TIGULLIO 2024 II Congresso Nazionale di ARITMOLOGIA

16-17 Aprile Sestri Levante (GE)

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Guido Parodi, Lavagna

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Sede Congressuale
Hotel Vis a Vis ****
Sestri Levante

Diagnosi e terapia ablativa della tachicardia ventricolare destra

Massimo Tritto

UOC Elettrofisiologia ed Elettrostimolazione
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HUMANITAS UNIVERSITY · HUMANITAS CARDIO CENTER · HUMANITAS MATER DOMINI

Conflitti di interesse

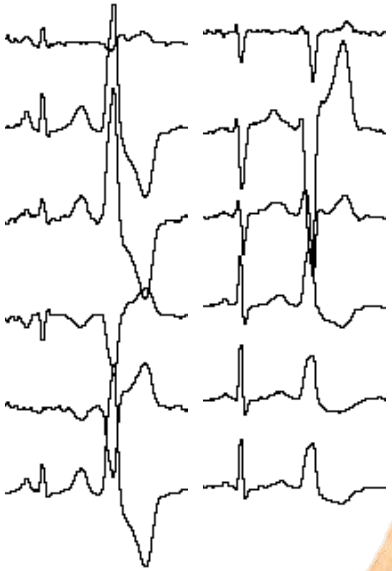
- nulla da dichiarare

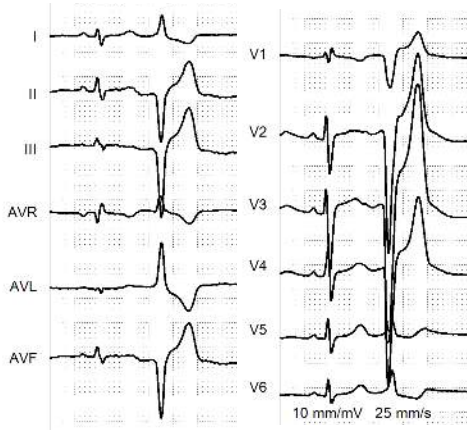
Aritmie ventricolari ad origine dal ventricolo destro

Eziologia	Tipo di aritmia	Sede di origine	Meccanismo
Idiopatiche	■ ExVe, TV nS ■ TVs monomorfa	■ Tratto di efflusso (~80%) ■ Anello tricuspidale ■ Banda moderatrice ■ M. papillari ■ Crux cordis	■ Automatismo anomalo ■ Post-depolarizzazioni tardive
C. Aritmogena dx	■ TVs monomorfa	■ Tratto di efflusso ■ Parete libera	■ Rientro scar-related
C. Infartuale	■ TVs monomorfa	■ Parete libera	■ Rientro scar-related

Aritmie ventricolari ad origine dal VD: aspetti ECG

- ☑ Tipo di blocco di branca in V1
- ☑ Asse del QRS
- ☑ Transizione precordiale
- ☑ Durata del QRS





Asse inferiore

Ant

BBS

BBD

DX

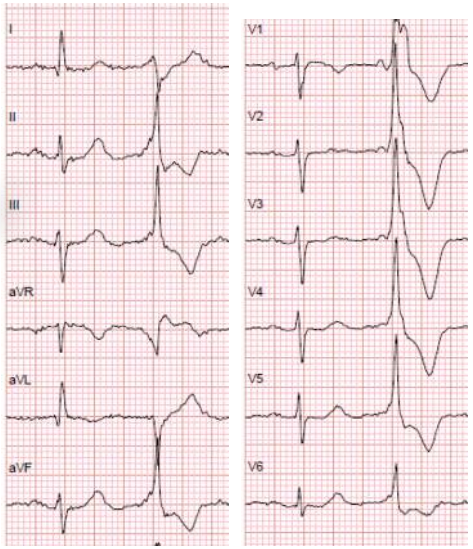
SN

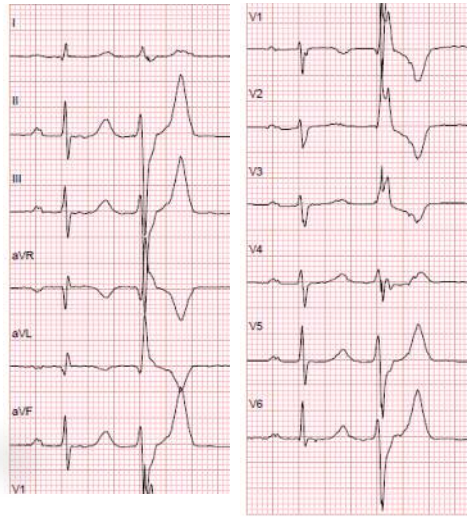
VDx

VSn

Post

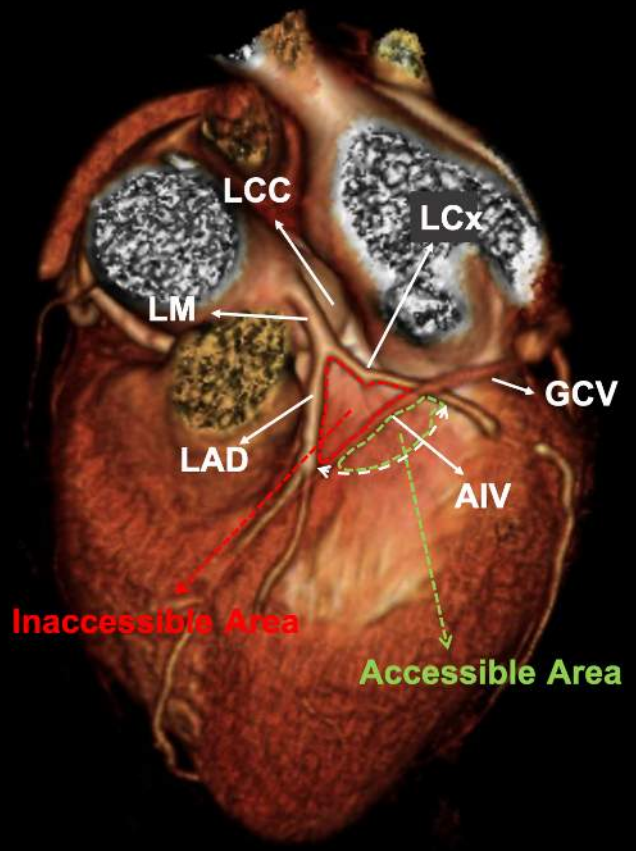
Asse superiore





Una accurata conoscenza dell'anatomia è importante non solo per la corretta identificazione della sede di origine dell'aritmia, ma anche per sfruttare "punti di vista" più vantaggiosi per il trattamento di substrati profondi o che originano dalla camera controlaterale

OT VT



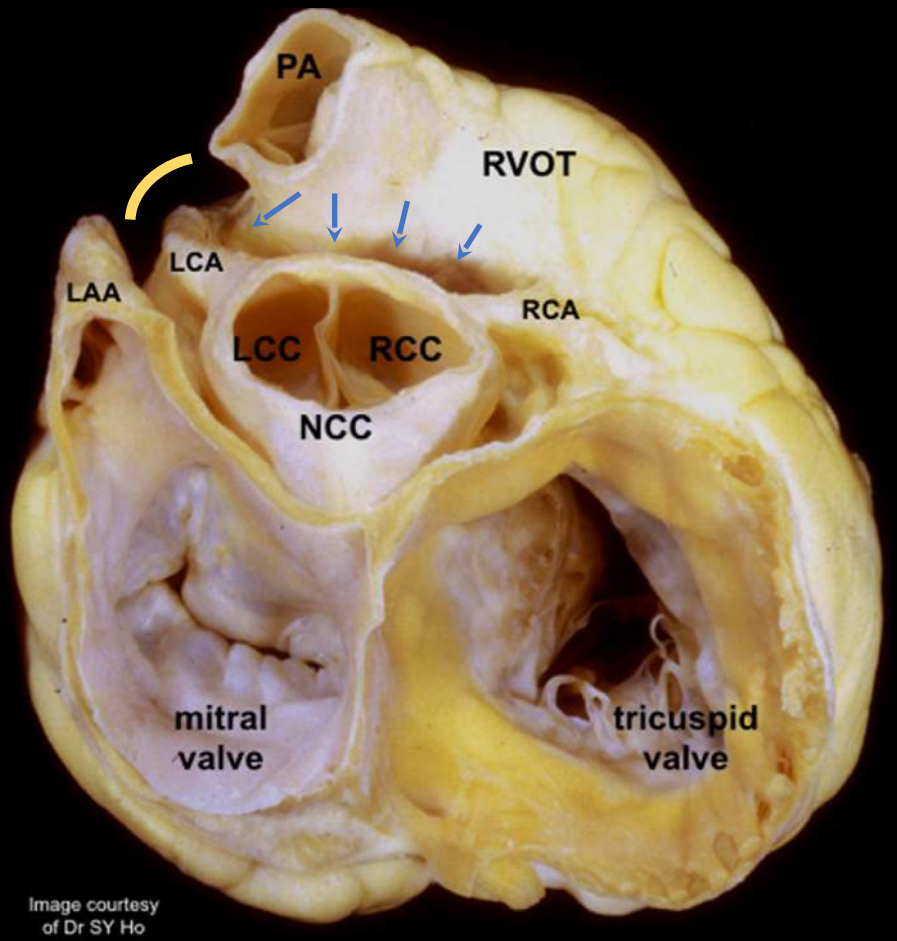


Image courtesy of Dr SY Ho

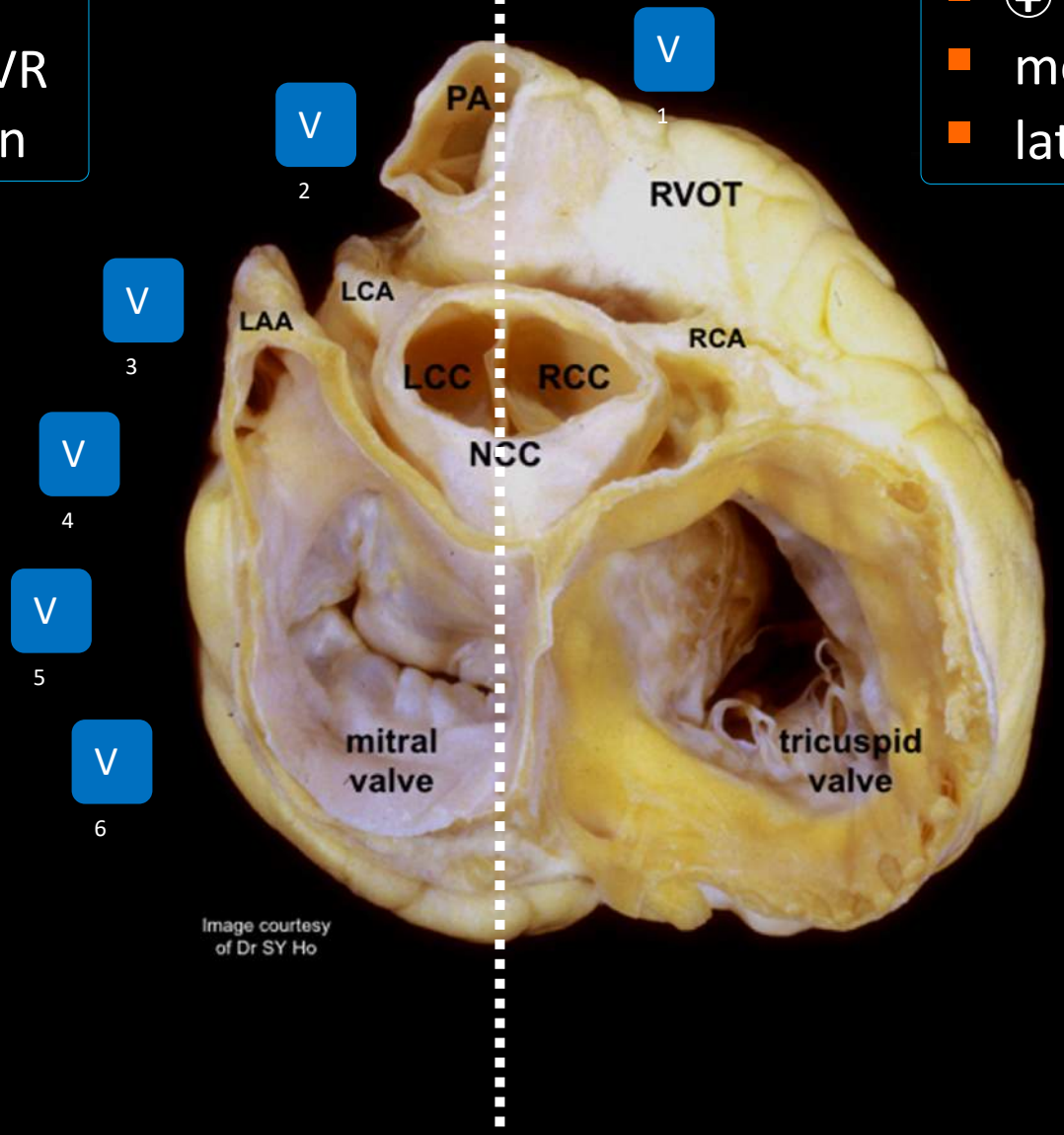
OT VT

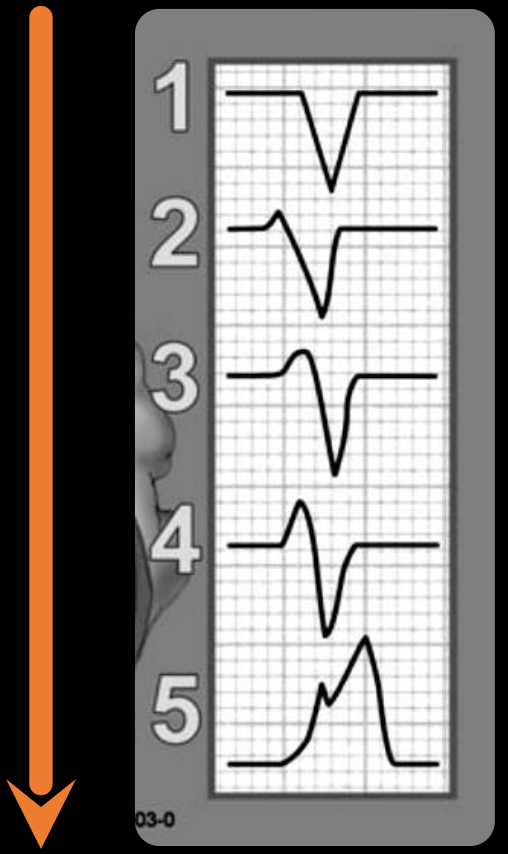
- ⊖ lead I
- more ⊖ aVL than aVR
- earlier R/S transition

Leftward

Rightward

- ⊕ lead I
- more ⊖ aVR than aVL
- later R/S transition





Come distinguere le aritmie ventricolari destre idiopatiche da quelle legate a malattia strutturale?

