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Supplementary Methods: Electrophysiology procedure

The procedures were performed in the fasting state, under deep sedation or general anesthesia, with continuous invasive monitoring of arterial pressure, arterial oxygen saturation, and acid/base balance, according to our standard protocol. ICD therapies were inactivated.

A hexapolar catheter, with an additional electrode positioned in the inferior vena cava, to be used as the reference (negative) input of the unipolar electrogram, was placed in the RV apex. In patients without a cardiac resynchronization device, a decapolar catheter was placed in the coronary sinus. Access was gained to the endocardial LV cavity through either an anterograde transseptal approach, a retrograde aortic approach or a combined approach, depending on clinical factors. If the clinical VT or the pre-procedural imaging suggested an epicardial circuit, epicardial access was performed.

High-density mapping was performed with the Carto®3 Version 6 or 7 mapping system, using the Pentaray® mapping catheter (Biosense Webster, Inc., CA, USA) with interelectrode spacings of 2-6-2 mm. Bipolar electrograms were filtered from 30 to 250 Hz. Substrate maps were collected before any radiofrequency ablation either with SR (preferably with atrial pacing from the proximal electrode of the coronary sinus catheter), RV pacing or LV pacing (by inserting a catheter into a LV venous branch via the coronary sinus or using the epicardial lead in patients with a biventricular pacing system). To minimize the effect of frequency on the spatial distribution of slowly conduction regions, paced maps were performed at a constant cycle length of 600ms, if tolerated, with a pulse width of 2ms and current of ≥2 times the capture threshold. In patients with atrioventricular block or permanent atrial fibrillation, mapping was only performed during activation from two directions.

VT was induced by programmed stimulation and an activation map was performed of any sustained and hemodynamically stable circuit. Mapping of VT was considered complete when >90% of the tachycardia cycle length was mapped and the common channel was properly delineated. The VT isthmus location was confirmed by entrainment mapping and/or pace mapping and subsequently by response to ablation.

The ablation strategy was not modified because of the participation in this study. Radiofrequency ablation was delivered with a sensor enabled open-irrigated catheter with contact force monitoring [Thermocool Smarttouch® (Biosense Webster)] at 30–50 W.

When available, the critical isthmuses of the VTs were first ablated, followed by regions of LAVAs from all maps. Procedural endpoints were VT non-inducibility and LAVAs elimination.

Venous and arterial sheaths were withdrawn after the procedure. ICD therapies were reactivated at the end of the procedure. Patients were monitored in hospital for at least 2 days. Antiarrhythmic-drug therapy was continued according to operator discretion.

Supplementary Methods: Late potential map alghorithm.

The LPM algorithm includes a LAVA annotation algorithm, a localized bipolar voltage assessment and an algorithm for automatic regional vector analysis, allowing to determine the directionality of the electrical wavefront and to display a quantitative assessment of LCV¹².

The LAVA annotation algorithm analyses all components of the unipolar signal within the window of interest and proceeds with a sequential analysis to better discriminate the near-field signal. Based on unipolar dV/dT analysis, all the candidate components to be recognized as potential signals of interest are identified, and priority for annotation is given to the latest one if its voltage is higher than the noise level and its timing is consistent in ≥2 consecutive heartbeats. The operator programs the late boundary threshold, which sets the local activation time value from which any candidate component becomes a priority for annotation. The operator has the option to adjust the unipolar dV/dT parameters used for candidate EGM components selection, impacting algorithm accuracy, with three different set modes available: standard, specific, and sensitive.

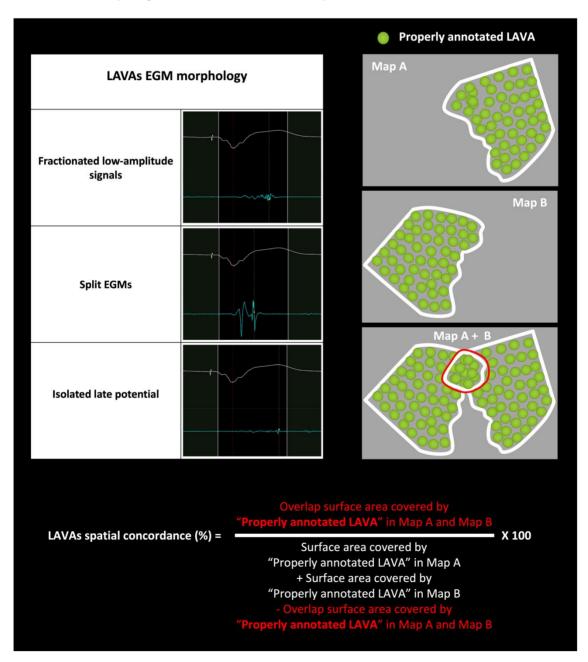
The localized bipolar voltage assessment aims to restrict the bipolar voltage measurement to near-field signals. To achieve it, the algorithm analyses the complete signal to determine whether it is a complex EGM (with multiple candidate components). In such cases, the window of interest for localized bipolar voltage assessment surrounds the near-field LAT annotation and its left curtain is dynamically adjusted to selectively exclude any high-voltage far-field components

