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Left Atrial Slow-Conduction Regions as Predictors of Pulmonary Vein Isolation Outcome in Persistent Atrial Fibrillation

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Abstract

PURPOSE

Recurrence after durable pulmonary vein isolation (PVI) is common in persistent atrial fibrillation (AF), and left atrial (LA) slow-conduction regions are potential contributors. We assessed their impact on PVI outcomes in persistent AF.

METHODS

Fifty consecutive patients with persistent AF undergoing first-time PVI were prospectively enrolled. After sinus rhythm restoration by electrical cardioversion, high-density LA mapping was performed during coronary sinus pacing before PVI. Slow-conduction regions corresponded to discrete zones bordered by lines of block and characterized by fragmented electrograms, decremental conduction, and a conduction velocity about 70% slower than that of the remaining LA. Median follow-up was 18 months.

RESULTS

Slow-conduction regions were found in 27 of 50 patients (54%). AF or atrial tachycardia recurred in 9 of 27 patients with slow-conduction regions (33%) versus 1 of 23 without (4%; log-rank $P = 0.010$). At redo (mean, 7 months), PVI was durable in 8 of 10 patients, with unchanged slow-conduction regions. Slow-conduction region presence was the strongest independent predictor of recurrence (odds ratio 6.36; 95% confidence interval 1.26–63.06; $P = 0.023$), regardless of voltage. Restricting to durable PVI and no new substrate, recurrence was 8 of 26 with slow-conduction regions versus 0 of 22 without (log-rank $P = 0.005$).

CONCLUSIONS

In persistent AF, the absence of LA slow-conduction regions predicts near-complete response to PVI alone, whereas their presence predicts recurrence despite durable PVI, supporting patient selection and tailored ablation.

CAPSULE SUMMARY

In 50 patients with persistent atrial fibrillation treated with pulmonary vein isolation alone, left atrial slow-conduction regions identified by pre-ablation high-density mapping were present in 54% and predicted recurrence despite durable isolation, whereas their absence was associated with a near-complete response.

1 Introduction

The mechanisms responsible for recurrent atrial fibrillation (AF) in patients with durable pulmonary vein isolation (PVI) remain unclear. Although AF originating outside the pulmonary veins (PVs) was initially attributed to multiple wavelets, it is now increasingly linked to focal activity [1–3]. Several structural and electrogram-based concepts have been studied as potential AF sources, but none has proved fully reliable in clinical practice. Recently, regions of slow conduction in the left atrium (LA) have attracted considerable attention because of their presumptive role in both paroxysmal and persistent AF [4–7]. However, whether these regions impact the outcome of PVI in patients with persistent AF has not yet been specifically investigated.

We conducted a prospective, single-center study of consecutive patients with persistent AF undergoing a first ablation procedure limited to PVI. The study evaluated whether outcomes following durable PVI differed according to the presence or absence of LA regions of slow conduction.

2 Methods

2.1 Study Population

Between January 2024 and February 2025, 50 consecutive patients with persistent AF undergoing PVI were prospectively enrolled at a single French electrophysiology center (Les Franciscaines Private Hospital, Nîmes, France). Persistent AF was defined according to current international guidelines [8]. Exclusion criteria were prior catheter ablation or cardiac surgery involving the LA, uncontrolled ischemic or valvular heart disease, hypertrophic cardiomyopathy, congenital heart disease, contraindication to anticoagulation, stroke or transient ischemic attack within the previous 6 months, untreated hyperthyroidism, and inability to complete follow-up. All patients underwent a standardized ablation strategy consisting exclusively of PVI, irrespective of clinical presentation, atrial dimensions, or electroanatomic substrate characteristics. Sex (female/male) was recorded as documented in the medical records. The study was approved by the local ethical review board, and all participants provided written informed consent. The study complied with the Declaration of Helsinki.

2.2 Electrophysiologic Study and Characterization of Slow-Conduction Regions

All procedures were performed under general anesthesia with uninterrupted direct oral anticoagulation using the CARTO 3™ system (Johnson & Johnson MedTech, Irvine, CA). Intravenous unfractionated heparin maintained an activated clotting time > 350 s. Double ultrasound-guided femoral venous access was obtained, and a decapolar catheter was positioned in the coronary sinus (CS). Transseptal puncture was performed under transesophageal echocardiographic guidance. LA activation and voltage maps were created with a high-density mapping catheter (PENTARAY™ Johnson & Johnson MedTech) during mid-CS pacing at 600 ms before PVI, ensuring reproducible measurements and revealing slow-conduction regions. Patients in AF at procedure start underwent electrical cardioversion (200-J QRS-synchronized shock, up to three times) to restore sinus rhythm (SR).