- Morfologia dei QRS all'ECG
- Modalità di presentazione clinica
- Caratteristiche elettrofisiologiche (modalità di induzione, risposta alle manovre di pacing)
- Metodiche di imaging (ECO, RMN, coronarografia, mappaggio EA)

Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: morfologia dell'ECG

An electrocardiographic scoring system for distinguishing right ventricular outflow tract arrhythmias in patients with arrhythmogenic right ventricular cardiomyopathy from idiopathic ventricular tachycardia

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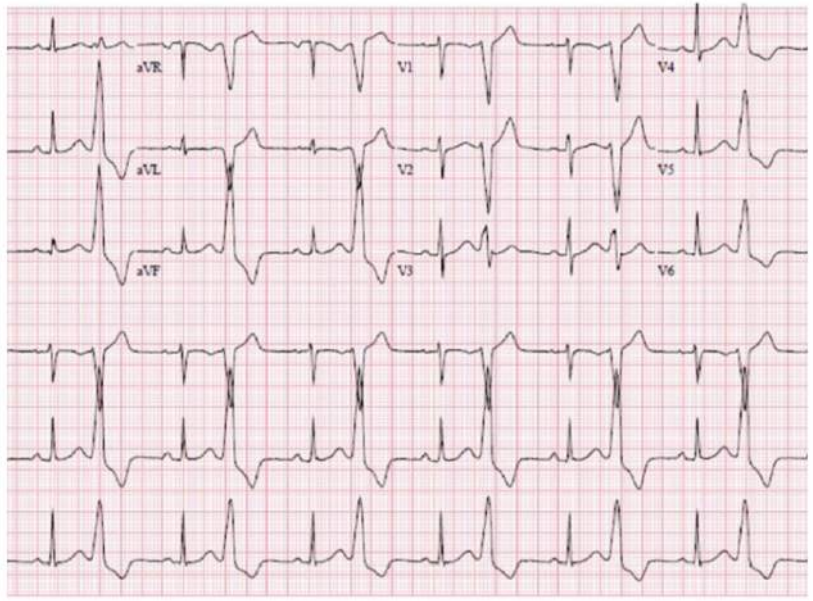
Heart Rhythm 2013;10:477–482

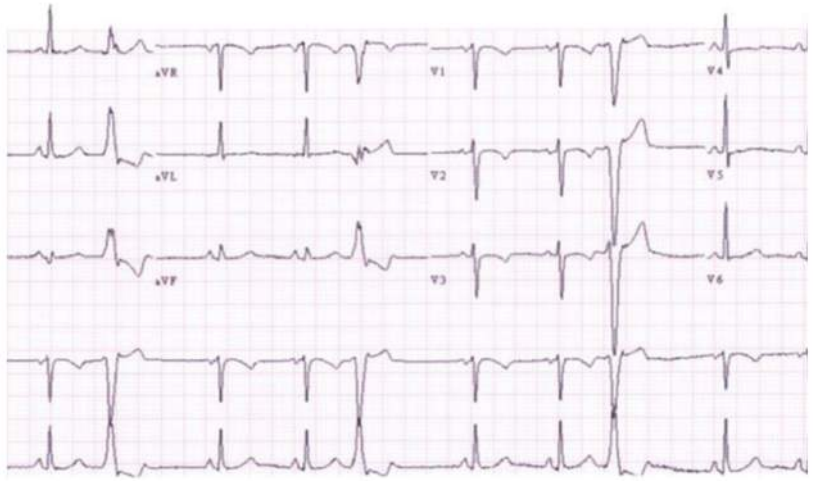
ECG characteristic	Points
Anterior T-wave inversions (V_1–V_3) in sinus rhythm	3
VT/PVC	
Lead I QRS duration $\geq$120 ms	2
QRS notching (multiple leads)	2
V_5 transition or later	1
Maximum total	8

Score ≥5 points: ARVC

Sensitivity: 84%

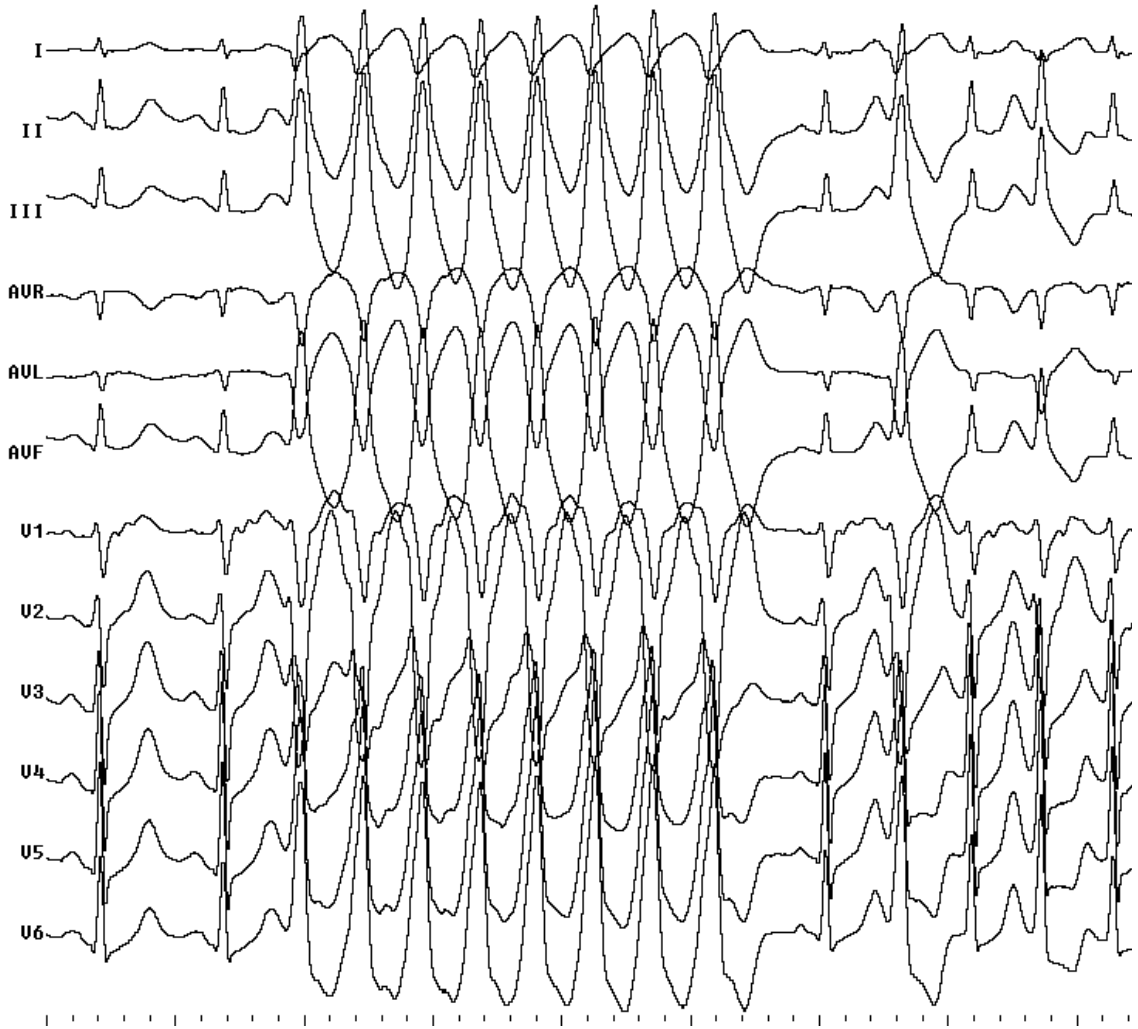
Specificity: 100%



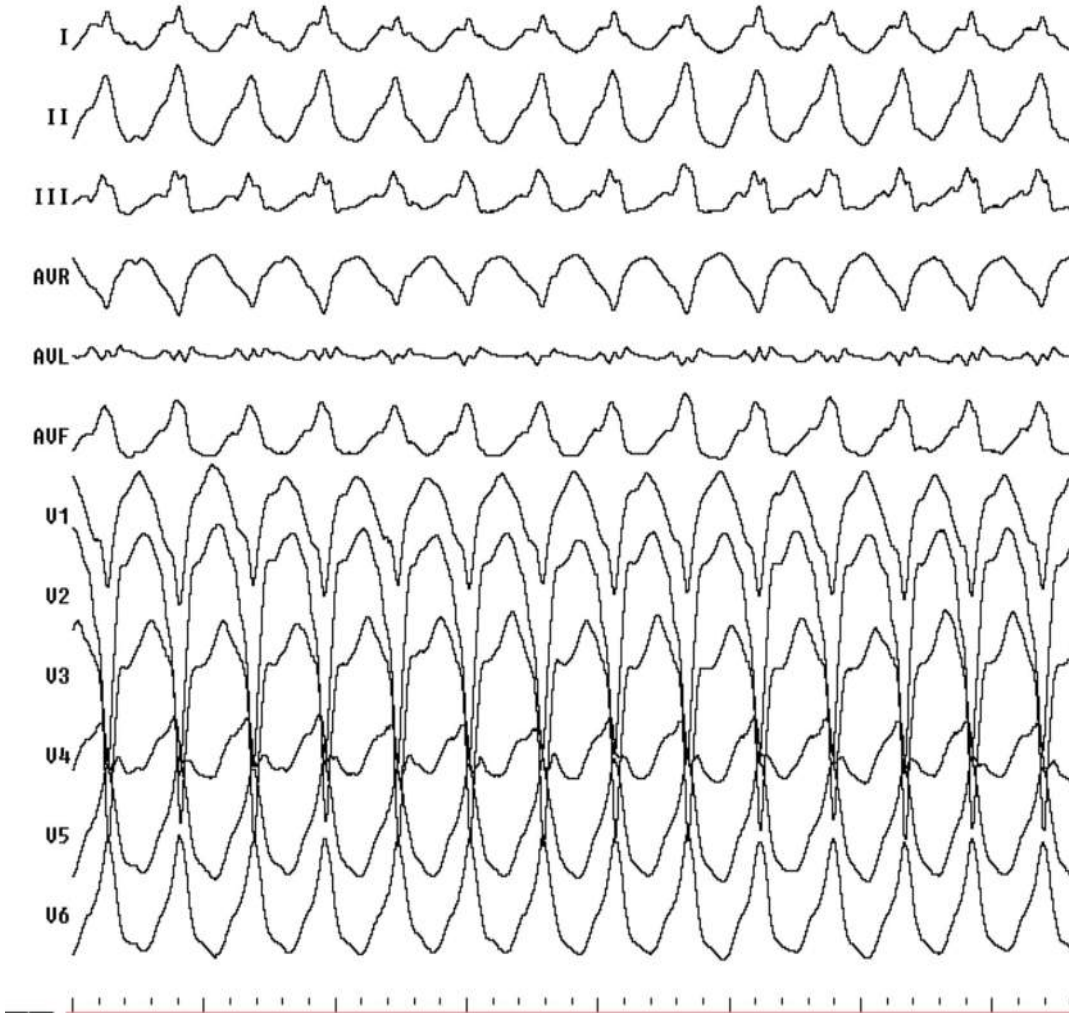


Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: presentazione clinica

ExVe – TV non sostenuta (più probabile idiopatica)



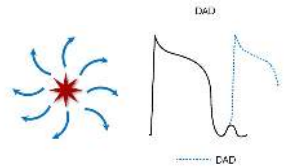
TV sostenuta (più probabile c. strutturale)

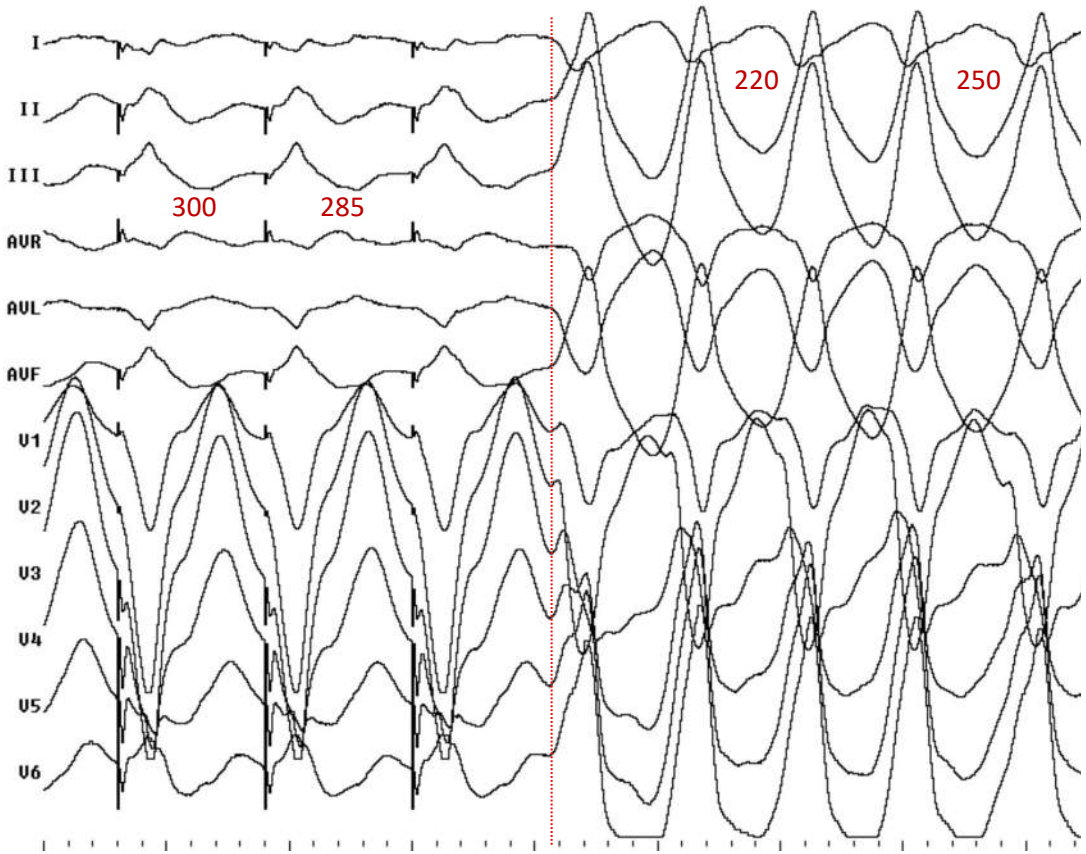


Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: modalità di induzione

Stimolazione ventricolare in burst

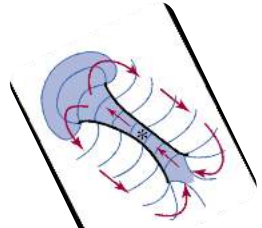
(più probabile idiopatica)

