Dispersion electrogram detection with an artificial intelligence software in redo paroxysmal atrial fibrillation ablation

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Introduction

Atrial fibrillation (AF) is a sustained cardiac arrhythmia whose drivers and mechanisms are complex and incompletely understood. The most current estimates predict that up to 12 million people in the United States will be affected by 2050.[1] Pulmonary vein isolation (PVI) is the cornerstone of treatment for paroxysmal AF but has lower efficacy in persistent AF, where the drivers perpetuating AF often lie outside the pulmonary veins (PV).[2–4]

Certain ablation strategies for AF treatment aim to target AF drivers.[5] Recently, a signature of electrical AF drivers has been demonstrated and defined as "dispersion."[6] Dispersion areas are described as local clusters of electrograms that may or may not be fractionated, and they display interelectrode time and space dispersion such that activation is spread over the entire AF cycle length (Figure 1). Studies have shown a high degree of acute termination with a low level of AF recurrence in persistent/long-standing persistent AF patients undergoing dispersion-based ablation.[6]

As visual dispersion mapping is challenging to perform, a real-time artificial intelligence (AI) software (VX1; Volta Medical, Marseille, France) compatible with current electrophysiology mapping systems and trained to determine multipolar electrogram dispersion has been developed.[7] We report the first redo paroxysmal AF procedures using the AI software, and compare them to our initial 2 persistent AF patients.

KEY TEACHING POINTS

- VX1 (Volta Medical) is an artificial intelligence (AI)-based software solution designed to obtain real-time adjudications of multipolar electrograms.
- This software-based mapping approach may be integrated in the workflow of patients admitted for both de novo persistent and redo paroxysmal atrial fibrillation (AF).
- Future work needs to establish whether AI protocols, algorithms, and software solutions will need to be adjusted based on procedural history and AF types.
- In the cases presented herein, noninducibility and favorable outcomes were achieved after a relatively extensive ablation set. Future studies will need to establish whether a standardized ablation vs patient-tailored set may suffice to achieve satisfactory outcomes.

Case report

vAF ablation was performed in the usual fashion, briefly described as follows. Patients were placed under general anesthesia and vascular access was obtained in the femoral veins. A multi-pole catheter was placed into the coronary sinus, and a single transseptal puncture was performed under intracardiac echocardiography guidance after administration of intravenous heparin, to maintain an activated clotting time of 350–400 seconds. Esophageal temperature was monitored, using the Esosure esophageal deviation tool if necessary. Mapping was performed with a PENTARAY catheter (Biosense Webster, Irvine, CA). The AI software (VX1; Volta Medical) user interface (Figure 1) was placed on a mobile table visible to the operator with a separate screen for the mapping technician. During initial anatomic mapping, dispersion points were collected by placing the catheter in one position for 3–4 seconds and manually tagging points the system determined to be dispersed with a very high likelihood (red signal) or high likelihood (orange signal). Biatrial mapping took 20–30 minutes. Ablation was performed

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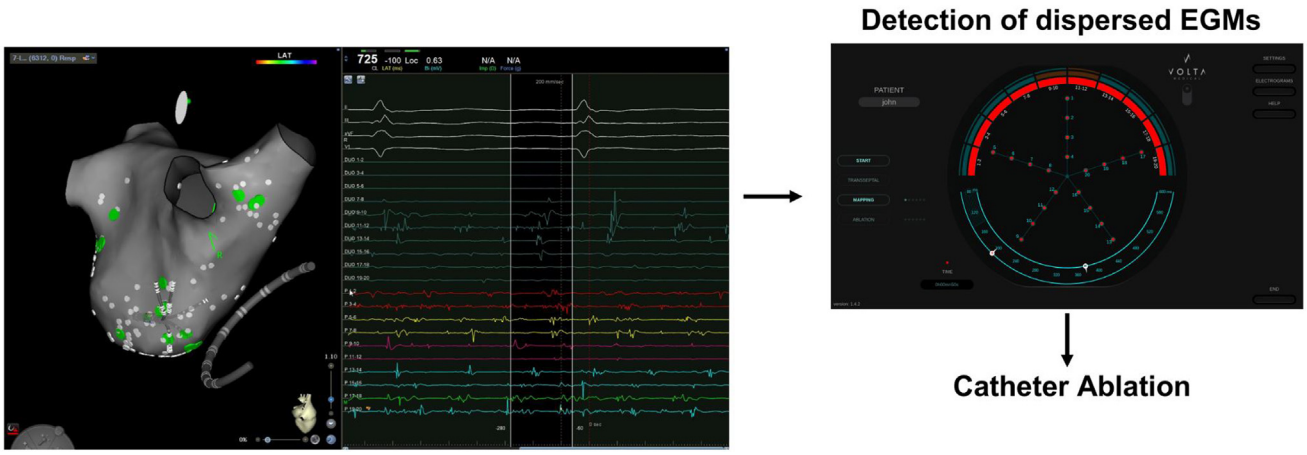