Mapping settings were uniform: bipolar voltage > 0.5 mV normal, 0.2–0.5 mV low voltage, < 0.2 mV scar; mapping density 1 mm; stability filters 4 mm; tissue proximity index on; wavefront annotation; and fast anatomical mapping interpolation. Conduction velocity was measured throughout the LA. Slow-conduction regions appeared as localized zones of delayed activation with multiple connected corridors, bordered by lines of block, showing fragmented electrograms and decremental conduction. Conduction velocity was calculated manually as v = D/t (mean of 3 measurements) and confirmed with the Local Velocity Vector algorithm (CARTO 3 V8). Decremental conduction was assessed by mid-CS pacing at 400 ms with extrastimuli down to refractoriness.

2.3 Pulmonary Vein Isolation

PVI was performed with a 7.5-F, 3.5-mm irrigated-tip, temperature-controlled radiofrequency catheter (SmartTouch SF, Johnson & Johnson MedTech) connected to an nGEN radiofrequency generator (Johnson & Johnson MedTech). Point-by-point applications (50 W; 48°C; irrigation 15 mL/min) were delivered at the antral level using an ablation index–guided approach (target 450 on the anterior wall and 350 on the posterior wall) with a target interlesion distance of 3 to 4 mm [9–12]. Entrance and exit block were confirmed for all PVs at the end of the procedure. Patients were discharged within 24 hours.

2.4 Follow-up

After the index procedure, all patients were discharged on anticoagulation for ≥ 2 months according to the CHA_2DS_2-VASc score, and no antiarrhythmic drugs were prescribed. Patients attended outpatient visits at 3, 6, 12, and 18 months after the index procedure (12-lead electrocardiogram during each visit). Arrhythmia recurrence was defined as any documented episode of AF or atrial tachycardia (AT) lasting ≥ 30 seconds after a 90-day blanking period. During the blanking period, recurrences were managed with antiarrhythmic drugs, electrical cardioversion, or both. All patients with recurrence after the blanking period were offered a redo ablation procedure.

2.5 Redo Procedures

Redo procedures were performed with the same setup and materials as the index procedure. Voltage and activation mapping were conducted to assess the durability of PVI, characterize the LA electroanatomic substrate, and identify changes relative to the index procedure. The ablation strategy was left to the operator's discretion and consisted of PV re-isolation, if applicable, plus substrate modification if deemed necessary.

2.6 Statistical Analysis

Continuous variables are expressed as mean ± standard deviation or median (Q1–Q3), as appropriate; normality was assessed with the 1-sample Kolmogorov-Smirnov test. Continuous variables were compared with the Welch *t*-test or the Mann-Whitney *U* test, and categorical variables, presented as n (%), with the Fisher exact test. Arrhythmia-free survival was estimated with the Kaplan-Meier method and compared with the log-rank test. Logistic regression with Firth's penalized-likelihood correction was used to identify factors independently associated with arrhythmia recurrence; because of the limited number

of events, each multivariable model included at most 2 predictors. A 2-sided *P* value < 0.05 was considered statistically significant. Group comparisons were performed with IBM SPSS Statistics version 26.0 (IBM Corp); Firth logistic regression was performed in Python (version 3.11; SciPy 1.17), and log-rank tests were computed with an online calculator (Statistics Kingdom) [13]. Generative AI (Claude Opus 5.5, Anthropic) was used to implement the Firth penalized logistic regression in Python; all outputs were reviewed and verified by the authors, who take full responsibility for the content.

3 Results

3.1 Baseline Characteristics

A total of 50 patients with persistent AF were included (median age 70 years [Q1–Q3: 62–78 years]; 13 women [26%]). Compared with patients without slow-conduction regions, patients with these regions were generally older and had a higher prevalence of previous stroke or transient ischemic attack, as well as higher CHA_2DS_2-VASc scores. Their median body mass index was lower, at 26 versus 28 kg/m^2. None of these differences reached statistical significance, and baseline characteristics were overall comparable between groups (Table 1). The proportion of women was identical in both groups (26%).

Table 1
Baseline characteristics

Age, y	SC Regions (n = 27)	No SC Regions (n = 23)	All Patients (n = 50)	*P* Value
	72 [64–79]	**68 [61–74]**	**70 [62–78]**	**0.123**
Female	7 (26)	6 (26)	13 (26)	1.000
Hypertension	18 (67)	15 (65)	33 (66)	1.000
Diabetes mellitus	3 (11)	2 (9)	5 (10)	1.000
History of stroke/TIA	6 (22)	1 (4)	7 (14)	0.107
Obstructive sleep apnea	10 (37)	9 (39)	19 (38)	1.000
BMI, kg/m^2	26 [24–30]	28 [27–31]	27 [25–31]	0.161
CHA_2DS_2-VASc score	3 [2–4]	2 [1–4]	3 [2–4]	0.077
Time since AF onset, mo[a]	9 [1–18]	4 [2–33]	5 [2–28]	0.860
AF at procedure onset	16 (59)	15 (65)	31 (62)	0.773
Mapping points	1,490 [1,072–1,898]	1,783 [1,265–2,114]	1,683 [1,113–1,930]	—
LA volume, mL	160 [130–183]	130 [93–165]	150 [116–180]	0.080
LVEF, %	60 [53–60]	60 [50–60]	60 [50–60]	0.584

Values are median [Q1–Q3] or n (%).