LCV algorithm determines local vectors for the regions where high-resolution characterization of the electrical wavefront is available. Each vector calculation is based on the local activation time and positions of the surrounding EGM data points using a principal component analysis. The algorithm has a minimal number of point criteria within predefined radiuses and takes into consideration the temporal dispersion of the local activation time values to identify conduction discontinuity. The distance between EGMs used for vector analysis is measured from the projection of the electroanatomical points on the cardiac shell. Therefore, to limit spatial inaccuracy, the cardiac shell was carefully analyzed at the area of interest, and the zones where the cardiac surface had been pushed were shaved. Vectors are displayed as arrows, with an origin and a direction that indicates the directionality of the local electrical wavefront. Additionally, the algorithm determines LCV at each vector and that information is graphically represented by the thickness of the arrows. The operator has

the option to set the cutoff to be used in the slow conduction zones representation, ranging from 0.10 to 2mm/ms. The algorithm depicts with thick arrows the vectors displaying LCV below the chosen threshold and with thin arrows the remaining ones, in static or dynamic displays. Finally, the LCV algorithm includes a compression tool, which may be set from 0 to 3, based on principal component analysis modeling of the vector information, reducing local vector dispersion, which, in turn, may help the physician to concentrate attention on the central portion possible channels. By analyzing the vectors presenting a common direction in close proximity, the compression tool searches for the dominant one, which may theoretically better locate the conducting tissue corridor.

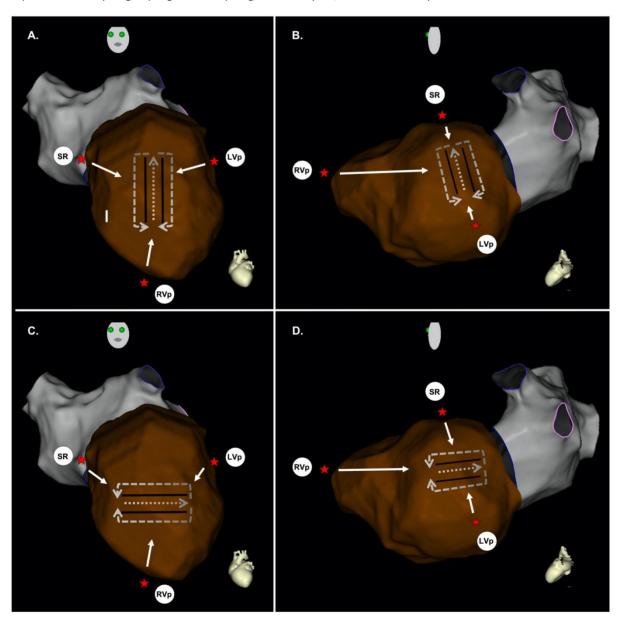
Supplementary Figure 1. LAVAs classification and area measurement (Left) Fractionated Low-

Amplitude EGMs: Signals with multiple (≥5) intrinsic deflections, amplitude <0.50 mV, duration ≥133 ms; Split EGMs: Signals with double or multiple components (irrespective of voltage amplitude) separated by an isoelectric interval or a period of very low-amplitude signals of ≥50-ms duration, occurring during the surface QRS or within 50 ms after its completion. Isolated Late EGMs: Local high-frequency signals (distinct from far field components), often of low amplitude, occurring ≥50 ms after the QRS⁹. (Right and bottom) LAVAs were tagged with green markers and its surface area measured. Overlap surface area and LAVA spatial concordance between two different pacing wavefronts were calculated as pictured.