Figure 1 Spatiotemporal dispersion of multipolar electrograms (EGMs), detection with AI software (VX1; Volta Medical, Marseille, France), and catheter ablation. Left panel displays examples of dispersed EGMs from patient 1. EGMs are processed in real time by VX1 system (VX1 interface right panel) on 1.5-second signals and are considered dispersed when they display 1 continuously fractionated electrogram with a cumulative duration longer than 80% of the 1.5-second window. Stable intracardiac atrial dispersed electrograms are identified by VX1 algorithm on each dipole 1-2 to 19-20 (the dipoles schematized in the upper half cycle are blinking red). The lower part of the frame displays the average cycle length as recorded by the reference catheter (R) and the mapping catheter (M).

using a Biosense Webster ST/SF ablation catheter at 45 W, targeting a Surpoint ablation index of 350–500 depending on the anatomic site. Large areas with dense dispersion areas were encircled; PVI was performed after initial dispersion ablation sets. For patients presenting in sinus rhythm, burst pacing in the atrium was performed down to 160 ms or failure to capture. If that failed to induce sustained AF, isuprel was initiated at 2 mcg/min with repeated pacing for induction and titrated to 10 mcg/min as necessary for induction. Isuprel was discontinued several minutes prior to mapping. AF ablation was performed in 4 patients (Table 1).

Patient 1

The first patient with paroxysmal AF is a 68-year-old man with hyperlipidemia and AF, who underwent PVI in 2017. The patient had early recurrence after several months but mild symptoms. Rhythm was monitored daily using a Kardia monitor, with burden of 20%–40%. Fatigue and exercise intolerance experienced by the patient led to a repeat ablation. The patient was in and out of AF during mapping. Left superior PV and both right PVs were reconnected. Dispersion mapping during AF defined regions of dispersion in the low posterior left atrium (LA) inferior to the left inferior PV, in the right carina, and anteriorly between the right superior PV and mitral annulus. Dispersion was noted at the coronary sinus ostium. Targeted ablation of dispersion areas in the LA was first performed, after which paroxysms of AF were no longer seen. The PVs were then reisolated. Rapid atrial pacing failed to reinduce any arrhythmia (Figure 2A). At follow-up to 1 year, the patient had no reported symptoms or evidence of AF on daily Kardia recordings.

Patient 2

The second patient with paroxysmal AF is a 53-year-old man with prior mild cerebrovascular accident and persistent AF who had undergone ablation in 2016 (Table 1). The patient had presented again with a small cerebrovascular accident after having been off anticoagulation, and was found to have recurrent AF with very rapid ventricular response and palpitations. The patient presented for ablation in normal sinus rhythm (NSR) and sustained AF was induced during isoproterenol infusion. Dispersion areas were found in the low posterior wall, anterior to the right superior PV, and in the reconnected right carina (the right PVs were reconnected; the left PVs remained isolated). Termination of AF occurred during ablation at the right carina. The remainder of the dispersion areas were ablated, and the right veins were then reisolated (Figure 2B). The AI software mapped points and electrograms at the most dispersed region and site of termination are shown in Figure 3. Because the patient had remained paroxysmal, there was PV reconnection and significant dispersion in the right PV carina with termination of induced AF at the most dispersed region, the right atrium was not mapped. The patient was not inducible at the conclusion of the case. The patient has not had symptomatic recurrence.

Patient 3

The third patient is a 65-year-old man who had long-standing persistent AF and history of coronary artery disease (Table 1). The patient reported an initial diagnosis of AF about 10 years prior to evaluation and was paroxysmal at first, but AF was continuous for the last 2 years. The patient described exertional dyspnea and palpitations. Echo demonstrated moderate biatrial enlargement without significant valvular disease and normal ejection fraction. Dispersion mapping noted extensive dispersion in the LA, with limited areas in the right atrial septum. Dispersion regions in the LA roof, anterior wall, and inferoposterior wall were ablated. PV were then isolated, resulting in posterior wall isolation. Right atrial dispersion was noted near the atrioventiruclar