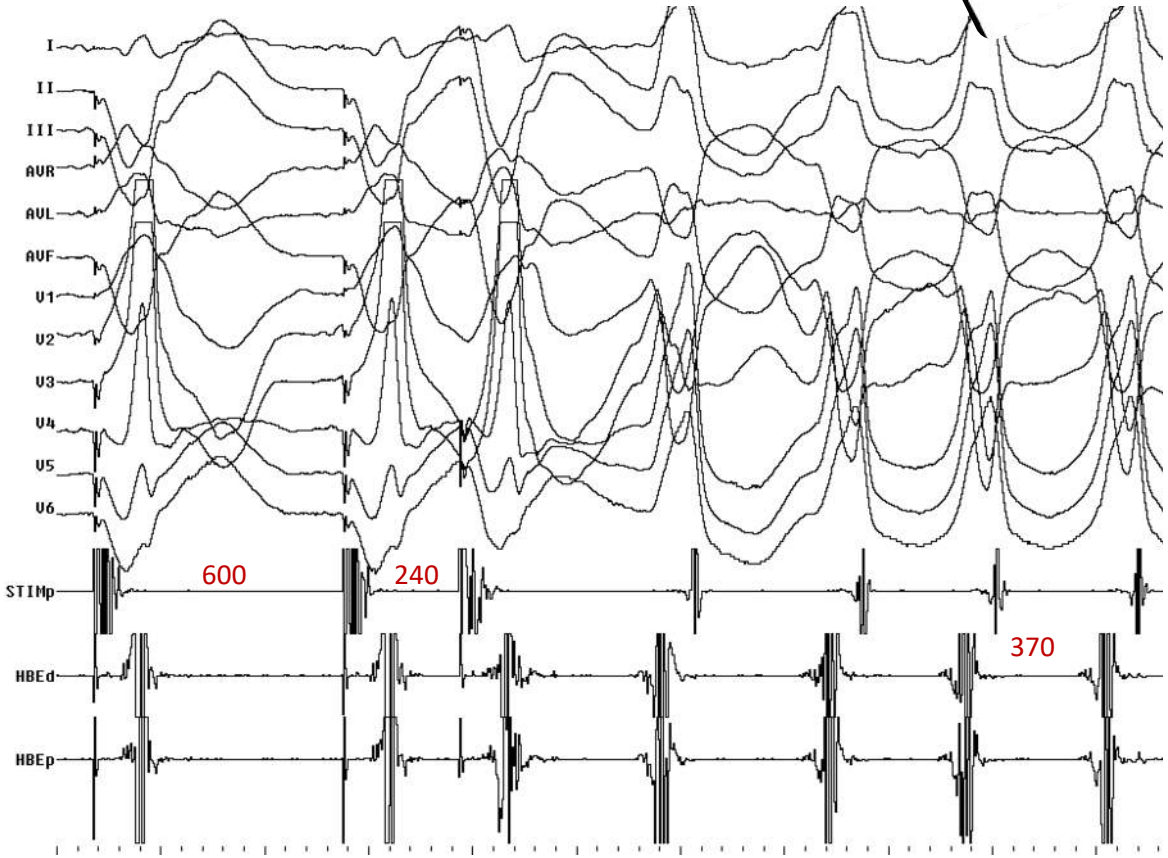




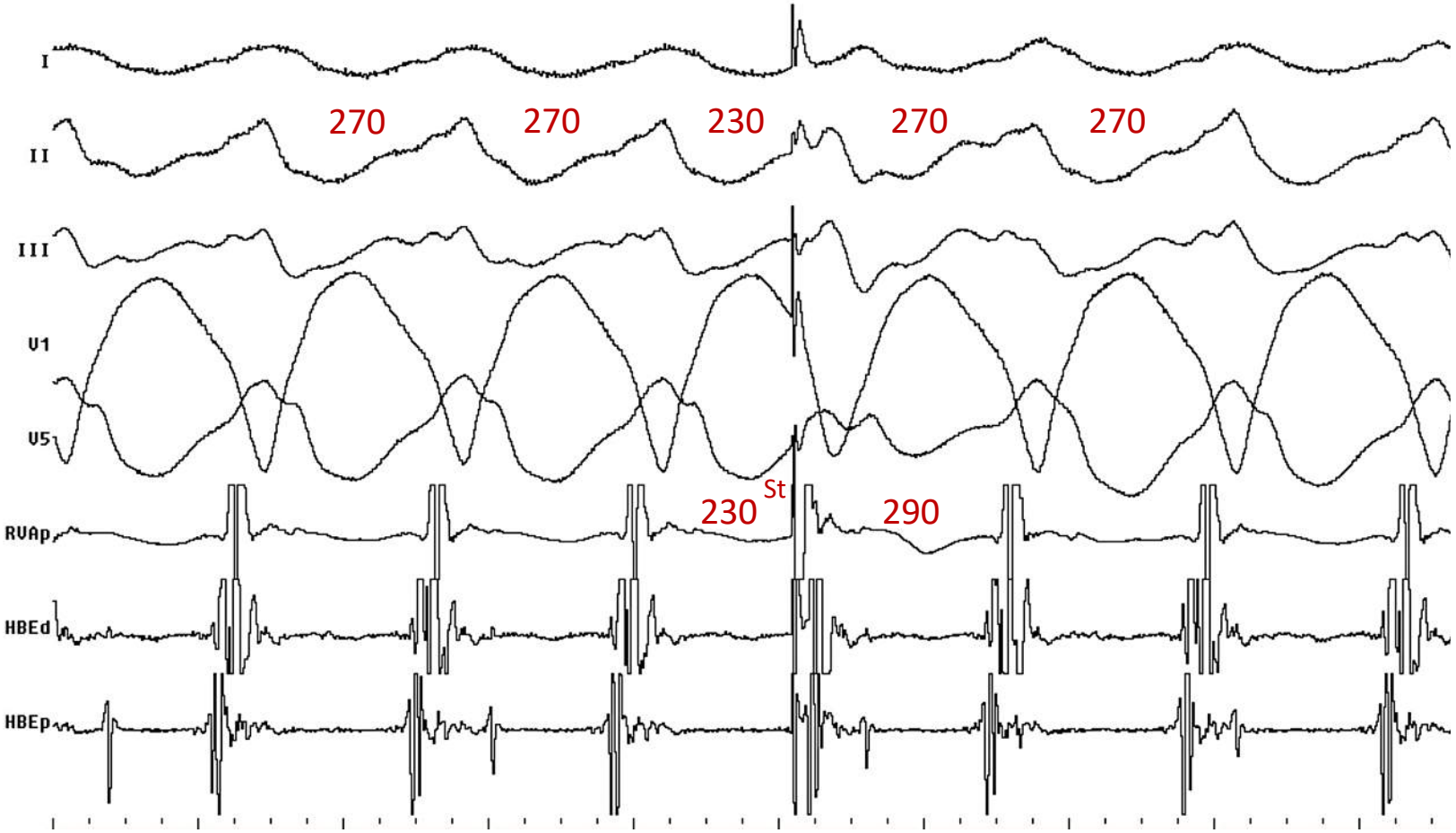
Stimolazione ventricolare prematura

(più probabile c. strutturale)

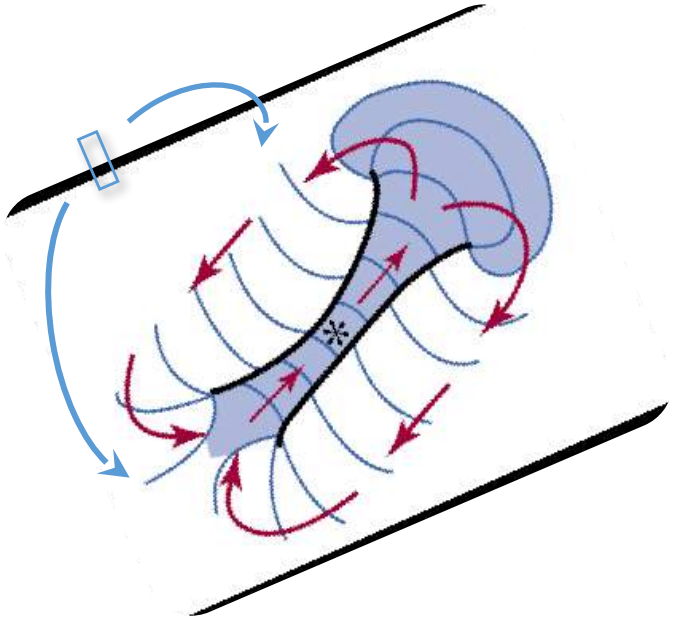




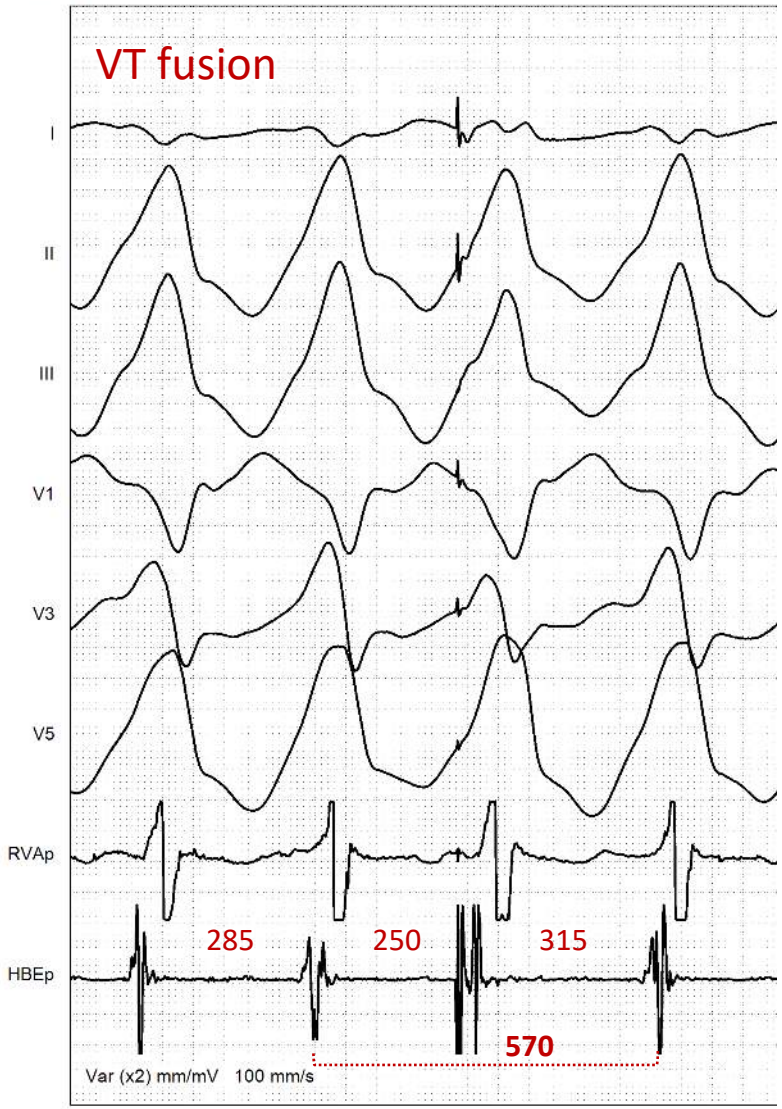
Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: risposta al pacing