Supplementary Figure 2. Activation front direction and VT isthmus orientation. (A) and (C) show a patient with an anterior/septal wall infarction with a longitudinal and circumferential VT isthmus, respectively. Notice that while in (A) SR and LVp wavefronts are perpendicular and RVp wavefront is parallel to the VT isthmus, respectively; in (C) SR and LVp wavefronts are parallel and RVp wavefront is perpendicular to the VT isthmus, respectively. (B) and (D) show a patient with an inferior/lateral wall infarction with a circumferential and longitudinal VT isthmus, respectively. Notice that while in (B) SR and LVp wavefronts are parallel and RVp wavefront is perpendicular to the VT isthmus, respectively; in (D) SR and LVp wavefronts are perpendicular and RVp wavefront is parallel to the VT isthmus, respectively.

LVp: Left Ventricular pacing; RVp: Right Ventricle pacing; SR: Sinus Rhythm; VT: Ventricular Tachycardia.



Supplementary Table 1. Baseline characteristics of the patients 112

Patient Characteristics*	N = 16
Age - yr.	65±12
Male sex - no. (%)	15 (94)
Ischemic heart disease	
Time since last myocardial infarction - yr.	12±10
Left main disease - no. (%)	2 (12)
Multivessel disease - no. (%)	9 (56)
Previous percutaneous revascularization - no. (%)	12 (75)
Previous surgical revascularization - no. (%)	3 (19)
Heart failure and comorbidities	
NYHA functional class	
I/II - no. (%	3 (19)/10 (62)
III - no. (%	3 (19)
Median NT-proBNP (IQR) – pg/ml	865 (422-3511)
LV ejection fraction - %	31±9
Previous stroke - no. (%)	3 (19)
Peripheral artery disease - no. (%)	1 (6)
Atrial fibrillation or flutter - no. (%)	6 (38)
Hypertension - no. (%)	15 (94)
Diabetes - no. (%)	6 (38)
Active smoking - no. (%)	5 (31)
Obesity - no. (%)	6 (38)
Renal failure** - no. (%)	6 (38)
Estimated GFR†	67 ± 27
Chronic pulmonary obstructive disease - no. (%)	1 (6)
Cardiovascular medication	
Antiarrhythmic drug - no. (%)	15 (94)
Amiodarone - no. (%)	15 (94)
Beta-blocker "	16 (100)
Renin-Angiotensin blocker [#] - no. (%)	9 (56)
Neprisylin inhibitor - no. (%)	5 (31)
Mineralocorticoid receptor blocker - no. (%)	12 (75)
Sodium-glucose co-transporter-2 inhibitors	2 (12)
Antiplatelet therapy - no. (%)	12 (75)
Anticoagulant therapy - no. (%)	6 (38)
Diuretic - no. (%)	11 (69)
Statins - no. (%)	13 (81)
Previous type of ICD device	
Indication and type of ICD before the index procedure - no. (%)	14 (88)
Primary prevention - no. (%)	8 (50)
Secondary prevention - no. (%)	6 (38)
Single-chamber - no. (%)	5 (31)
Dual- chamber - no. (%)	3 (19)
CRT defibrillator - no. (%)	6 (38)
ICD implanted after index procedure - no. (%)	2 (12)
Previous VT ablation - no. (%)	2 (12)
Indication for ablation	
Sustained VT requiring external cardioversion - no. (%)	7 (44)
Appropriate ICD shock - no. (%)	9 (56)
VT storm - no. (%)	4 (25)

¹¹⁴ 115 116 117 118 119 120 * Plus-minus values are means ±SD.

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^{**} Estimated glomerular filtration rate \leq 60 ml/m2.

[†] The estimated glomerular filtration rate (GFR) was calculated with the use of the Cockcroft–Gault formula.

[#] Angiotensin-converting-enzyme inhibitor or angiotensin receptor blocker

CRT: cardiac resynchronization therapy; ICD: Implantable cardioverter defibrillator LV: Left ventricle; NYHA: New York Heart Association; VT: Ventricular tachycardia

