In this report, local electrocardiogram characteristics (such as sharp QS on unipolar) and proprietary software measurements provide good discrimination between true-focal and pseudo-focal tachycardias. Entrainment maneuvers are still crucial in differentiating tachycardia mechanisms.

4.2 Previous reports on epicardial left atrial tachycardias

There have been previous reports on epicardial LATs. One of the pioneering reports on epicardial LATs by Garcia et al. showed two cases of LATs that could be presumably explained by septopulmonary bundle [12]. The authors report roof-dependent tachycardias on previously isolated posterior walls with no near-field electrograms. They emphasized high-output pacing, which yielded concealed entrainment, and targeted the tachycardias by placing new

Table 1 Baseline clinical characteristics

Age, years	65.7±11
Gender,	
Female *n* (%)	9 (64.3)
Diabetes mellitus, - *n* (%)	6 (42.9)
Hypertension, - *n* (%)	2 (14.3)
Coronary artery disease, - *n* (%)	4 (28.6)
Smoking, - *n* (%)	
Prior	7 (50)
Current	0 (0)
No smoking history	7 (50)
Prosthetic mitral valve, - *n* (%)	4 (28.6)
Prior stroke, - *n* (%)	1 (7.1)
Left atrial diameter, - *n* (%)	50.2 ± 6.8
Left ventricular ejection fraction, -%	53.6 ± 9.9
Atrial fibrillation , - *n* (%)	13 (92.8)
Anticoagulation, - *n* (%)	
Warfarin	4 (28.6)
Apixaban	5 (35.7)
Dabigatran	0 (0)
Edoxaban	0 (0)
Rivaroxaban	5 (35.7)
BMI, kg/m^2	27.2 ± 3.5
Prior ablation history (AF, or AT), - *n* (%)	6 (42.9)

Table 2 Procedural characteristics

Mean number of ATs per patient	1.43 (1–3)
Total number of epicardial AT	16
Mean per patient	1.14 (1–2)
Procedure duration, minutes	152 ± 20.1
Rf time, minutes	18.5 ± 10
Left atrial volume*, ml	153.2 ± 16.7
Follow-up, months	
Mean	10 ± 5
Minimum	4
Maximum	18

AT, atrial tachycardia; *Rf*, radiofrequency; *as reported by 3-dimensional mapping systems

roof or floor lines or far-field signals inside isolated posterior walls. We have targeted EB sites; however, even after tachycardias terminated or new tachycardias ensued, we have targeted complete posterior wall isolation, confirmed by high-output pacing. Of note, posterior wall isolation in SPB dependent Epi Ats was performed at operator discretion with the aim of decreasing long-term recurrence, despite the lack of definite evidence. Nakatani et al. have demonstrated properties of ATs utilizing BB, VOM, and SPB epicardial bypass tracts, as well as coronary sinus-great cardiac vein and fossa ovalis tracts [10]. Similarly to our study, entrainment mapping has been crucial in understanding the mechanism of LATs. The main difference between the present study and that of Nakatani et al. is ablation target selection. In our study, we have repeatedly found that EB sites were successful in terminating ATs, while the EB targeting led to AT termination in only 1 out of 5 patients in the study by Nakatani et al. This difference may be explained by several factors. First, there was a small sample (only 5 patients vs. 13 patients) where EB ablation was targeted. Second, power settings and duration may have varied: we have employed 45 Watts for at least 20 seconds uniformly; however, specific power settings on EB ablation targets are not known. Nakatani et al. have successfully terminated VOM dependent LATs in two patients. While we agree that is a reasonable approach, we have reserved it for failed EB ablation due to potential increase in fluoroscopy time, procedure duration, and complications. We acknowledge that isthmus targeting is a good approach; however, defining epicardial isthmus may not be possible without mapping the epicardium. Even if an epicardial approach is attempted, certain sites may not be accessible due to anatomical constraints.

Epicardial bridging has been reported in another study by Nayak et al. [13]. In this study, the authors have described potential sites, three of which are the same as in our study, with an additional site being the coronary sinus. In this study, percutaneous approach for epicardial mapping yielded direct evidence for bridging. Similar to our study, Nayak et al. have targeted adjacent endocardial sites, with good acute and long-term outcome. Interestingly, they report failure of endocardial Rf ablation in a subset of patients despite prolonged Rf duration (up to 100 s), which contrasts our study since we have observed tachycardia termination/activation change usually in less than 20 s after Rf initiation. In this small subset of patients, Nayak et al. report that epicardial mapping provided direct evidence of participation in the circuit and Rf effectively terminated ATs.