Table 1 Patient characteristics, surface area, and dispersion quantification

Patient characteristics	Patient 1	Patient 2	Patient 3	Patient 4
Age (years)	68	53	65	61
Sex	Male	Male	Male	Male
Race	White	White	Asian	White
Cardiovascular risk factors				
Hypertension	Yes	Yes	Yes	Yes
Hyperlipidemia	Yes	Yes	Yes	Yes
Diabetes mellitus	No	No	Yes	No
Prior stroke	No	Yes	No	No
BMI	29.4	25.5	23.0	26.5
Medications				
Anticoagulation	Xarelto	Eliquis	Xarelto	Eliquis
Antiarrhythmics	Amiodarone	-	-	Amiodarone
Beta-blockers	Yes	Yes	Yes	Yes
Structural heart disease	None	None	None	None
Coronary artery disease	No	No	Yes	Yes
Atrial fibrillation type	Recurrent PAF	Recurrent PAF	LS persistent AF	Persistent AF
AF history (years)	4	5	10	7
Arrhythmia max duration (months)	-	-	24	-
Prior ablation	PVI	PVI	None	None
Presenting symptom	Palpitations	CVA	Palpitations	Heart failure
Echocardiogram	Normal RV function	Normal RV function	Mild RV dysfunction	Normal RV function
LVEF (%)	55–60	30–35	50–55	30–35
Surface area (cm^2)				
Right atrium	190	N/A	153	178
Left atrium	209	137	232	185
Biatrial	399	N/A	385	363
Dispersion sites	54	30	205	268
Left atrial ablation	101	54	145	109
Dispersion points (number)				
Right atrium	50	0	18	64
Left atrium	188	43	170	267
Dispersion ablation regions (number)	3	5	6	4

AF = atrial fibrillation; BMI = body mass index; CVA = cerebrovascular accident; LS = long-standing; LVEF = left ventricular ejection fraction; N/A = not applicable; PAF = paroxysmal atrial fibrillation; PVI = pulmonary vein isolation; RV = right ventricle.

node and limited ablation was undertaken (Figure 2C). The patient was cardioverted at the end of the procedure. At follow-up the patient developed recurrent atrial tachycardia requiring reablation; he has maintained NSR on dofetilide.

Patient 4

The last patient was a 61-year-old man who had persistent AF and history of severe cardiomyopathy related to uncontrolled ventricular rate (Table 1). AF was first diagnosed in 2012 but progressed to persistent AF, and cardioversion was required in 2014 and 2016. Recently he suffered a recurrence, this time associated with severe cardiomyopathy, and a third cardioversion was performed after amiodarone initiation. AF was induced by rapid atrial pacing and extensive areas in the LA exhibited dispersion. The PVs were isolated, but the patient remained in AF. Given extensive posterior wall dispersion, posterior wall isolation was attempted but was limited by esophageal temperature rises. Ablation on the LA roof, at a dense cluster of dispersed electrograms, resulted in termination of AF into sinus rhythm (Figure 2D). He has remained in NSR at all follow-up visits to 1 year.

Discussion

Although multiple mapping technologies have been implemented to explore the extra-PV substrate in patients undergoing de novo ablation,[8,9] much less is known about the feasibility of using electrogram-based approaches for reablation patients. In the past, the Topera technology and, more recently, the Ablacon technology have been suggested to improve the outcome of reablation patients.[10,11] Here, we present 2 reablation paroxysmal AF cases in which a real-time mapping technology was implemented. Although the acute and medium-term outcomes in these 2 patients seemed satisfactory (noninducibility and no relapse), it should be emphasized that a larger clinical investigation is required.

Here, we have presented 2 de novo ablation patients and 2 reablation patients, all of whom were managed with intraprocedural mapping using the AI software. From the description