[a]Available in 24 patients (10 with SC regions, 14 without).

AF = atrial fibrillation; BMI = body mass index; LA = left atrial; LVEF = left ventricular ejection fraction; SC = slow-conduction; TIA = transient ischemic attack.

3.2 Electroanatomic Findings

High-density LA mapping was performed in all patients, with a comparable median number of mapping points in patients with and without slow-conduction regions (1,490 [Q1–Q3: 1,072–1,898] vs 1,783 [Q1–Q3: 1,265–2,114]). Patients showed moderate LA dilatation (median LA volume 150 mL [Q1–Q3: 116–180 mL]), with a trend toward larger LA volumes in patients with slow-conduction regions (160 mL [Q1–Q3: 130–183 mL] vs 130 mL [Q1–Q3: 93–165 mL]; $P = 0.080$).

Activation mapping identified 34 slow-conduction regions in 27 of 50 patients (54%): 20 patients (74%) had a single region and 7 (26%) had 2 regions. Slow-conduction regions were located within low-voltage areas (LVAs) in 14 of 27 patients (52%) and in normal-voltage tissue in the remaining 13 (48%). Twelve of

50 patients showed isolated LVAs without corresponding slow-conduction regions on the activation map.

Slow-conduction regions covered discrete regions accounting for approximately 1% to 2% of the total LA surface area (range: 0.6%–2.9%). All slow-conduction regions displayed fragmented electrograms, were bordered by lines of conduction block, and exhibited single or multiple entry and exit sites. Conduction velocity within slow-conduction regions was uniformly slow (0.36 ± 0.09 m/s), approximately one-quarter to one-third of that measured in the remaining LA (1.17 ± 0.34 m/s) (Fig. 1). The location of the slow-conduction region—within LVAs or in normal-voltage areas—did not affect conduction velocity or the degree of wavefront slowing (Table 2).

Table 2
Electroanatomic features of SC regions

Mapping points	**SC Region Within LVA (n = 14)**	**SC Region in Normal-Voltage Area (n = 13)**	**All Patients With SC Regions (n = 27)**	***P* Value**
	1,757 [1,390–2,262]	**1,072 [716–1,820]**	**1,490 [1,072–1,898]**	**—**
LA volume, mL	150 ± 38	162 ± 45	155 ± 41	0.466
SC region area, % of LA surface	1.5 [1.3–1.8]	1.2 [1.1–1.4]	1.4 [1.2–1.6]	0.075
Patients with 1 SC region	10 (71)	10 (77)	20 (74)	1.000
Patients with 2 SC regions	4 (29)	3 (23)	7 (26)	1.000
SC region length, mm	25 ± 3	23 ± 3	24 ± 3	0.060
SC region width, mm	31 [30–35]	28 [27–30]	30 [28–34]	0.029
Conduction velocity within SC region, m/s	0.35 ± 0.09	0.38 ± 0.08	0.36 ± 0.09	0.387
Conduction velocity outside SC region, m/s	1.18 ± 0.28	1.16 ± 0.41	1.17 ± 0.34	0.898
Conduction velocity ratio[a]	0.28 [0.26–0.37]	0.34 [0.30–0.37]	0.30 [0.27–0.37]	0.214

Values are median [Q1–Q3], mean ± SD, or n (%).

[a]Conduction velocity within the SC region divided by conduction velocity outside the SC region.

LA = left atrial; LVA = low-voltage area; SC = slow-conduction; SD = standard deviation.

Consistent with previous reports, pacing maneuvers revealed decremental conduction within slow-conduction regions [4, 5]. These regions were predominantly located on the LA anterior wall (70%), followed by the posterior wall (12%), the mitral isthmus and LA appendage region (6%), the interatrial septum (6%), the LA roof (3%), and perivenous areas (3%). The electroanatomic features of slow-conduction regions are summarized in Table 2 and Fig. 2.

3.3 Index Ablation Procedures

Median procedural and ablation times were 71 and 16 minutes, respectively. The only complication was pericarditis in 1 patient (2%). Procedural data are detailed in Online Resource 1 (Table S1).