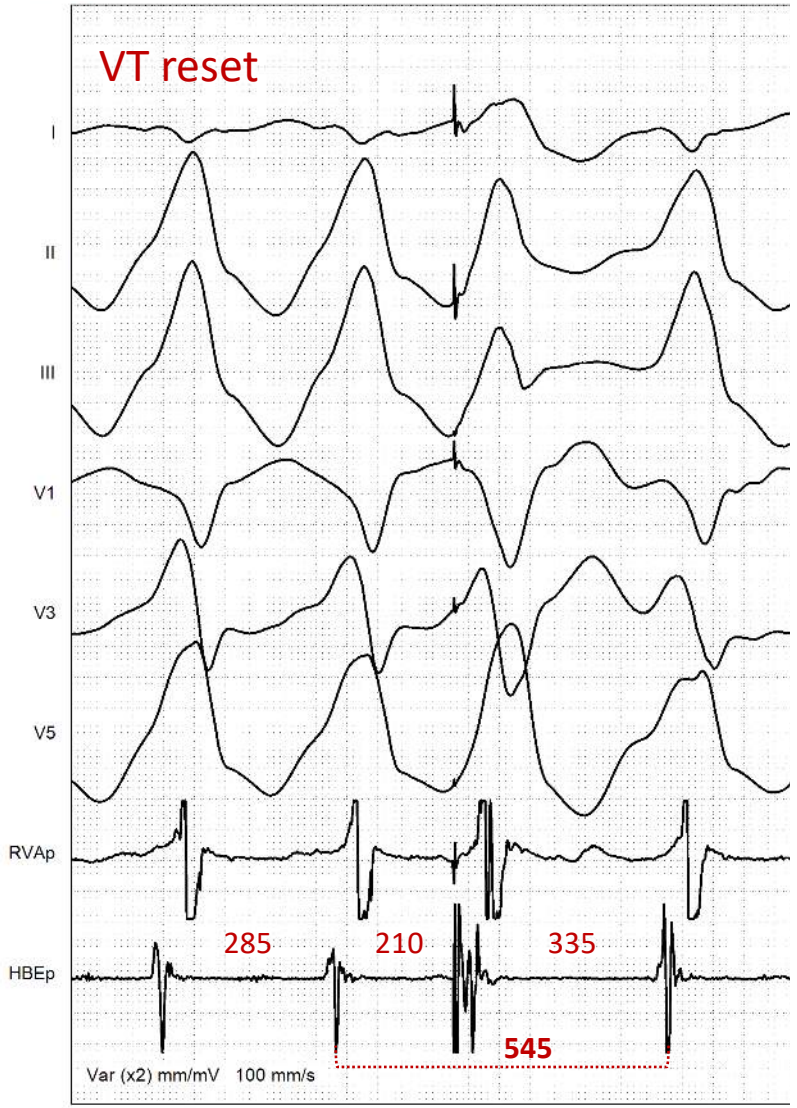


VT reset with overt fusion

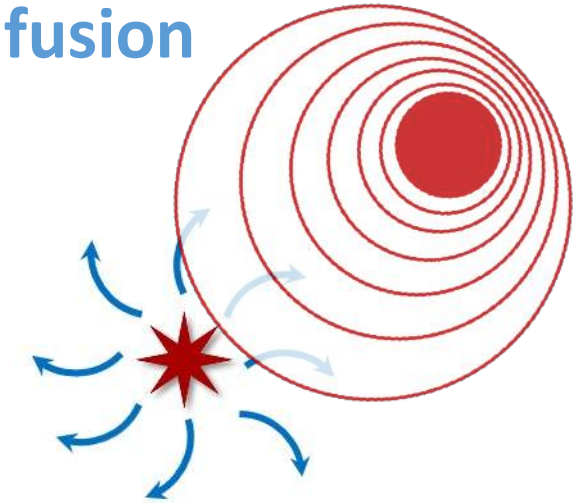


Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: risposta al pacing

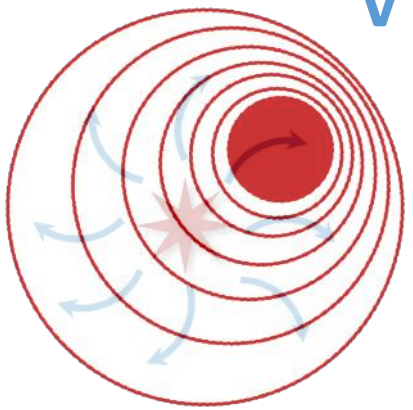




VT fusion

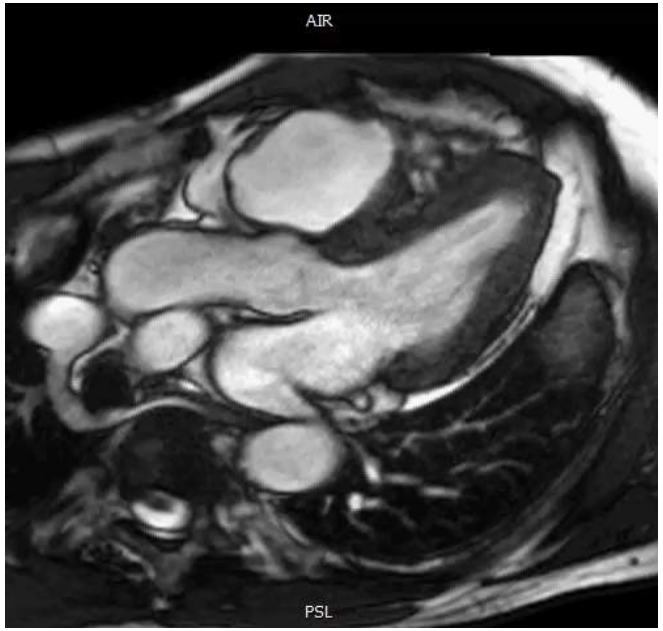


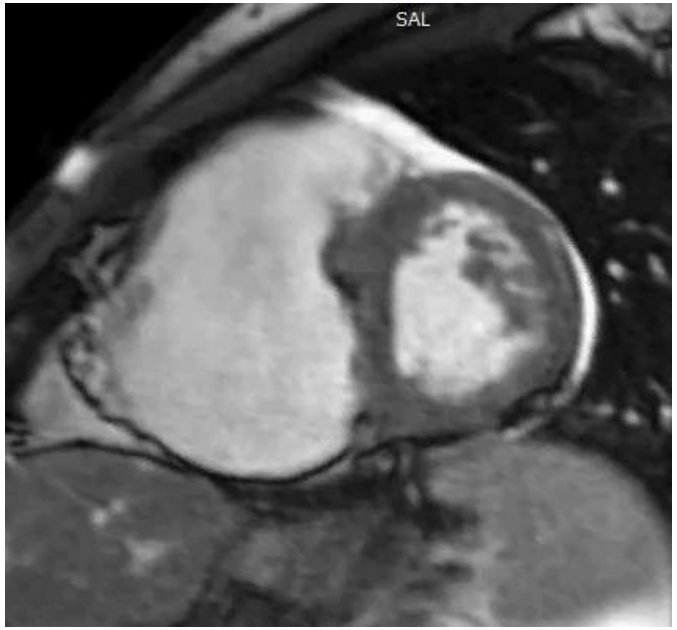
VT reset

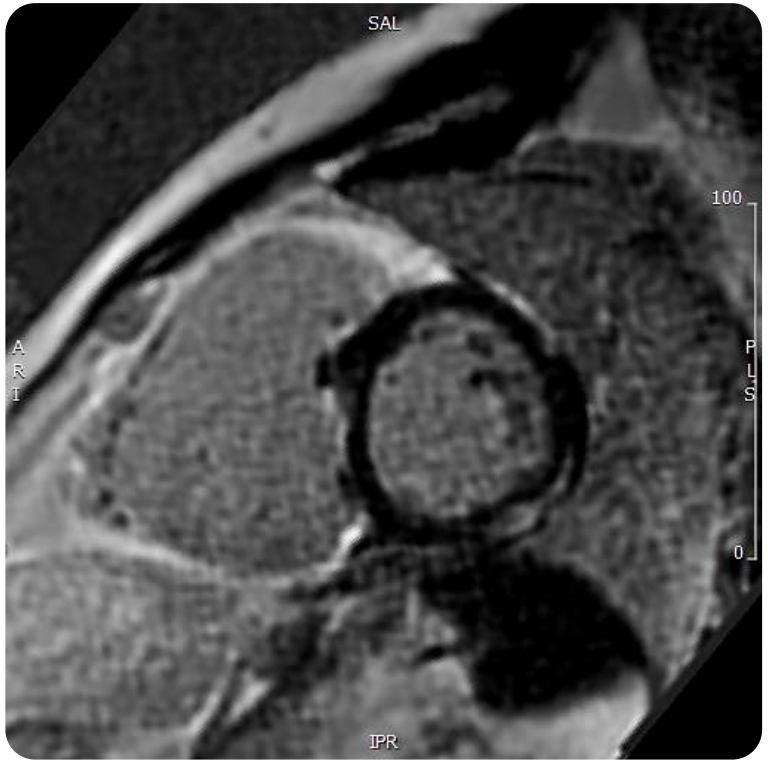


Cardiac magnetic resonance

- Severe RV dilatation (183 ml/m2) and dysfunction (EF: 27%), WM abnomalities (RV apex, ant free wall)
- Normal LV dimensions and function
- Extensive LGE along the RV free wall and at the epicardial aspect of the lateral LV wall

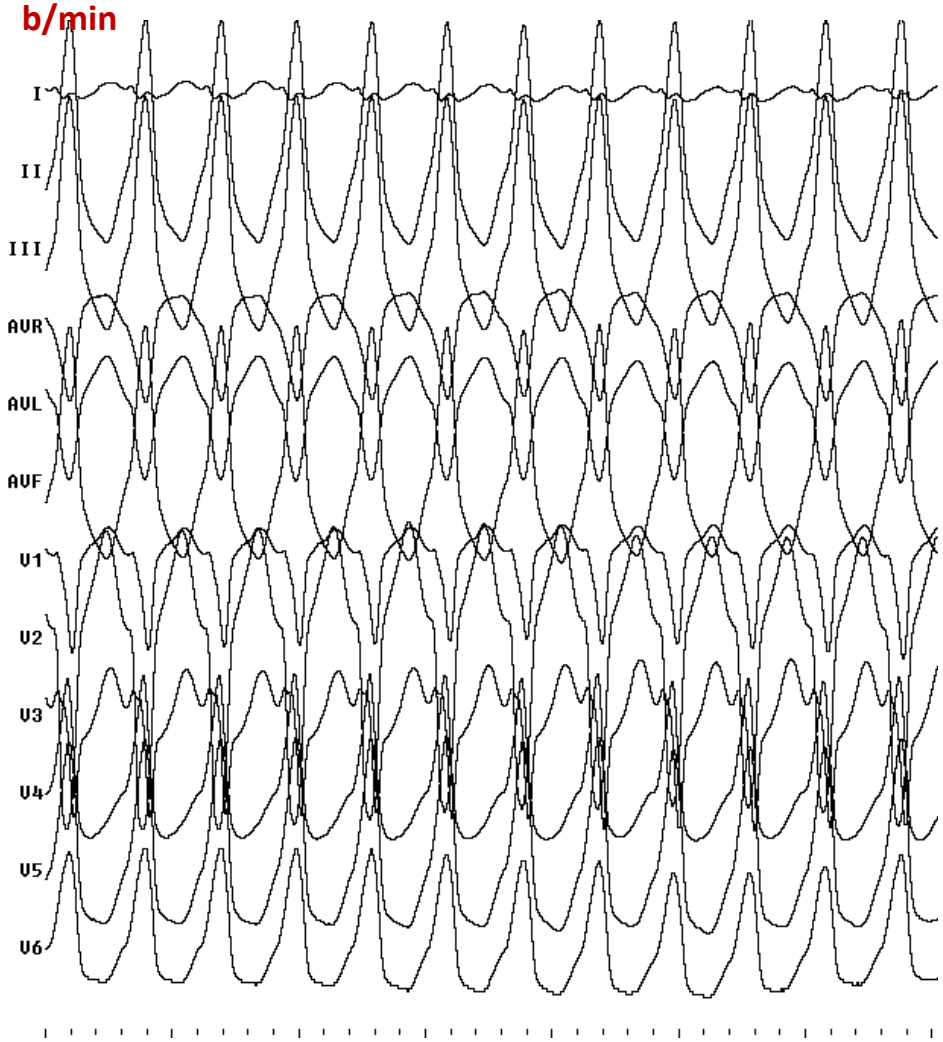






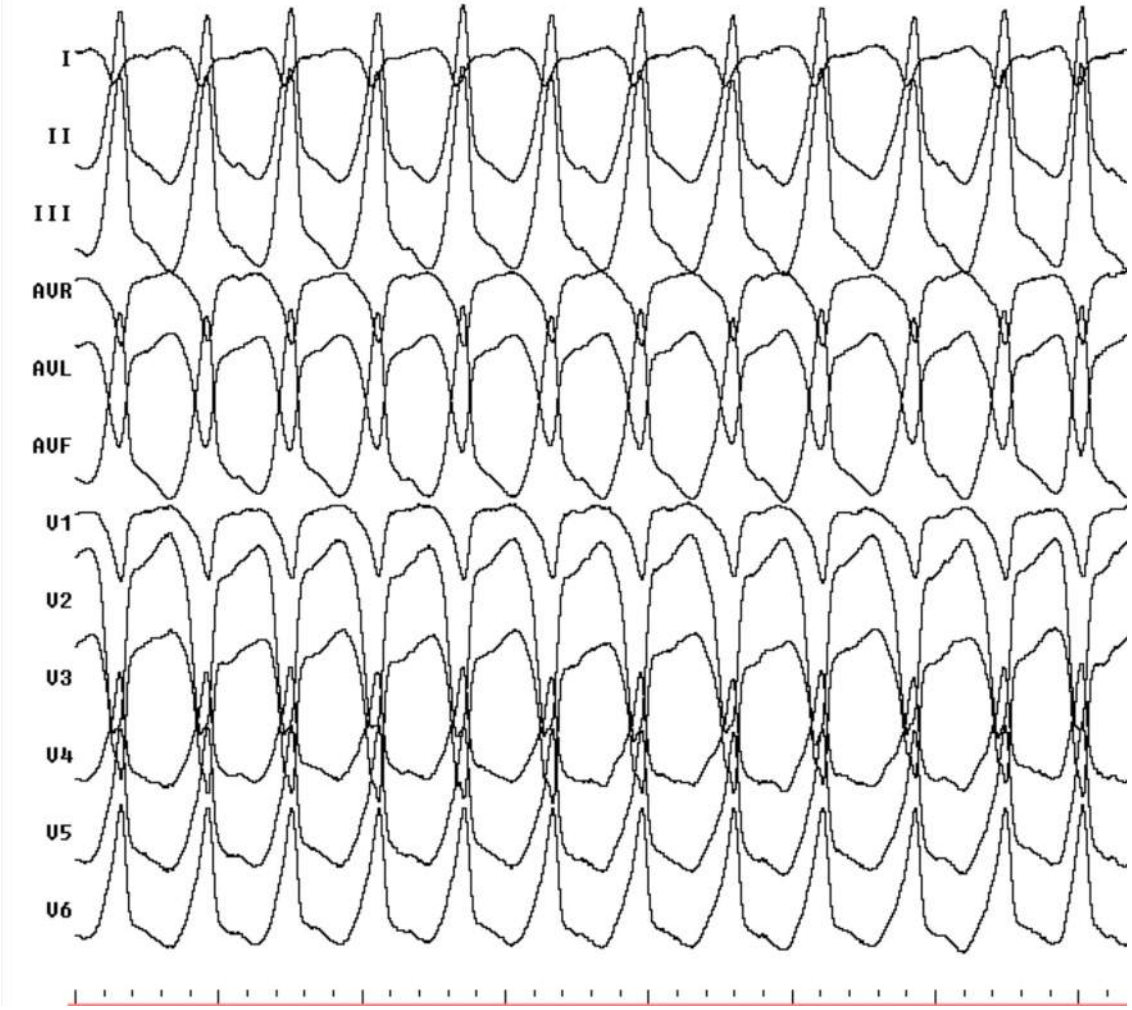
Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: mappaggio EA