In a recent study on anterior wall LATs, four patients with epicardial bypass LATs were reported [14]. The authors have reported that one patient had LAT terminated, two patients' activation had changed, and one patient necessitated cardioversion, after targeting EB sites of BB. The authors have extensively mapped the anterior wall for possible line gap, which would exclude Epi AT. Additionally, we have demonstrated that Epi ATs are possible in *de novo* patients with no prior history of catheter or surgical ablation.

Recently, Smietan et al. have demonstrated an interesting concept of "natural surface epicardial mapping" exploiting anatomical relation between pulmonary artery branches and epicardial LA, which obviates the risks of percutaneous epicardial mapping and pericardial constrains [15]. They report successful entrainment which provides elegant proof of epicardial component in these tachycardia circuits.

Table 3 Tachycardia properties and outcomes

	All epicardial ATs $N = 16$	VOM dependent ATs $N = 7$	SPB dependent ATs $N = 5$	BB dependent ATs $N = 4$
Tachycardia CL (ms)	288 ± 104	271 ± 78	338 ± 150	280 ± 41
Gap (ms)	83.3 ± 26.1	79 ± 30	94 ± 30	77 ± 12
Gap % of CL	28.9	29.1	28.9	27.5
Termination				
Rf	10	4	2	4
Activation change	5	2	3	0
AF degeneration	1	1	0	0
Cardioversion	0	0	0	0
Recurrence	3 (18.7)	1 (14.2)	1 (20)	1 (25)

AF, atrial fibrillation; *BB*, Bachmann's bundle; *Rf*, radiofrequency; *SPB*, septopulmonary bundle; *TCL*, tachycardia cycle length; *VOM*, vein of Marshall

Table 4 Atrial tachycardia details

Tachycardia #	Patient #	Type	CL (ms)	Gap (ms)	Additional details
1	1	BB	280	80	Anteroseptal scar. 0.11 mV, 80 ms EGM and 0.16 mV, 40 ms EGM at endocardial borders. Rf at former site terminated the AT.
2	2	VOM	230	100	Activation starts at LAA ridge and LA activates in pseudo-focal fashion. 0.3 mV, 40 ms EGM at the breakthrough site. During Rf at breakthrough site tachycardia degenerates into AF.
3	3	SBP	247	70	Pseudo-focal activation at posterior RUPV. Low voltage area medial to activation site. Exit site EGM: 0.52 mV, 27 ms. Rf at breakthrough site leads to activation change (i.e., abrupt CL slowing and CS activation change to proximal-to-distal): CTI-dependent flutter. PW was isolated.
4	4	VOM	247	50	Pseudo-focal activation at LAA ridge. 0.32 mV, 27 ms EGM at breakthrough site. Breakthrough site Rf terminates.
5	5	SPB	255	70	Midline posterior wall low voltage area. Focal breakthrough next to posterior RUPV. 0.25 mV, 50 ms EGM. Breakthrough site Rf terminated the tachycardia. PW was isolated.
6	6	SPB	408	85	Midline posterior wall low voltage are. Pseudo-focal breakthrough next to RUPV with 112 ms, 0.13 mV EGM at that site. Breakthrough site Rf terminated the tachycardia.
7	7	VOM	212	60	Pseudo-focal activation at LAA ridge; 0.17 mV, 46 ms EGM at breakthrough site. RF there terminated the tachycardia.
8	8	SPB	210	103	Pseudo-focal exit at near RIPV, 0.07 mV, 80 ms EGM. Rf at breakthrough led to activation change (i.e., abrupt CL prolongation from 210 ms to 260 ms and CS activation pattern changed tachycardia #9).
9	8	VOM	260	108	LAA ridge breakthrough and pseudo-focal activation. 0.08 mV, 70 ms EGM at breakthrough site; Rf led to activation change (i.e., CL decreased to 230ms abruptly, remap showed a CTI-dependent flutter).
10	9	VOM	370	70	LAA ridge breakthrough, 0.35 mV, 45 ms EGM. Rf at breakthrough site terminated the tachycardia.
11	10	SPB	570	143	Endocardial posterior wall is a low voltage area. Pseudo-focal activation near RUPV. 50 ms, 0.1 mV EGM at breakthrough site. Rf here led to activation change (i.e., abrupt decrease in CL and CS activation pattern change, tachycardia #12).
12	10	VOM	390	120	LAA ridge breakthrough site has a 0.13 mV, 81 ms EGM. Rf at this site led to activation change (i.e., CL decrease to 250 ms, remap revealed a CTI-dependent flutter).
13	11	BB	280	89	Anteroseptal low voltage area. Targeting endocardial breakthrough site terminated the perimitral flutter.
14	12	VOM	190	45	LAA ridge pseudo-focal breakthrough. 0.25 mV, 60 ms EGM at this site. Rf terminated the tachycardia.
15	13	BB	330	60	Epicardial jump over BB, breakthrough site EGM 0.12 mV, 50 ms. Breakthrough site ablation terminated the tachycardia.
16	14	BB	230	80	CL = 230 ms, gap = 80 ms. Epicardial jump over BB, perimitral tachycardia. Anteroseptal low voltage area. Endocardial lateral breakthrough site EGM 0.1 mV, 60 ms. Rf at this siter terminated the tachycardia.