3.4 Follow-up and Redo Procedures

After a median follow-up of 18 months, arrhythmia recurred in 9 of 27 patients with slow-conduction regions (33%) and in 1 of 23 patients without (4%; log-rank $P = 0.010$). All 10 patients with recurrence underwent a redo procedure, performed a mean of 19 ± 4 days after documented recurrence and a mean of 7 months after the index procedure. At redo, 8 of the 10 patients had durable PVI and 2 had PV reconnection. LA volume remained stable relative to the index procedure, with a mean change of − 0.3 mL (range, − 27 to + 15 mL).

In all 9 patients with slow-conduction regions and arrhythmia recurrence, these regions remained unchanged at redo, with identical dimensions, architecture, and electrophysiologic characteristics. No new slow-conduction region or LVA emerged in these patients. The remaining patient with recurrence, who had neither slow-conduction region nor LVAs at the index procedure, showed PV reconnection and a new slow-conduction region on the LA anterior wall at redo.

Atrial flutter recurred in 3 of 10 patients; activation mapping showed LA re-entrant tachycardias propagating through the slow-conduction regions, which corresponded to the critical isthmus. Recurrence and redo-procedure characteristics are summarized in Online Resource 1 (Table S2).

3.5 Predictors of Recurrence

Among all clinical and electrophysiologic variables, the presence of slow-conduction regions was the strongest predictor of recurrence after PVI on both univariable (odds ratio 7.70; 95% confidence interval [CI] 1.55–76.62; $P = 0.010$) and multivariable analysis (odds ratio 6.36; 95% CI 1.26–63.06; $P = 0.023$). This association was independent of slow-conduction location (within LVAs or in normal-voltage areas) and was associated in both cases with recurrence on multivariable analysis ($P = 0.016$ and $P = 0.037$, respectively). By contrast, LA volume and LVAs alone were not significant predictors. Among slow-conduction region properties, the conduction velocity ratio was also associated with recurrence on univariable analysis ($P = 0.035$), with a borderline association on multivariable analysis ($P = 0.050$) (Table 3). Female sex was not associated with recurrence ($P = 0.684$); the small number of women precluded sex-stratified analyses. In sensitivity analyses, the association between slow-conduction

regions and recurrence remained significant after adjustment for each clinical covariate individually and for a propensity score for slow-conduction region presence (Online Resource 1, Table S3).

Table 3
Univariable and multivariable predictors of arrhythmia recurrence

Age, per year	**Univariable OR (95% CI)**	***P* Value**	**Multivariable OR (95% CI)**	***P* Value**
	0.99 (0.93–1.07)	**0.834**	**—**	**—**
Female	1.36 (0.29–5.58)	0.684	—	—
Hypertension	1.17 (0.30–5.45)	0.825	—	—
Diabetes mellitus	1.28 (0.12–8.10)	0.810	—	—
Stroke/TIA	3.79 (0.72–19.26)	0.113	—	—
Obstructive sleep apnea	1.14 (0.28–4.40)	0.851	—	—
BMI, per kg/m^2	1.02 (0.89–1.15)	0.733	—	—
CHA_2DS_2-VASc score, per point	1.02 (0.65–1.62)	0.915	—	—
LVEF, per 5%	1.31 (0.87–2.56)	0.226	—	—
LA volume, per mL	1.01 (1.00–1.03)	0.098	1.01 (0.99–1.03)	0.234[a]
Time since AF onset, per month[b]	1.01 (0.97–1.05)	0.466	—	—
AF at procedure onset	2.53 (0.60–14.69)	0.214	—	—
Presence of LVAs	2.88 (0.74–13.32)	0.130	—	—
Isolated LVAs (without SC region)	0.85 (0.14–3.77)	0.843	3.03 (0.37–35.43)	0.294[c]
Presence of SC regions	7.70 (1.55–76.62)	0.010	6.36 (1.26–63.06)	0.023[a]
SC regions within LVAs	3.32 (0.82–13.75)	0.092	8.68 (1.46–93.57)	0.016[d]
SC regions in normal-voltage areas	2.30 (0.54–9.39)	0.253	7.11 (1.12–78.28)	0.037[d]
Number of SC regions	2.09 (0.83–5.60)	0.118	—	—
SC region length, per mm[e]	0.87 (0.65–1.12)	0.276	—	—
SC region width, per mm[e]	0.86 (0.65–1.06)	0.165	—	—
Conduction velocity through SC region, per 0.1 m/s[e]	2.25 (0.89–6.79)	0.089	2.11 (0.83–6.29)	0.119[f]
Conduction velocity ratio, per 0.1[e]	2.64 (1.06–11.52)	0.035	2.43 (1.00–10.29)	0.050[g]