TV tipo BBS, asse inferiore, transizione in V4, @210 b/min



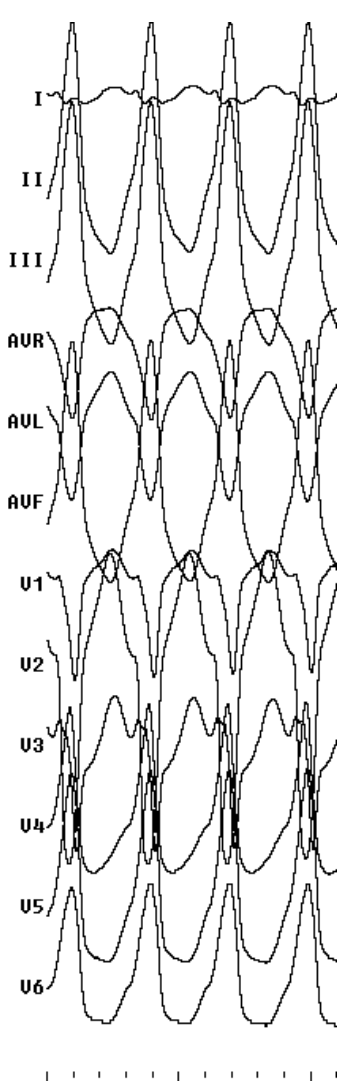
BC, f, 37 yrs

TV tipo BBS, asse inferiore, transizione in V4, @200 b/min

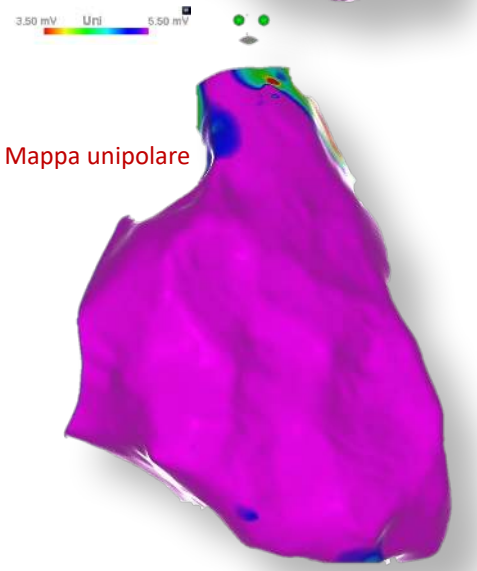


BV, f, 37 yrs

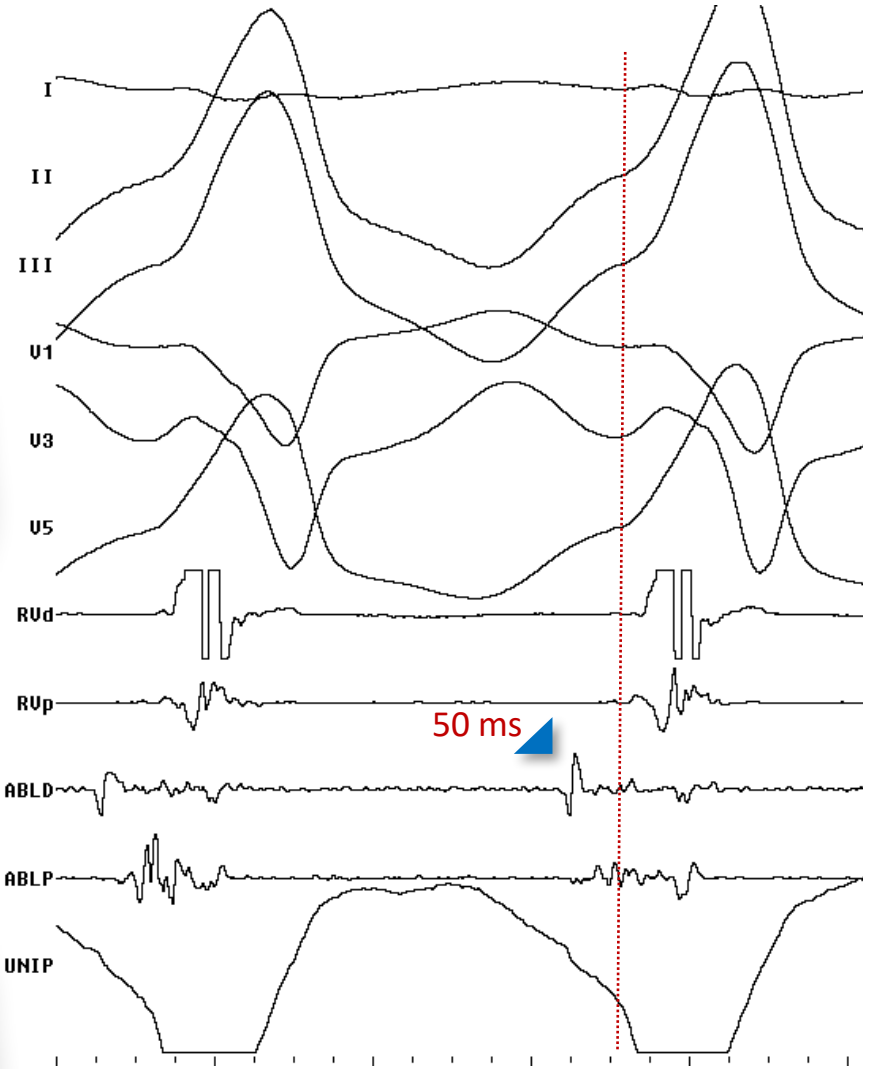
Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: mappaggio EA



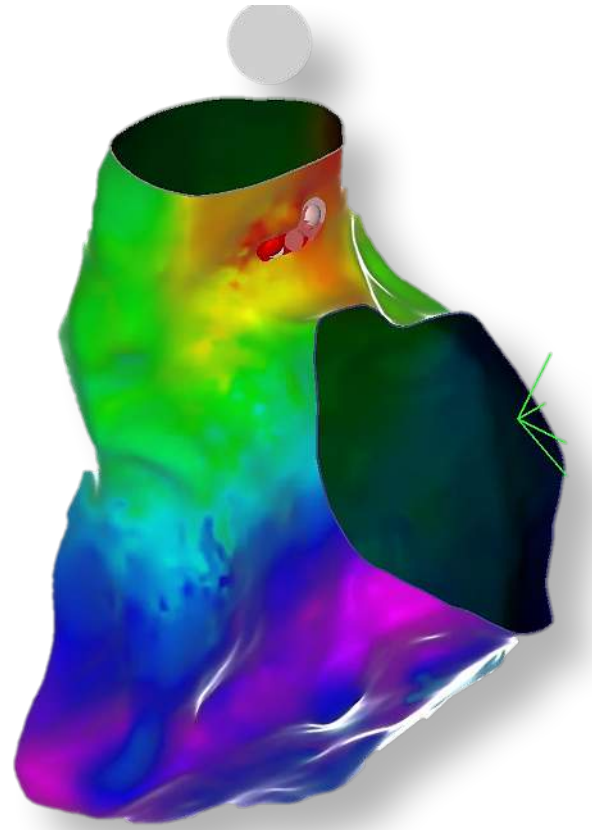
Mappa bipolare



Mappa unipolare



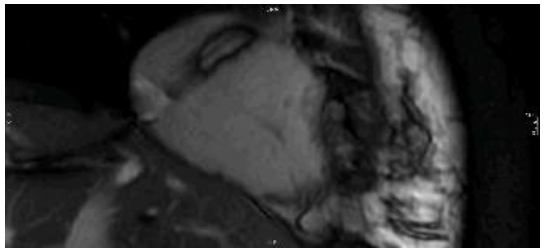
Mappa di attivazione

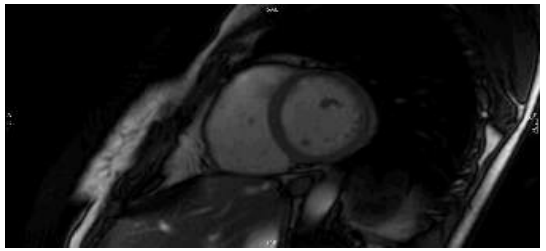


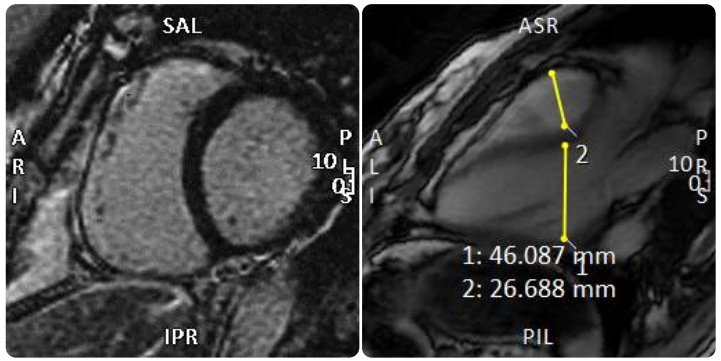
BC, f, 37 yrs

Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: mappaggio EA

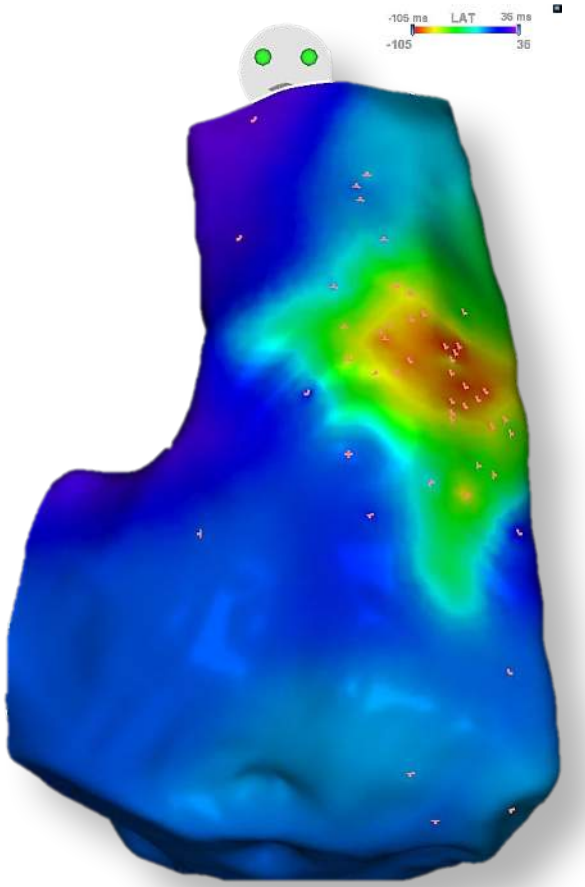
RMN



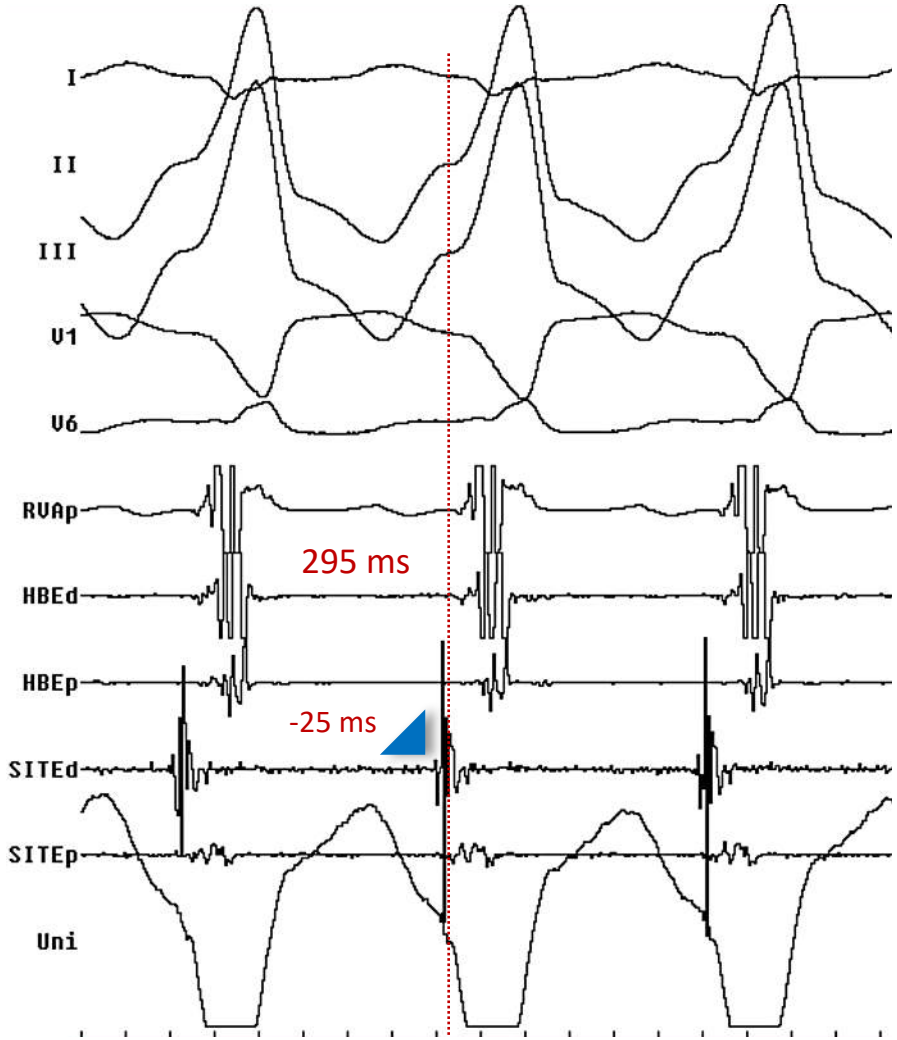




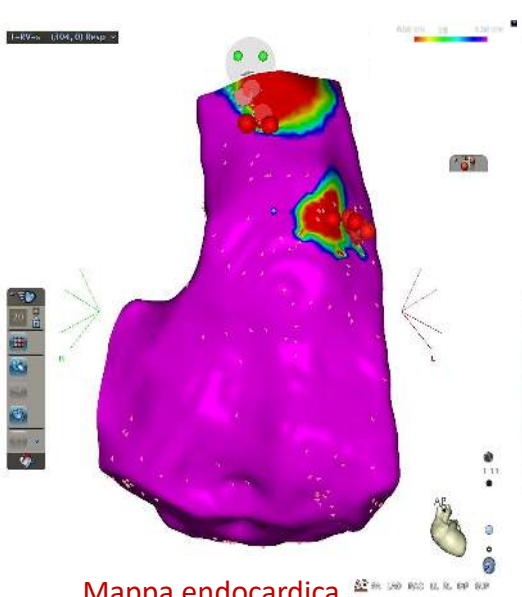
Mappaggio EA



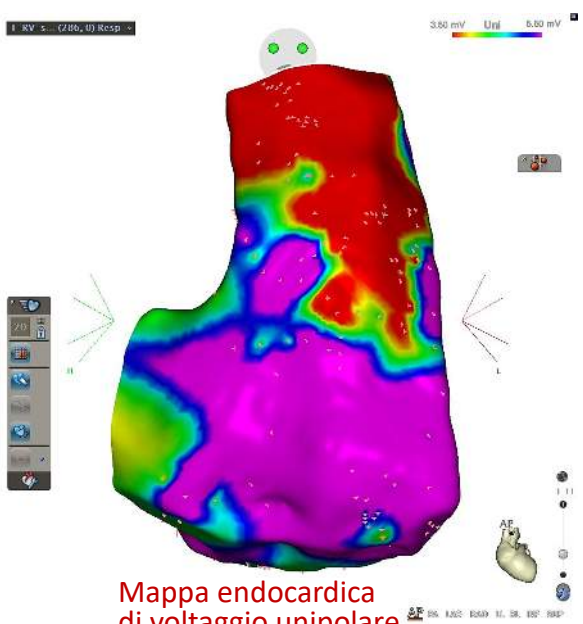
Mappaggio di attivazione (endo)