AF, atrial fibrillation; *BB*, Bachmann's bundle; *CL*, cycle length; *CS*, coronary sinus; *EGM*, electrocardiogram; *LAA*, left atrial appendage; *Rf*: radiofrequency; *RIPV*, right inferior pulmonary vein; *RUPV*, right upper pulmonary vein; *SPB*, septopulmonary bundle; *VOM*, vein of Marshall

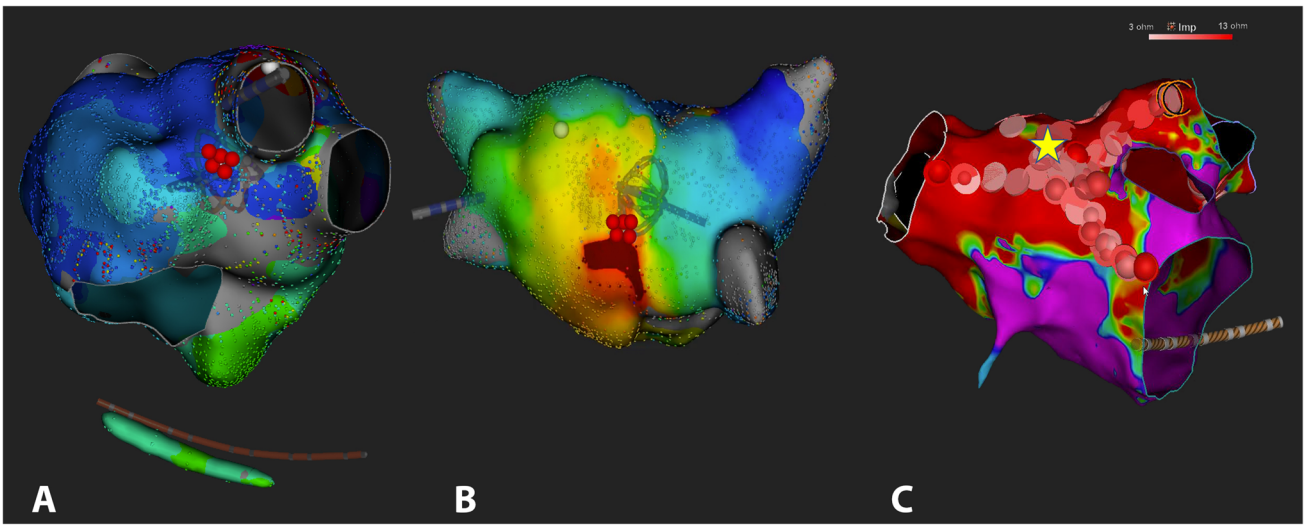

Fig. 4 Typical lesion sets for atrial tachycardias utilizing epicardial bypass tracts. (**A**) Atrial tachycardia thought to utilize vein of Marshall bypass tract. Endocardial breakthrough site (where post-pacing interval was within 10–20 milliseconds of cycle length) was targeted. (**B**) Atrial tachycardia presumed to utilize septopulmonary bundle bypass tract. Again, endocardial breakthrough site was targeted. These lesions were always incorporated into the posterior wall isolation lesions (not shown at this figure). (**C**) Endocardial breakthrough site (star) of the putative Bachmann's bundle dependent tachycardia was targeted for initial tachycardia termination. These lesions were incorporated into anterior mitral line aiming to eliminate all near-field and far-field appearing electrocardiograms