Supplementary Table 2. Patient and mapping characteristics

Patient	A 70	Gender	LVEF	Scar location	VT activation map	Substrate map				
ratient	Age	Gender	elidei LVEF Scali		(VT cycle length - ms)	Number of wavefronts	SR (number of points)	RVP (number of points)	LVP (number of points)	
1	48	М	15	Inferior/Lateral	No	3	Yes (1703)	Yes (1545)	Yes (2049)	
2	64	М	28	Inferior/Lateral	No	3	Yes (2332)	Yes (2623)	Yes (822)	
3	79	М	24	Anterior/Septal	No	3	Yes (2711)	Yes (2343)	Yes (2199)	
4	74	М	26	Inferior/Lateral	No	2	No	Yes (1477)	Yes (2052)	
5	44	М	25	Anterior/Septal	No	2	Yes (2962)	Yes (1654)	No	
6	71	М	23	Anterior/Septal	No	2	No	Yes (3070)	Yes (2250)	
7	83	М	27	Inferior/Lateral	No	2	No	Yes (3283)	Yes (3411)	
8	65	М	28	Anterior/Septal	No	2	Yes (2931)	Yes (3324)	No	
9	66	М	40	Inferior/Lateral	No	2	No	Yes (3341)	Yes (2835)	
10	43	М	43	Anterior/Septal	Yes (378)	1	Yes (4106)	No	No	
11	62	М	47	Inferior/Lateral	Yes* (597)	1	Yes** (2344)	No	No	
12	76	F	45	Inferior/Lateral	Yes (415)	1	Yes (1749)	No	No	
13	63	М	27	Inferior/Lateral	Yes (450)	1	No	Yes (3742)	No	
14	66	М	25	Anterior/Septal	Yes (510)	2	Yes (1327)	Yes (440)	No	
15	71	М	31	Anterior/Septal	Yes (440)	2	No	Yes (4135	Yes (4117)	
16	66	М	38	Inferior/Lateral	Yes (536)	2	No	Yes (3567)	Yes (6139)	

^{*} Endocardial-Epicardial ventricular tachycardia activation mapping. ** Endocardial map
F: Female; LVEF: Left ventricular ejection fraction; LVP: Left ventricular pacing; M: Male; ms – milliseconds; RVP: Right ventricular pacing; SR: Sinus Rhythm VT: Ventricular Tachycardia

Supplementary Table 3. Comparison of the electrophysiological properties in relation to the direction of wavefront activation

	:	SR vs. RVP (N=6)		RVP vs. LVP (N=9)			SR vs. LVP (N=3)		
	SR	RVP	P-value	RVP	LVP	P-value	SR	LVP	P-value
LV chamber surface area , mean ± SD, cm ²	169±37	177±36	0.53	187±60	196±56	0.17	145±11	171±40	0.42
EGM number, mean ± SD,	2283±636	1988±1001	0.28	2820±904	2875±1537	0.89	2248±509	1690±755	0.41
LAVAs number, mean ± SD, N	219±150	309±148	0.09	385±191	264±169	0.08	229±112	187±126	0.42
Scar area, mean ± SD, cm ²	102±25	96±30	0.31	90±30	111±61	0.25	87±15	82±30	0.59
LAVAs area, mean ± SD, cm ²	13.1±10.7	15.1±7.6	0.47	15.4±11.4	11.8±6.5	0.25	14.4±13.9	12.0±9.1	0.59
ISCC number, median, N	2.8	3.0	0.36	3.0	3.0	1.0	3.0	3.0	1

cm: centimetres; EGM: Electrogram; ISCC: Intra-scar conduction corridor; LAVA: Local Abnormal Ventricular Activity; LV: Left ventricle; LVP: Left ventricular pacing; RVP: Right ventricular pacing; SD: Standard deviation SR: Sinus Rhythm

Supplementary Table 4. Local conduction velocity at the critical portions of the VT isthmus, considering the following cutoffs: \leq 0.10; 0.11-0.15; 0.16-0.20; and >0.20 mm/msec.

	Entrance region conduction velocity (mm/msec)	Common channel conduction velocity (mm/msec)	Exit region conduction velocity (mm/msec)
Case 10	0.11-0.15	0.16-0.20	0.16-0.20
Case 11	0.11-0.15	0.16-0.20	0.16-0.20
Case 12	≤ 0.10	0.16-0.20	0.16-0.20
Case 13	≤ 0.10	0.11-0.15	0.11-0.15
Case 14	0.16-0.20	0.11-0.15	0.11-0.15
Case 15	0.11-0.15	0.11-0.15	≤ 0.10
Case 16	0.11-0.15	0.16-0.20	0.16-0.20

^{*} Highlighted cells represent the location of slower conduction. mm: millimeters; msec: milliseconds

Supplementary Movie 1. Local conduction velocity analysis and Intra-scar conduction corridors deliniation, superimposed in a local activation time substrate map during RV pacing in a patient with a history of inferior and infero-lateral wall infarction.