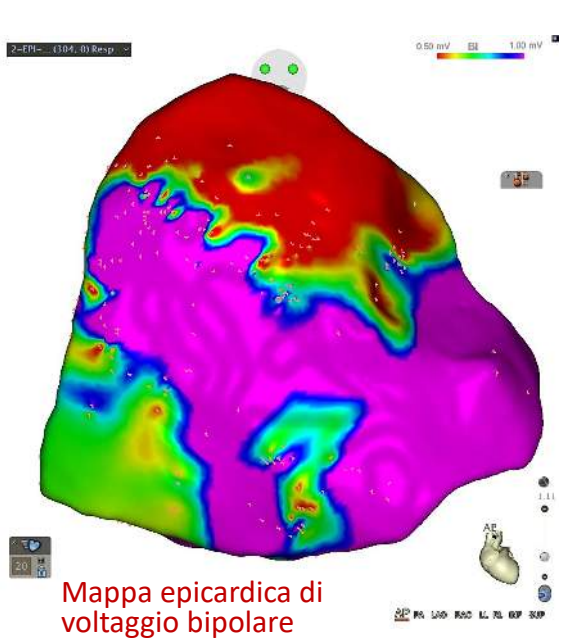
Diagnosi eziologica differenziale delle aritmie ventricolari ad origine dal VD: mappaggio EA



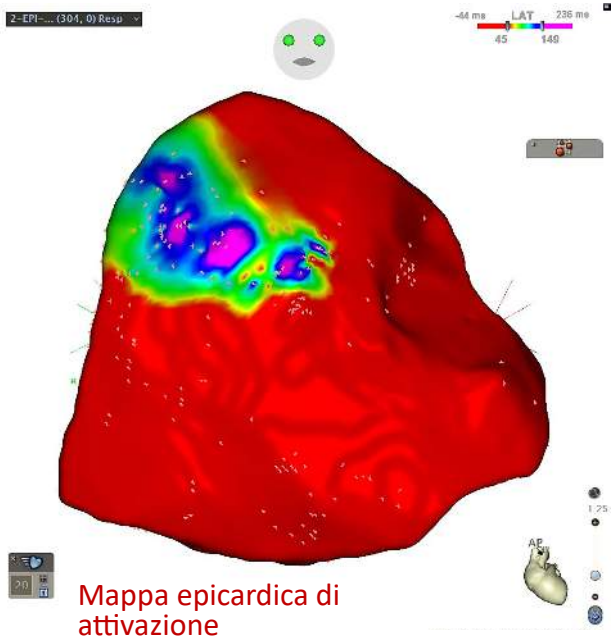
Mappa endocardica di voltaggio bipolare



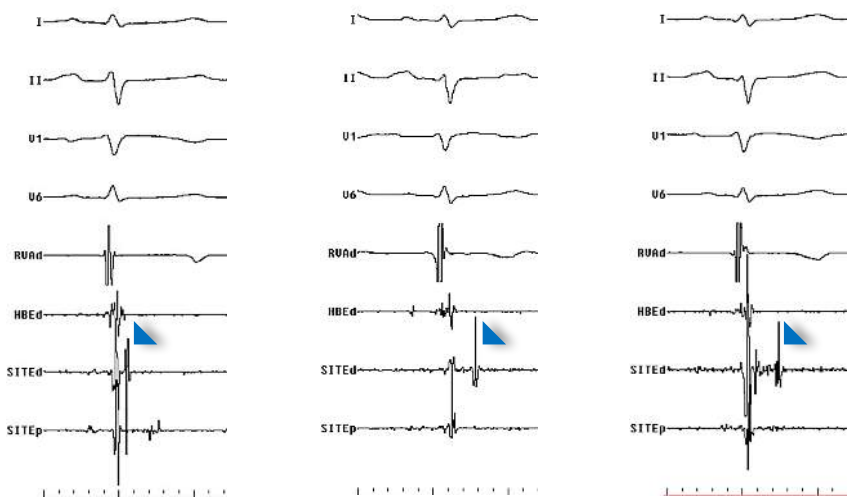
Mappa endocardica di voltaggio unipolare

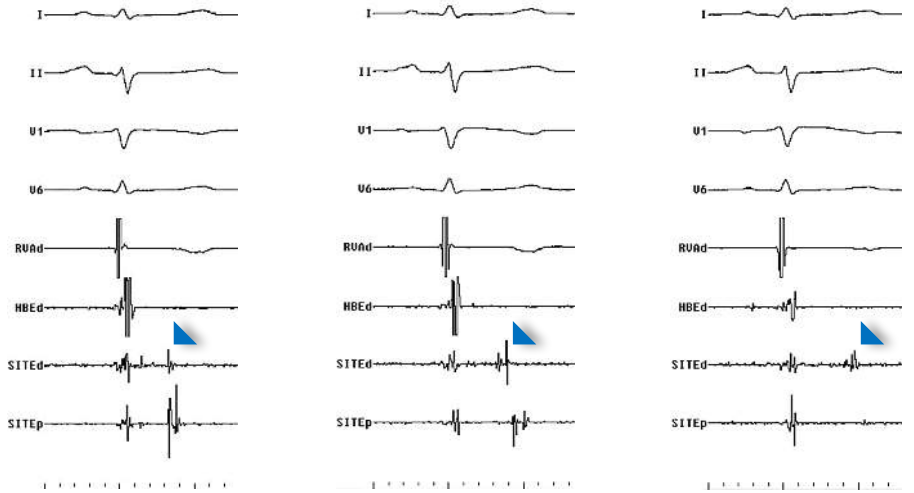


Mappa epicardica di voltaggio bipolare



Mappa epicardica di attivazione



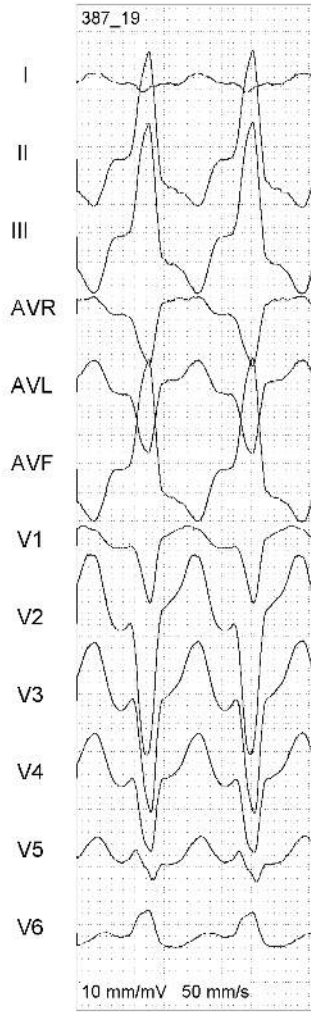


Perchè eseguire l'ablazione

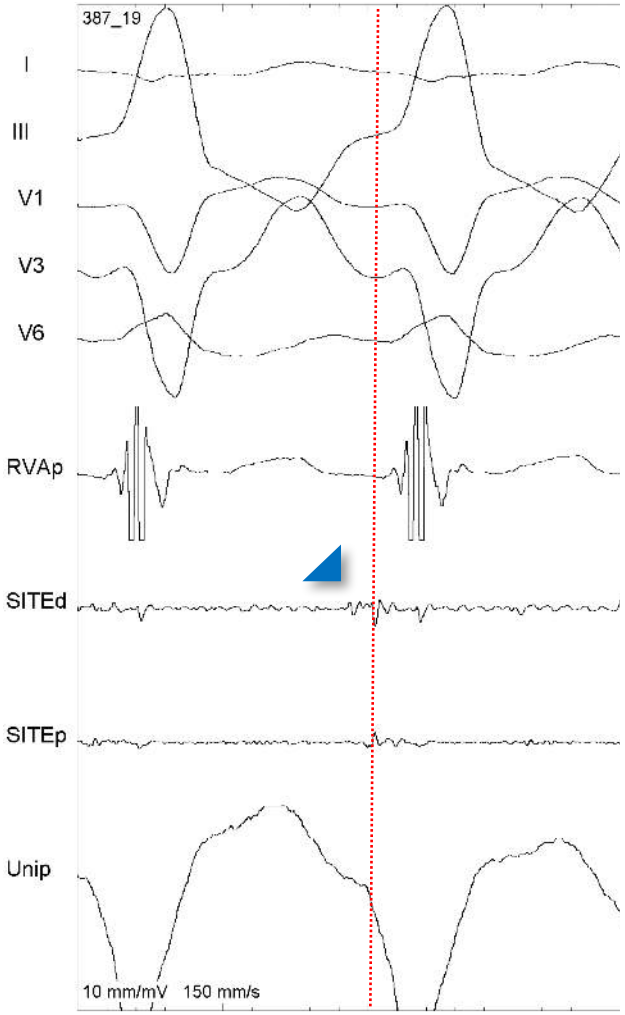
- Migliorare la qualità di vita (sintomi, interventi ICD)
- Regressione della disfunzione ventricolare sn indotta dalle extrasistoli frequenti
- Alternativa all'impianto di un ICD (pz selezionati con c. aritmogena destra)