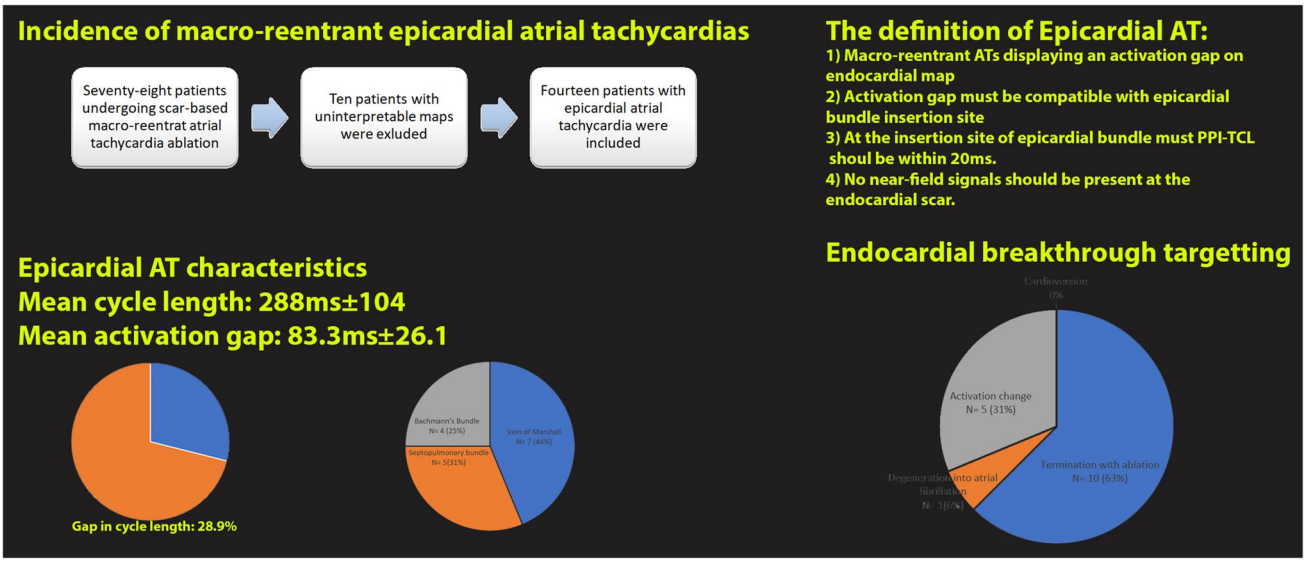

Fig. 5 The main findings of the study.

4.3 Limitations of the study

There are several limitations to this study that merit consideration. First, epicardial mapping was not performed due to associated risks in anticoagulated patients and limited incremental yield. VOM mapping was not performed due to absence of a dedicated small caliber catheter. All data presented in this study are derived from endocardial voltage, activation, and entrainment mapping; therefore, tachycardia circuits are deduced from these data. Second, small endocardial signals occurring in an incomplete line of block may have been missed; however, this is unlikely due to use of high-resolution mapping catheters and extensive mapping procedure. Third, sample size is small and follow-up duration

is not uniform reflecting relative rareness of this clinical diagnosis. Additionally, the incidence of Epi ATs may have been underestimated due to a selection bias (excluded patients with uninterpretable/incomplete maps have been excluded). Finally, this is a retrospective single center experience inheriting all limitations of its design nature.

5 Conclusion

Epicardial left atrial tachycardias are a distinct type of macro-reentrant tachycardia that can be characterized by activation and entrainment mapping without the need for epicardial access. EB site ablation reliably terminates these tachycardias with acceptable long-term success.

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Declarations

Conflict of interest The authors declare no competing interests.

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