Age, per year	Univariable OR (95% CI)	*P* Value	Multivariable OR (95% CI)	*P* Value
	0.99 (0.93–1.07)	**0.834**	**—**	**—**

Values are odds ratios (95% CIs) from Firth penalized logistic regression; 95% CIs and *P* values were derived from the penalized profile likelihood. Variables with univariable $P < 0.10$ were entered in multivariable models. In addition, SC region location (model b) and isolated LVAs (model e) were modeled irrespective of their univariable *P* value, to test whether the association with recurrence depended on SC region location and whether LVAs without SC regions carried independent prognostic value. With 10 events, each model included at most 2 predictors, and collinear variables (SC region presence, location, and number) were not combined.

[a]Model a: presence of SC regions + LA volume (LA volume reported from this model).

[b]Analyzed in patients with known time since AF onset (n = 24; 3 recurrences).

[c]Model e: isolated LVAs + presence of SC regions.

[d]Model b: SC region location—SC regions within LVAs + SC regions in normal-voltage areas, both compared with patients without SC regions (the 2 locations are mutually exclusive).

[e]Analyzed in patients with SC regions (n = 27; 9 recurrences), as these measures do not exist in patients without SC regions.

[f]Model c: conduction velocity through SC regions + LA volume, in patients with SC regions (n = 27; 9 recurrences).

[g]Model d: conduction velocity ratio + LA volume, in patients with SC regions (n = 27; 9 recurrences).

AF = atrial fibrillation; BMI = body mass index; CI = confidence interval; LA = left atrial; LVA = low-voltage area; LVEF = left ventricular ejection fraction; OR = odds ratio; SC = slow-conduction; TIA = transient ischemic attack.

To isolate the role of slow-conduction regions, the Kaplan-Meier analysis was restricted to recurrences occurring with durable PVI and without new LVAs or slow-conduction regions at redo. In this analysis, recurrence occurred in 8 of 26 patients with slow-conduction regions and in 0 of 22 patients without, corresponding to an 18-month arrhythmia-free survival of 69% vs 100% (log-rank $P = 0.005$) (Fig. 3). Recurrence rates according to LVA status at the index procedure are shown in Online Resource 1 (Fig. S1).

4 Discussion

In patients with persistent AF undergoing de novo PVI, LA slow-conduction regions identified a phenotype at high risk of AF/AT recurrence despite durable PVI, whereas their absence defined a phenotype of patients with near-complete response to PVI alone.

Consistent with prior studies [4, 5, 14], regions of slow conduction corresponded to discrete areas of the LA characterized by a central core of slowed conduction bordered by lines of block. These regions

displayed multiple entry and exit sites, fragmented electrograms, and decremental properties.

Of importance, slow-conduction regions were found both within LVAs and within zones of the LA displaying normal voltage values. Finally, some patients had isolated LVAs without conduction delay [5, 14]. In our study, neither LVAs as a whole nor isolated LVAs without slow-conduction regions predicted recurrence. These findings indicate that slow-conduction regions may be a more specific marker of PVI outcomes than LVAs.

Most patients with AF/AT recurrence showed no substantial change in LA substrate at the redo procedure, a mean of 7 months after the index ablation. Slow-conduction regions retained stable electroanatomic features over this period, suggesting that they are spatially and temporally stable structures rather than functional or transient phenomena, as previously reported [15].

In our population, 54% of patients exhibited 1 or more slow-conduction region, and this substrate was associated with higher arrhythmia recurrence at 18 months despite durable PVI. Our findings extend the predictive role of these regions from paroxysmal to persistent AF [5]. Notably, slow-conduction regions are more prevalent in persistent than in paroxysmal AF (54% vs 35%) [5], consistent with the more pronounced atrial remodeling in the former. Whether these regions also act as active sources of AF remains unknown.

Only 2 of the 10 patients with arrhythmia recurrence had reconnected PVs. The presence of slow-conduction regions was strongly associated with an increased risk of recurrence despite durable PVI, regardless of whether these regions were located within LVAs or in normal-voltage tissue. Patients without slow-conduction regions (46%) remained in SR at 18 months in 96% of cases after PVI alone (100% when only recurrences with durable PVI were counted), compared with 67% of those with slow-conduction regions. These findings suggest that PVI alone is highly effective for patients with persistent AF who do not have slow-conduction regions. However, additional substrate modification may be necessary when such regions are present. These conclusions require validation through randomized studies.