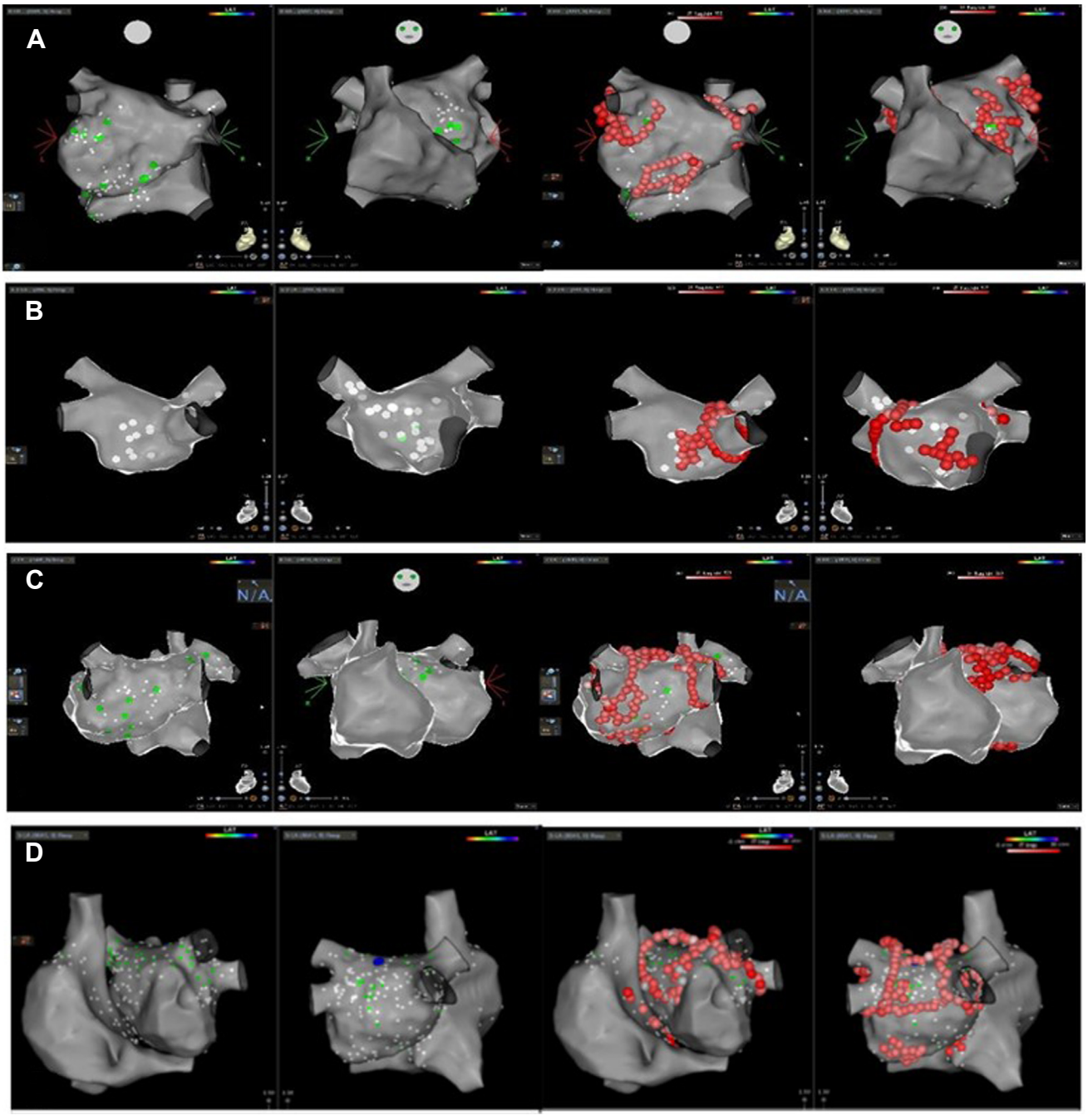

Figure 2 Electroanatomical maps from patients 1 (biatrial, **A**), 2 (left atrium, **B**), 3 (biatrial, **C**), and 4 (biatrial, **D**). Maps on the left demonstrate dispersion sites, where green points are highly dispersed and white points moderately dispersed. Right-sided maps show the dispersion and pulmonary vein isolation lesion sets. Blue dot in panels B and D shows point where termination of atrial fibrillation occurred.

of these cases, their operative workflow, and outcomes, it would seem that patients undergoing a de novo ablation in persistent AF and those undergoing a redo ablation have been managed similarly. It is paramount, however, to underline key differences between de novo ablated and reablated patients. Firstly, both patients admitted for reablation were suffering from paroxysmal AF episodes. By contrast, de novo ablation patients were in persistent AF. Arguably, ablation protocols and lesion sets need to be distinct when a patient is only in paroxysmal AF, albeit the PVs might still be isolated. One future direction for investigation will be to determine whether ablation sets need to be adapted to the AF presentation (persistent or paroxysmal) and to the procedural history (de novo vs redo), or to both. Here, artificial