Idiopathic RV arrhythmias: catheter ablation

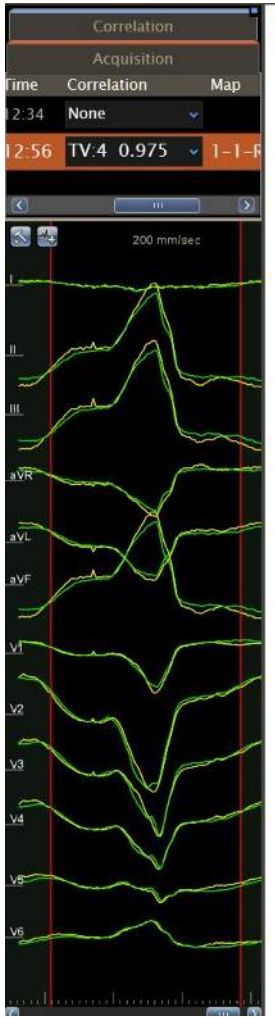
Baseline ECG



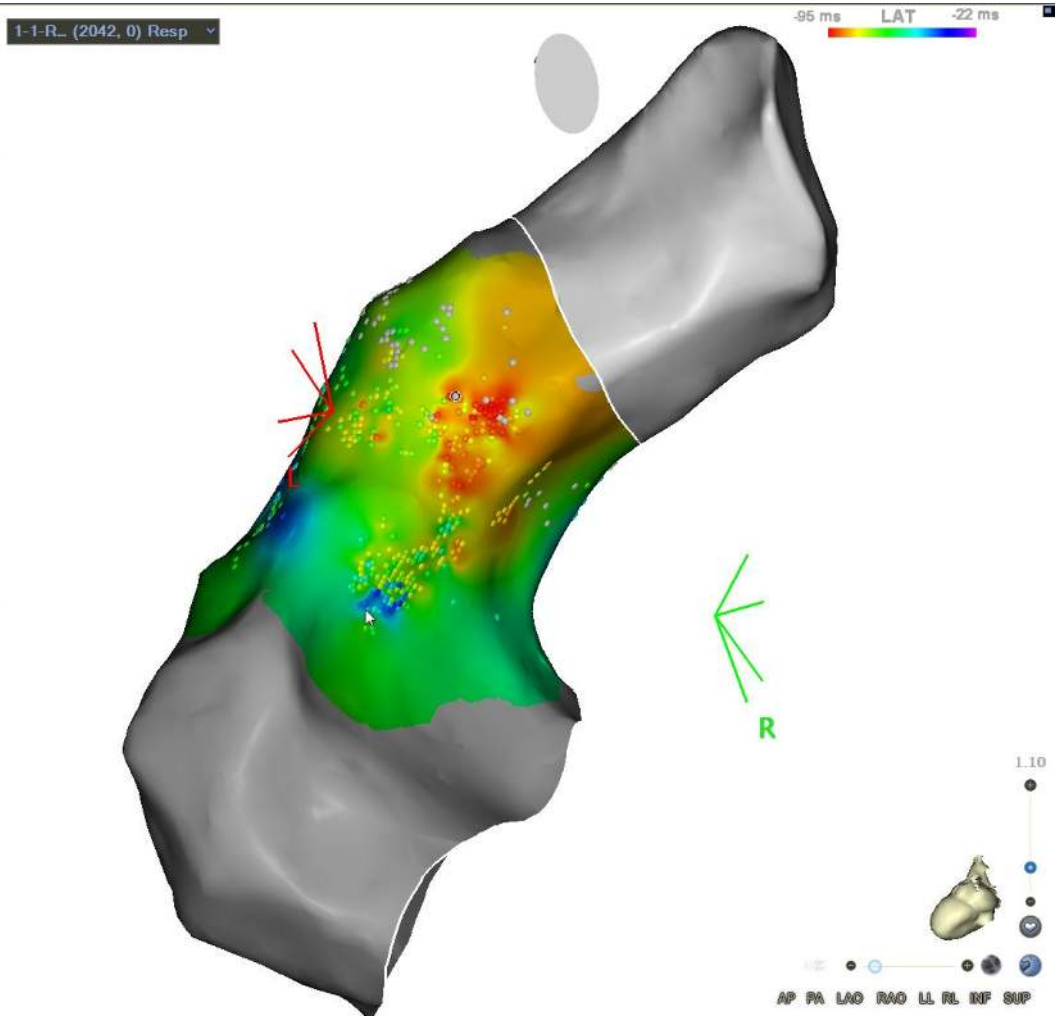
Activation mapping



Pace mapping

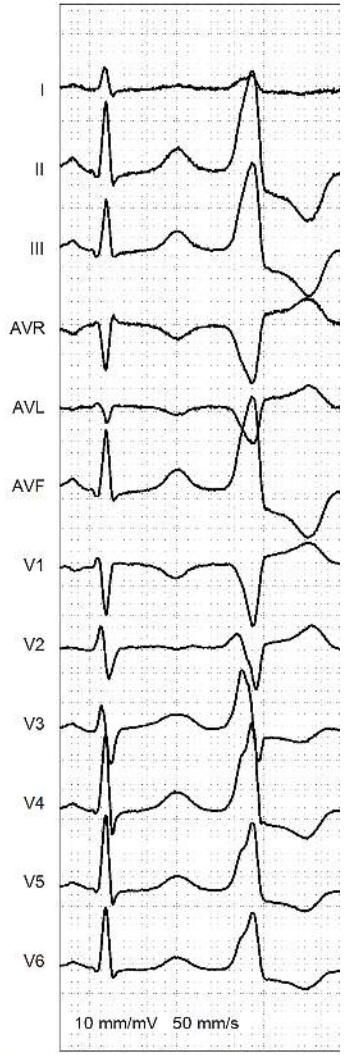


3D EAM activation mapping

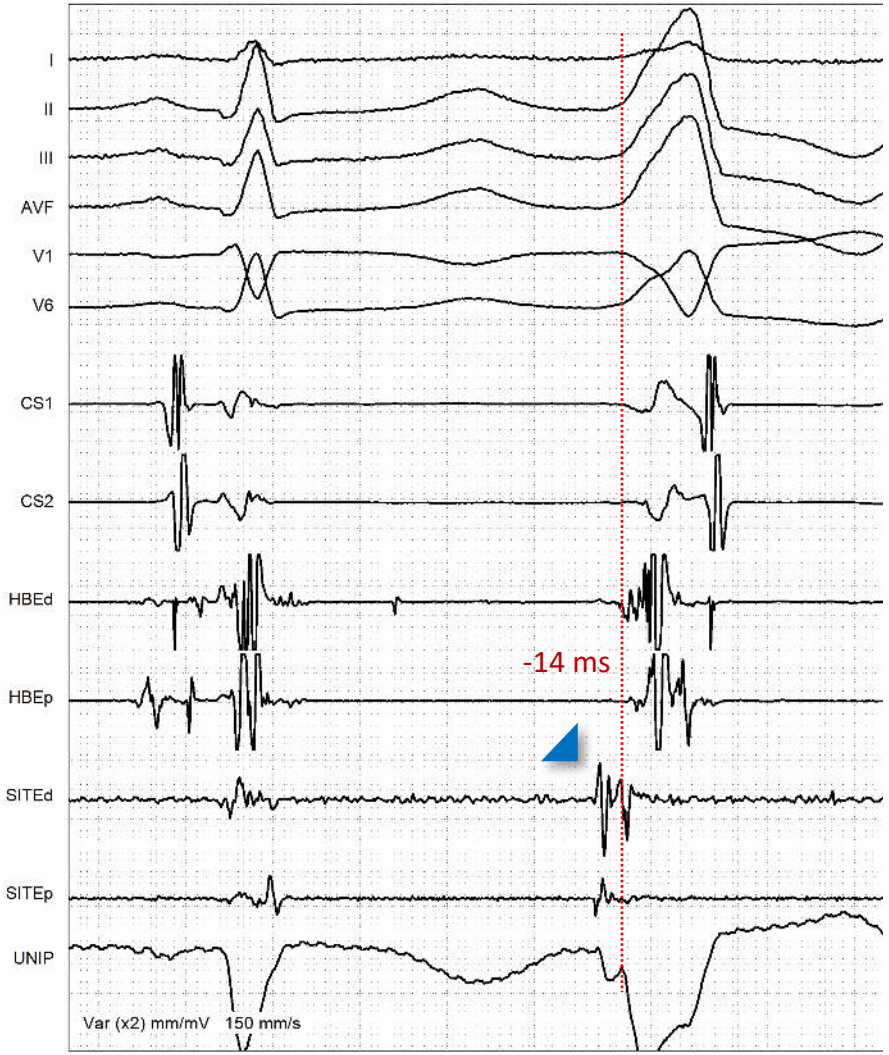


Catheter ablation of idiopathic RV arrhythmias: epicardial substrates

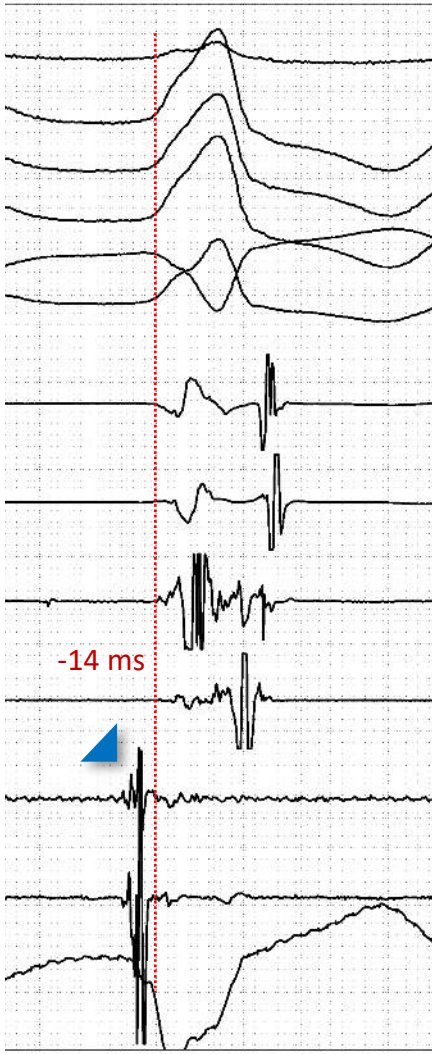
Baseline ECG

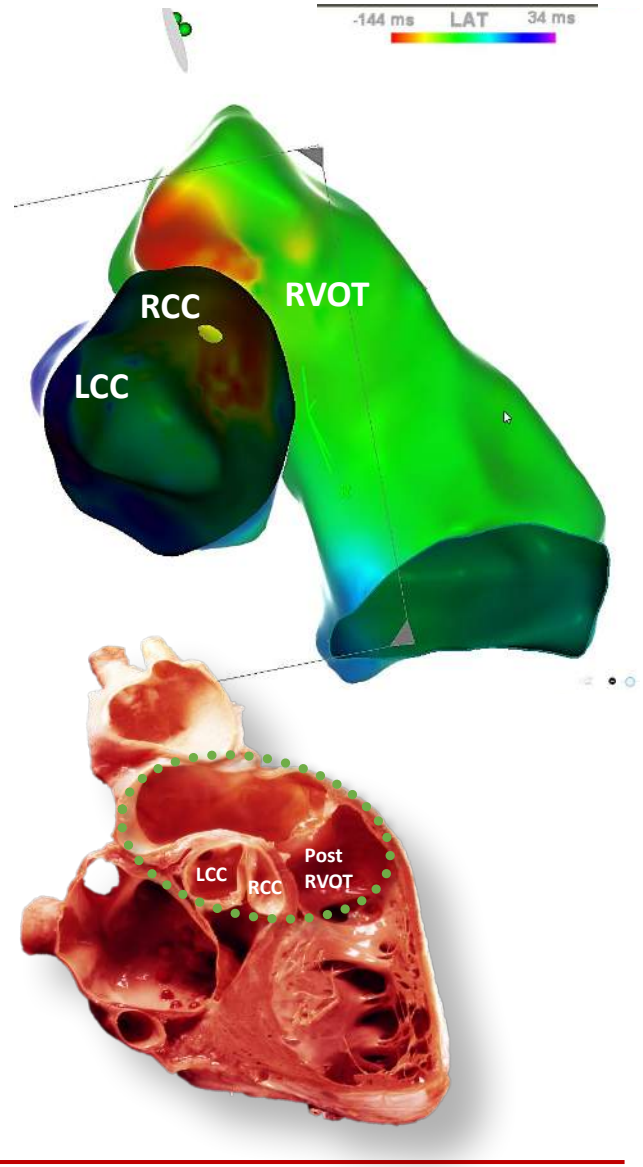


Right coronary cusp act mapping



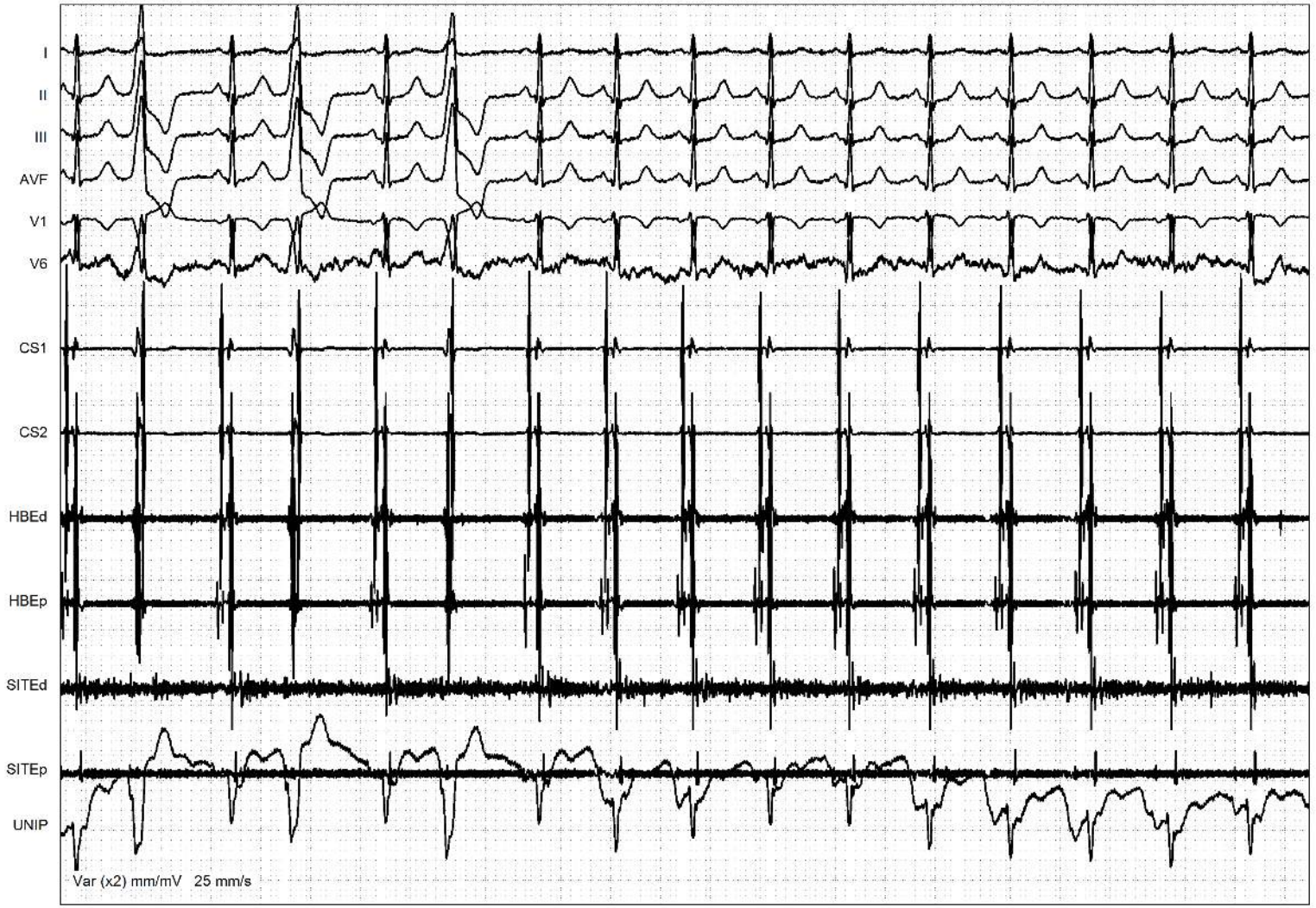
RVOT act mapping

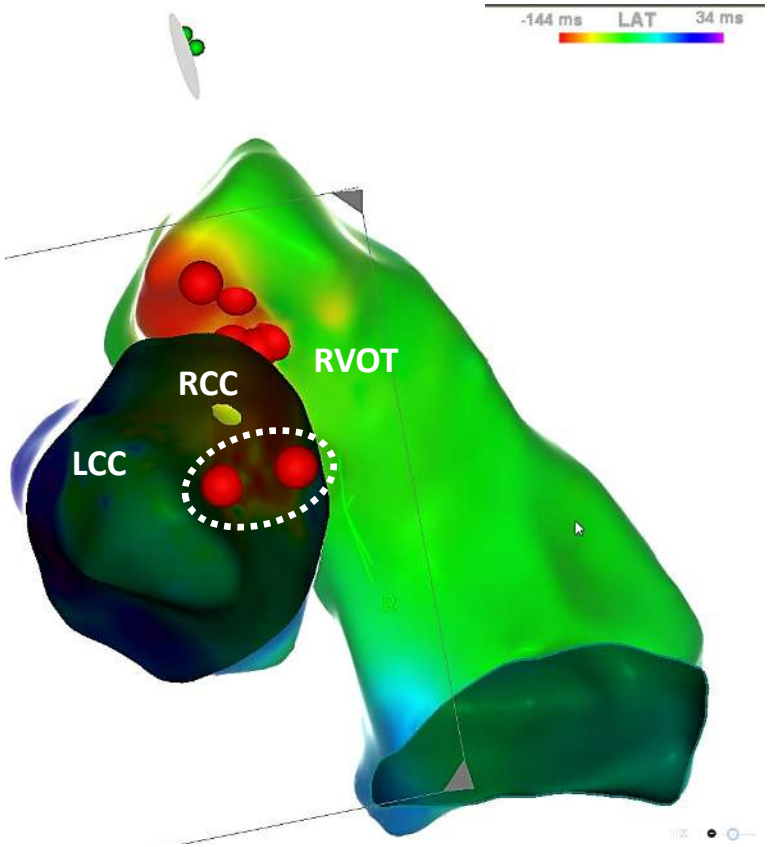




Catheter ablation of idiopathic RV arrhythmias: epicardial substrates

RF (RCC)