4.1 Study Limitations

Our study has several limitations. First, its observational, single-center design and limited sample size may have introduced bias, and the wide CIs of the odds ratios reflect the small number of events. Second, right atrial mapping was not performed; however, the high success rate of LA-only ablation in patients without slow-conduction regions suggests that it may add little value in this population. Finally, although LA maps were detailed (median of approximately 1,700 points per map), some scar areas may have been missed or inadequately characterized; advances in mapping technology may improve the detection of channels and other electrical patterns.

5 Conclusions

In patients with persistent AF undergoing catheter ablation, the absence of LA slow-conduction regions is associated with a high response to PVI alone, whereas their presence is associated with PVI nonresponse. These findings highlight the potential role of slow-conduction regions in predicting PVI outcome and refining patient stratification. Whether these regions merely mark procedural failure or may serve as therapeutic targets remains unclear and warrants further investigation.

Abbreviations

AF
atrial fibrillation
AT
atrial tachycardia
CI
confidence interval
CS
coronary sinus
LA
left atrium/left atrial
LVA
low-voltage area
PV
pulmonary vein
PVI
pulmonary vein isolation
SR
sinus rhythm

Declarations

Competing interests:

The authors have no relevant financial or non-financial interests to disclose.

Ethics approval:

This study was performed in line with the principles of the Declaration of Helsinki and was approved by the local ethics committee.

Consent to participate:

Written informed consent was obtained from all individual participants included in the study.

Consent to publish:

Not applicable. The manuscript contains no identifiable individual data or images.

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Author Contribution

A.M.M. and A.B. conceived the study. A.M.M. collected the data, performed the formal analysis, prepared Figures 1–3 and wrote the original draft of the manuscript. A.B. supervised the study. A.M.M., L.R.L. and A.B. reviewed and edited the manuscript. All authors read and approved the final manuscript.