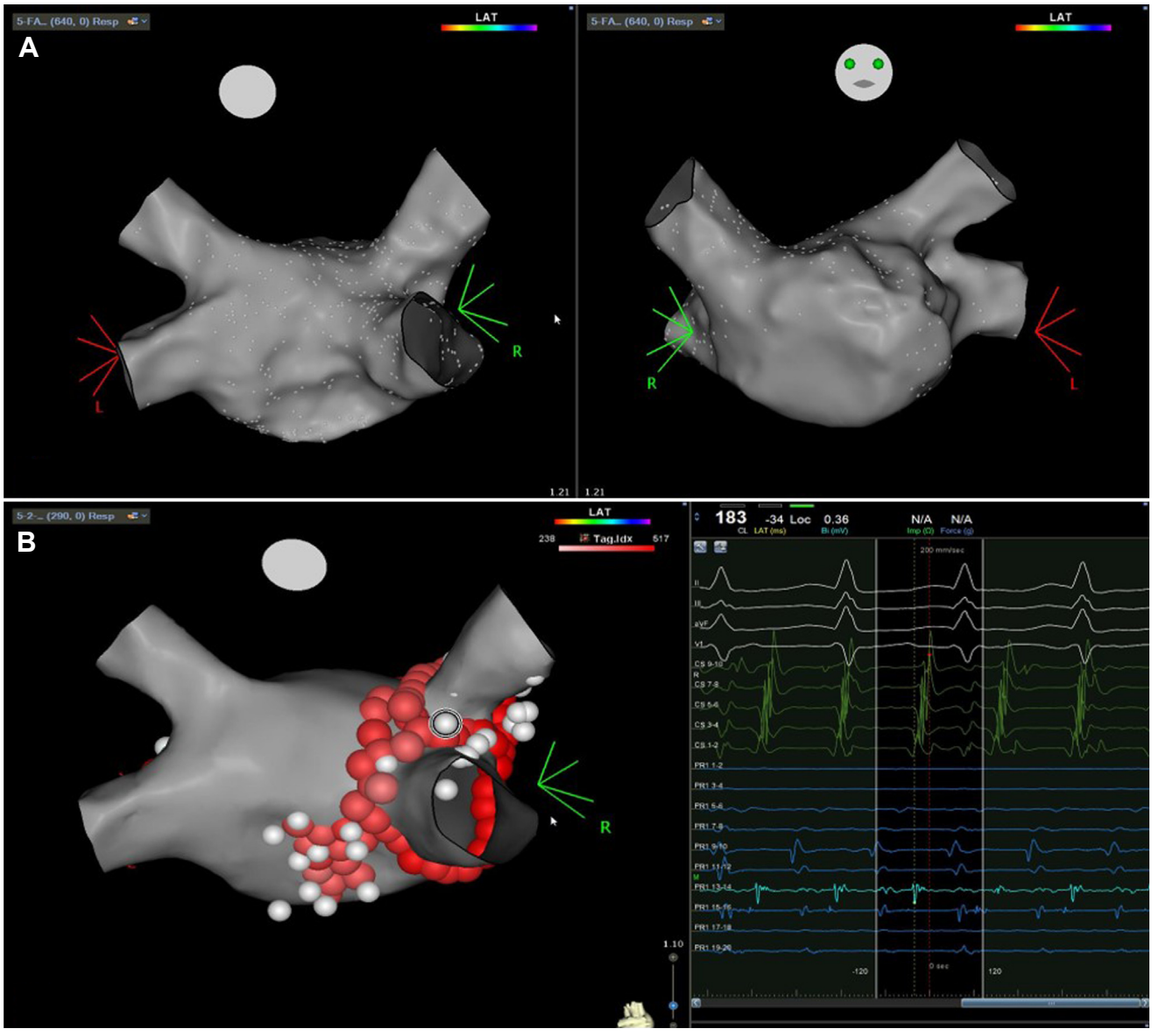

Figure 3 Electroanatomical maps from patient 2. **A:** Image shows 43 left atrial points (*white dots*) mapped with the PENTARAY (Biosense Webster, Irvine, CA) and the VX1 (Volta Medical, Marseille, France) system. **B:** PENTARAY signals in the right carina, with dispersion demonstrated across the PENTARAY splines. PENTARAY 13/14 (white dot with highlighted edge/blue-green electrogram) is at the site of termination during ablation.

intelligence approaches might be ideally suited to modulate the procedural management based on these parameters.[12]

In conclusion, VX1 is an AI-based software solution designed to obtain real-time adjudications of multipolar electrograms. This software-based mapping approach may be integrated in the workflow of patients admitted for both de novo persistent and redo paroxysmal AF. In the cases presented, noninducibility and favorable outcomes were achieved after a relatively extensive ablation set. The utility of dispersion ablation beyond the PVs in our paroxysmal patients is anecdotal, but a clinical trial is underway to specifically answer whether tailored dispersion-based ablation in repeat paroxysmal patients with isolated PVs improves clinical outcomes.[13]

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