Data Availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Figures

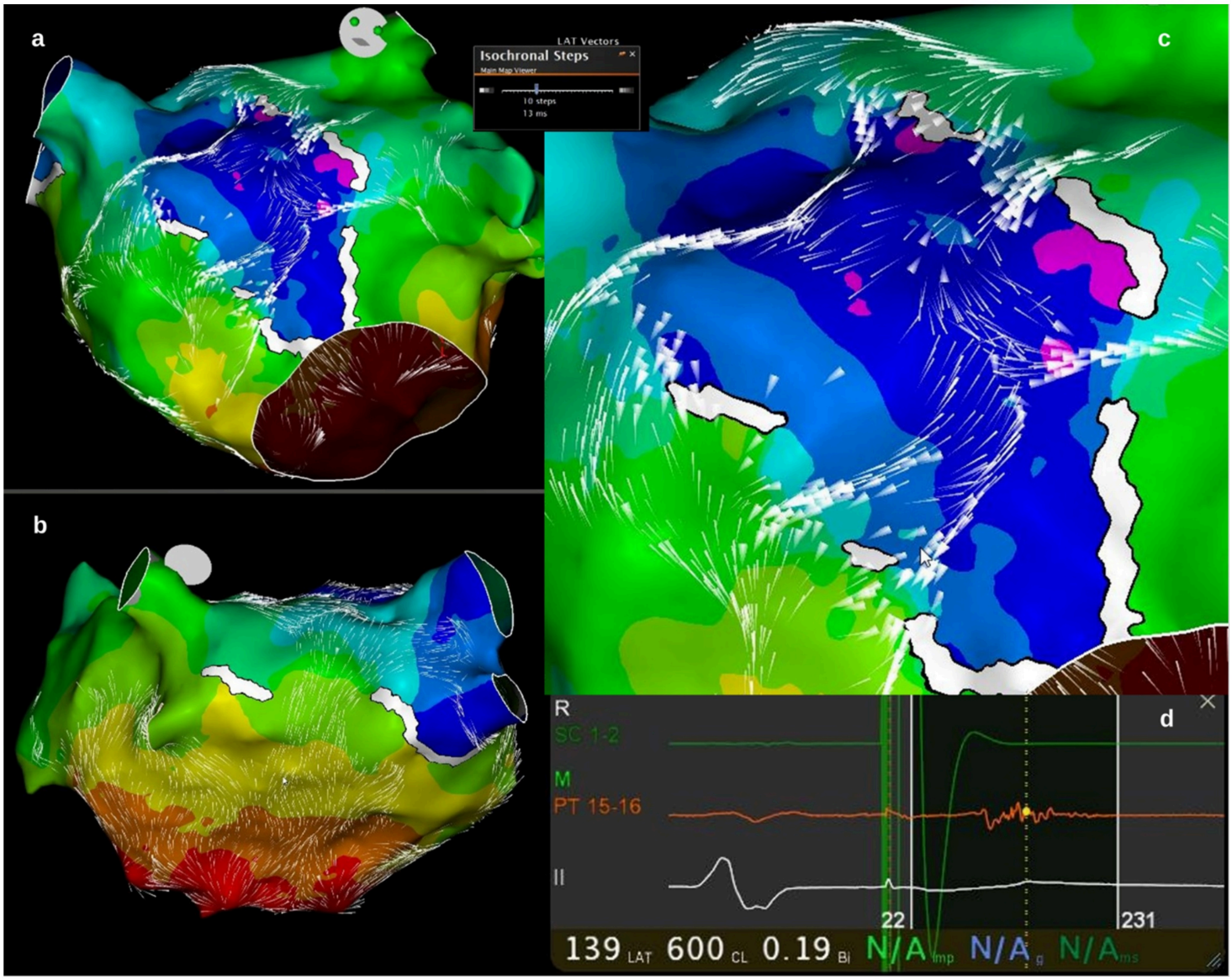


Figure 1

LA SC region at the index procedure. Isochronal activation maps with conduction velocity vectors (white arrows; 13-ms isochrones) acquired during mid-CS pacing at a cycle length of 600 ms, shown in anterior (a) and posterior (b) views. An SC region is visible on the anterior LA wall, where the wavefront decelerates through multiple arborized channels forming a complex network with multiple entry sites. (c) Magnified view of the SC region architecture. (d) Fragmented electrogram recorded within the SC region. CS = coronary sinus; LA = left atrial; SC = slow-conduction

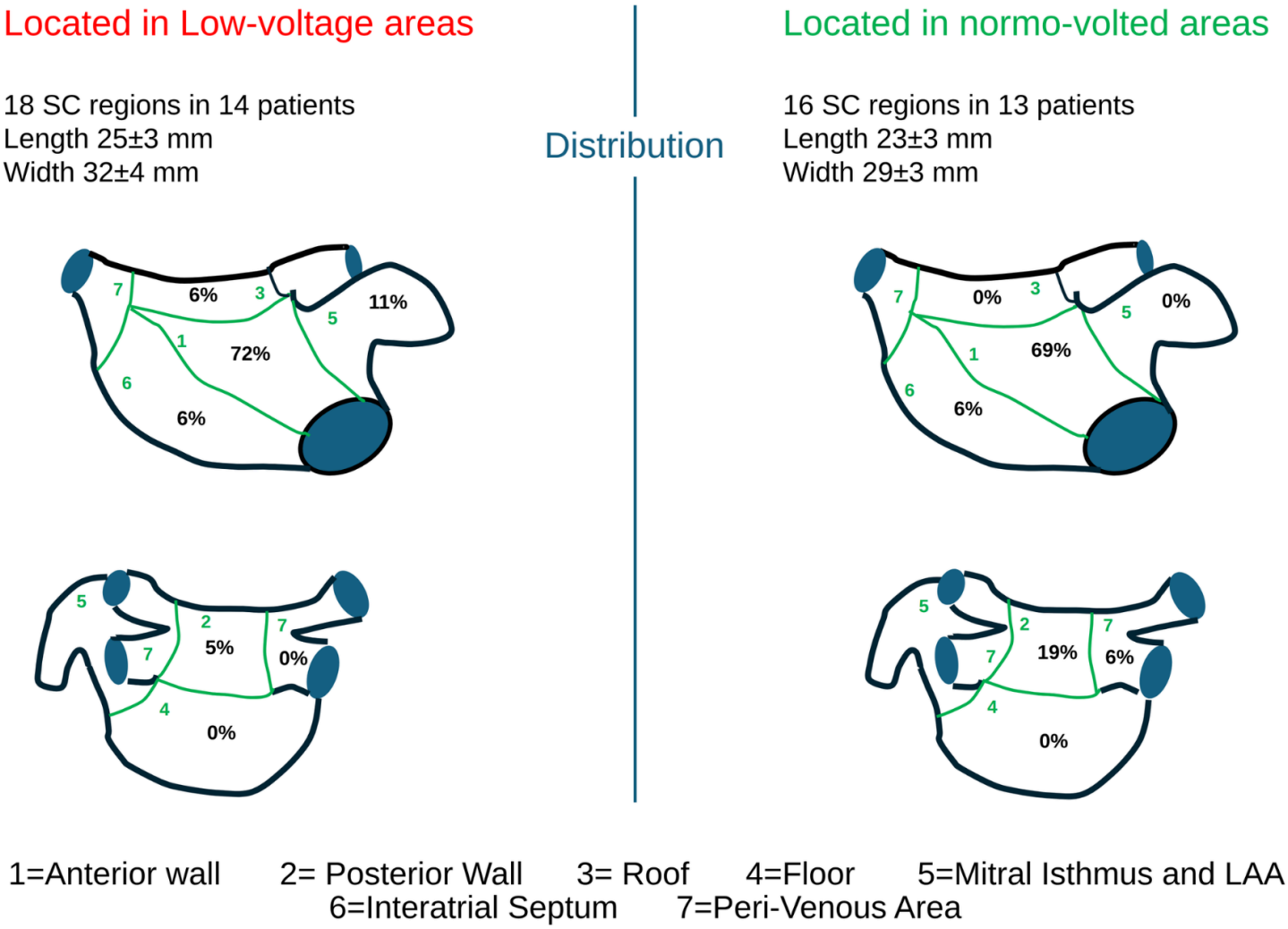


Figure 2

Distribution of SC regions in the LA. Schematic representation of the location of the 34 SC regions identified in 27 patients, shown separately for SC regions located within LVAs (18 in 14 patients) and in normal-voltage areas (16 in 13 patients). Numbers indicate LA regions; percentages indicate the proportion of SC regions in each zone. LA = left atrial; LAA = left atrial appendage; LVA = low-voltage area; SC = slow-conduction

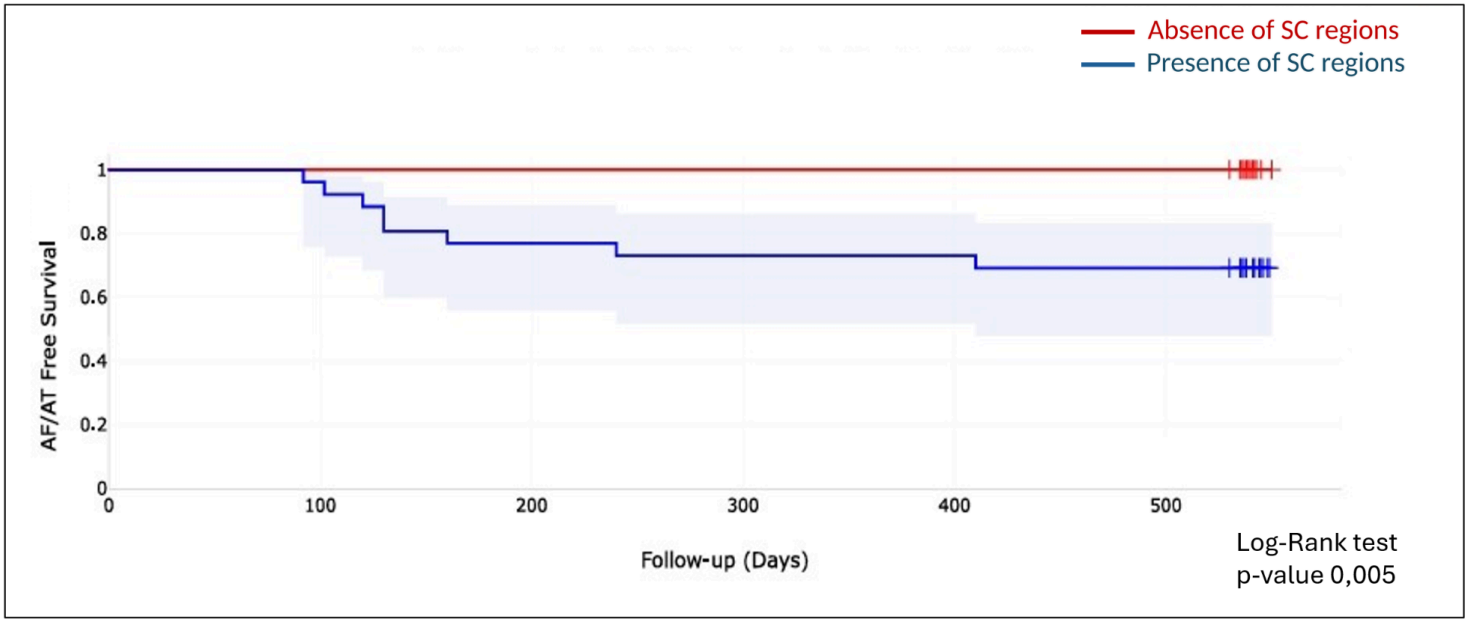


Figure 3

Arrhythmia-free survival after PVI according to SC region presence. Kaplan-Meier estimates of freedom from AF/AT after the index PVI in patients with vs without LA SC regions. Only recurrences with durable PVI and no new LVA or SC region at the redo procedure were counted as events (8 of 26 vs 0 of 22 patients; log-rank *P* = 0.005). Shaded area indicates the 95% CI. AF = atrial fibrillation; AT = atrial tachycardia; CI = confidence interval; LA = left atrial; LVA = low-voltage area; PVI = pulmonary vein isolation; SC = slow-conduction

Supplementary Files

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- SupplementaryMaterial.pdf