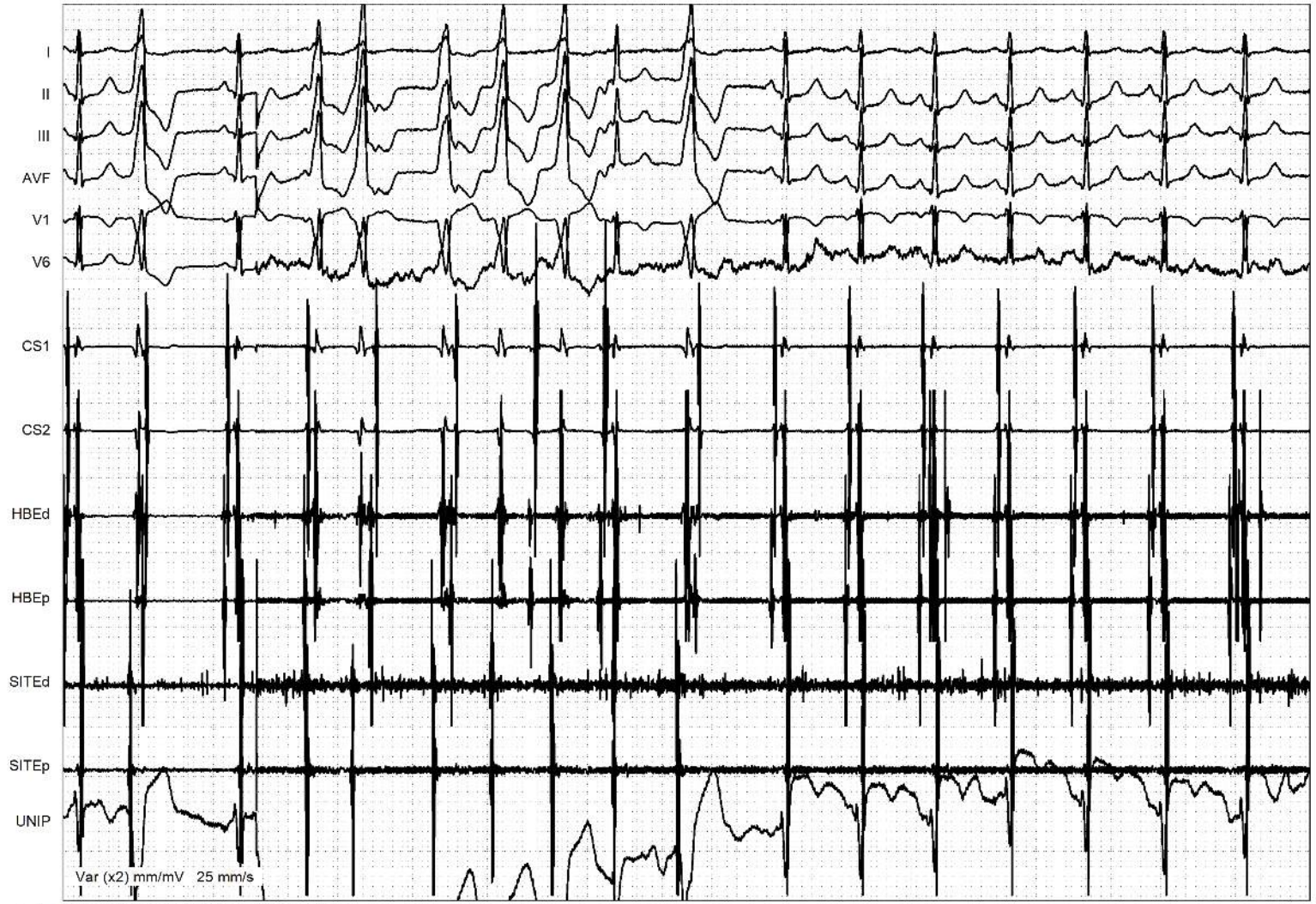


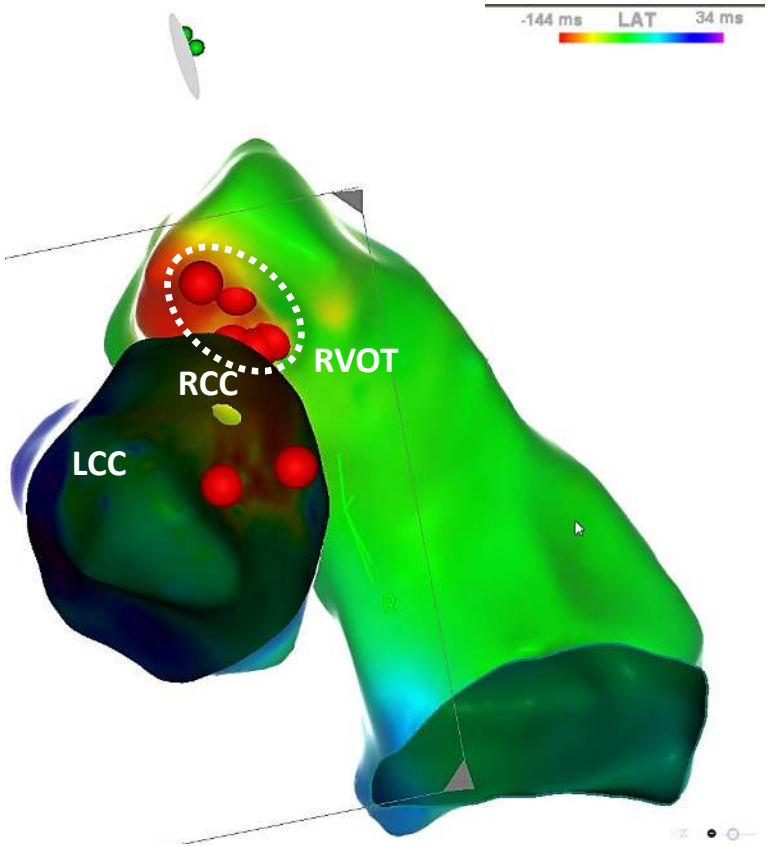


FM, m, 36 yrs

Catheter ablation of idiopathic RV arrhythmias: epicardial substrates

RF (posterior RVOT)

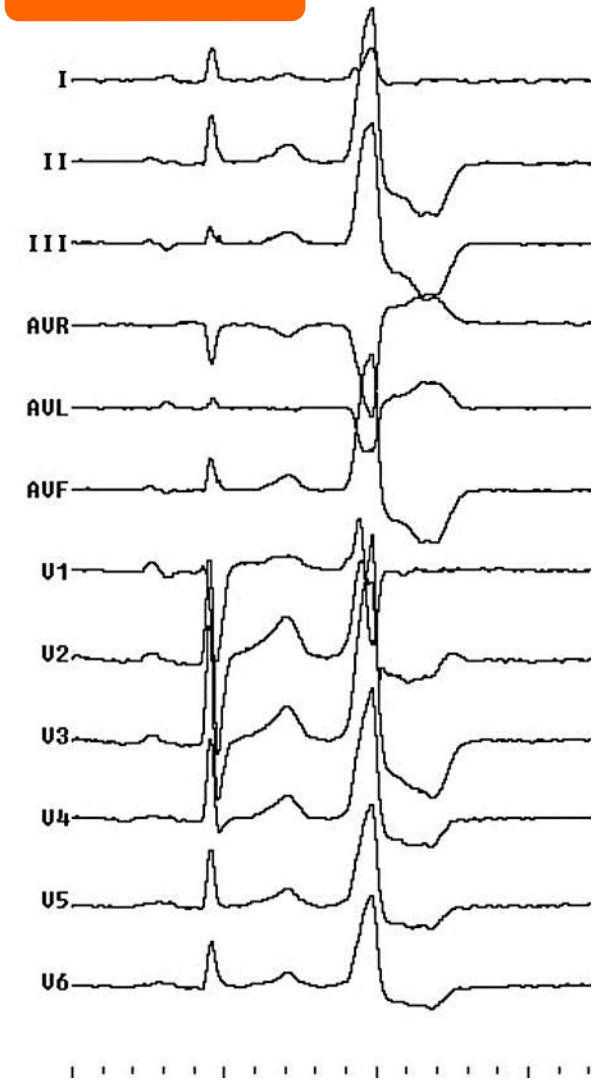




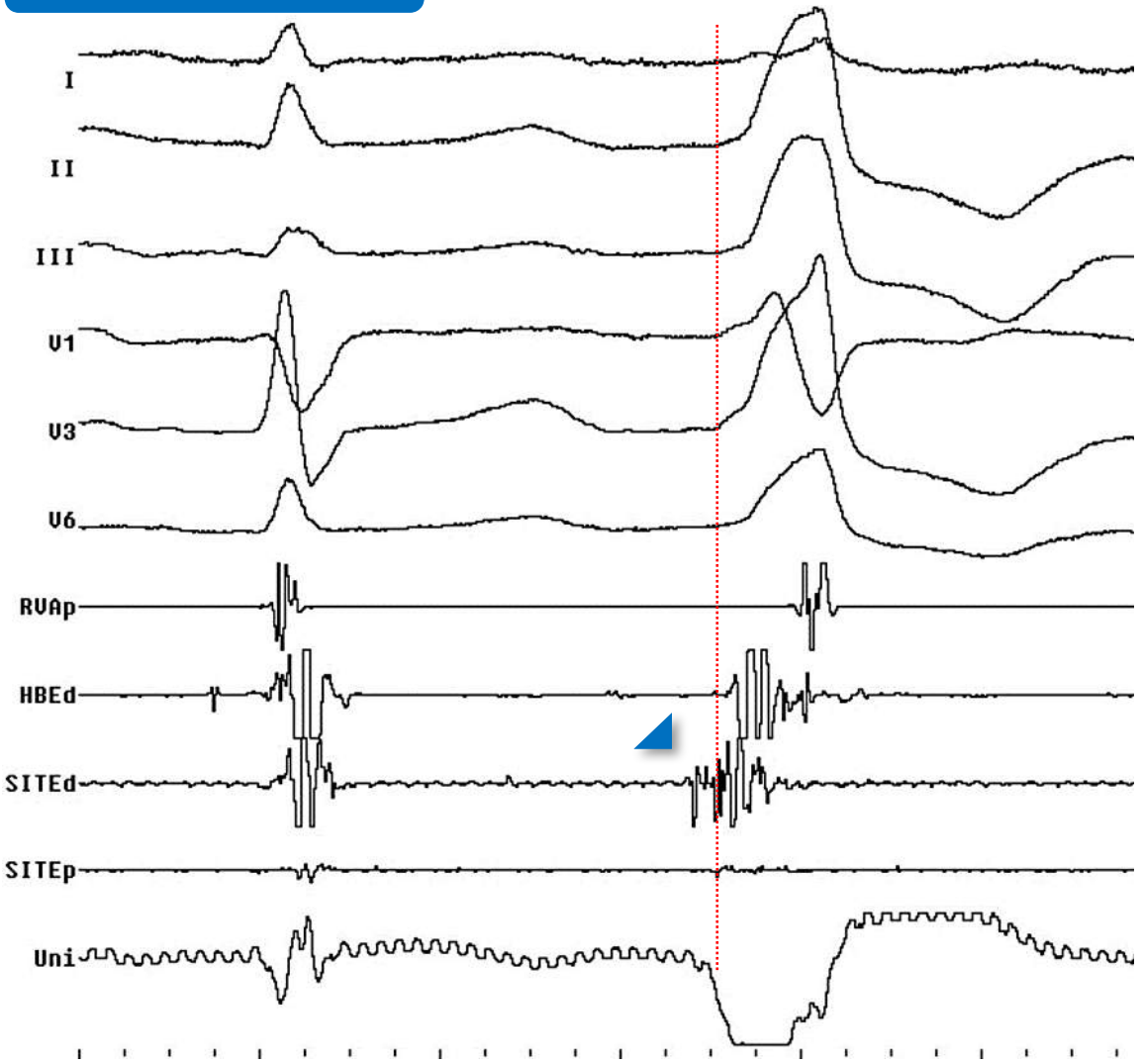
FM, m, 36 yrs

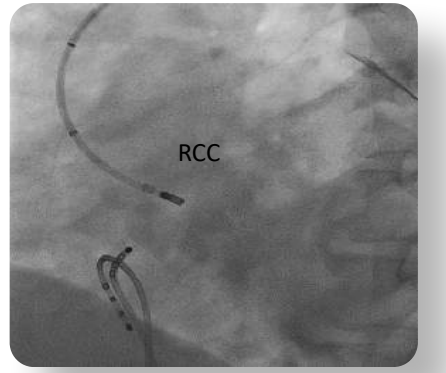
Catheter ablation of idiopathic RV arrhythmias: epicardial substrates

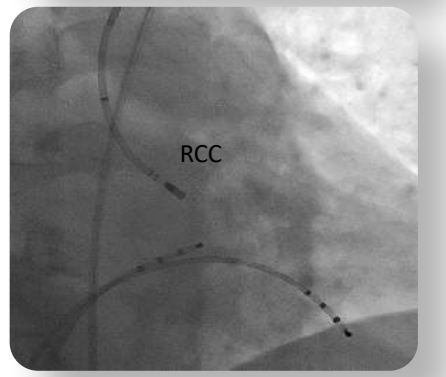
Baseline ECG



Activation mapping

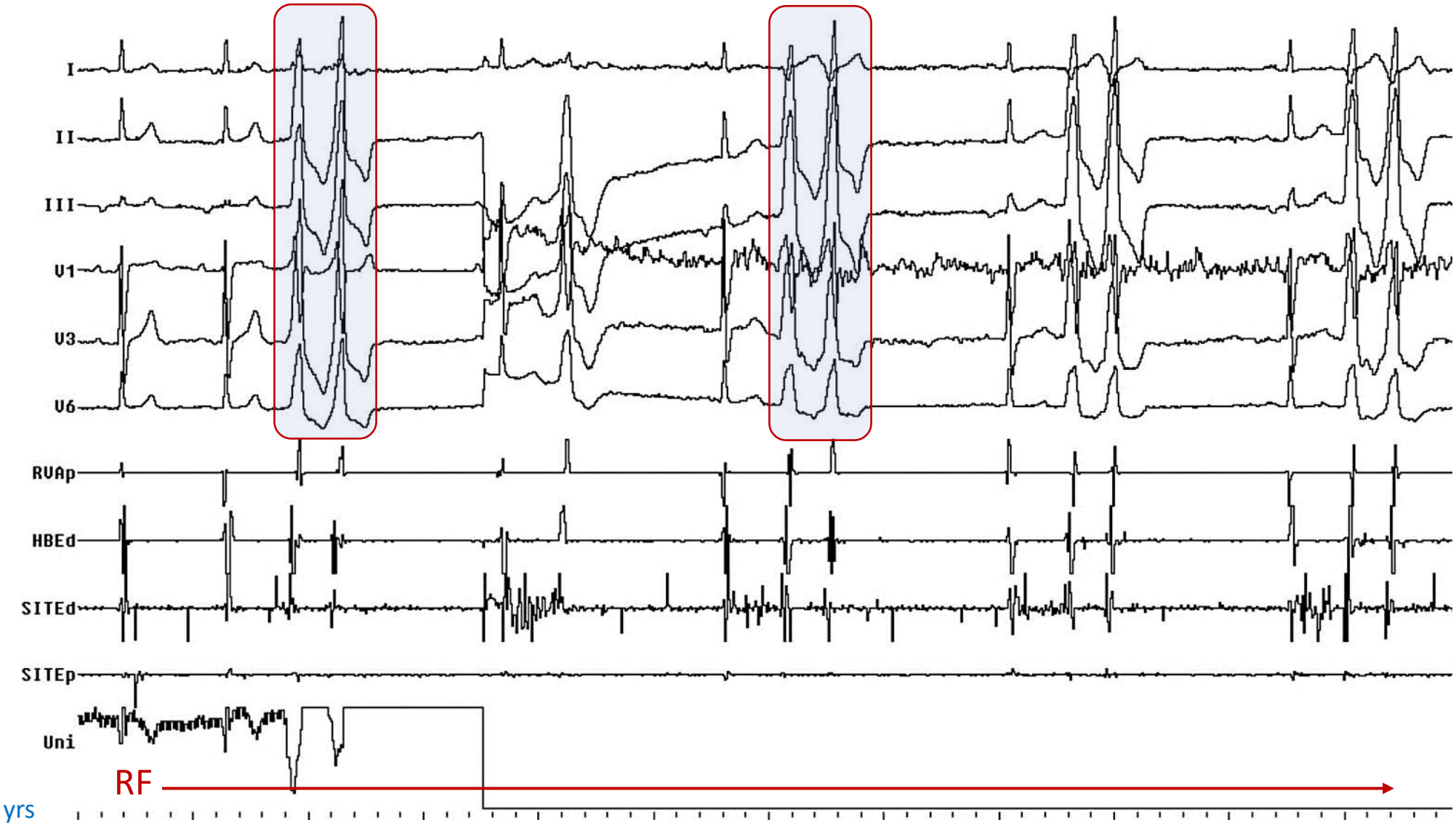






AA, m, 65 yrs

Catheter ablation of idiopathic RV arrhythmias: epicardial substrates

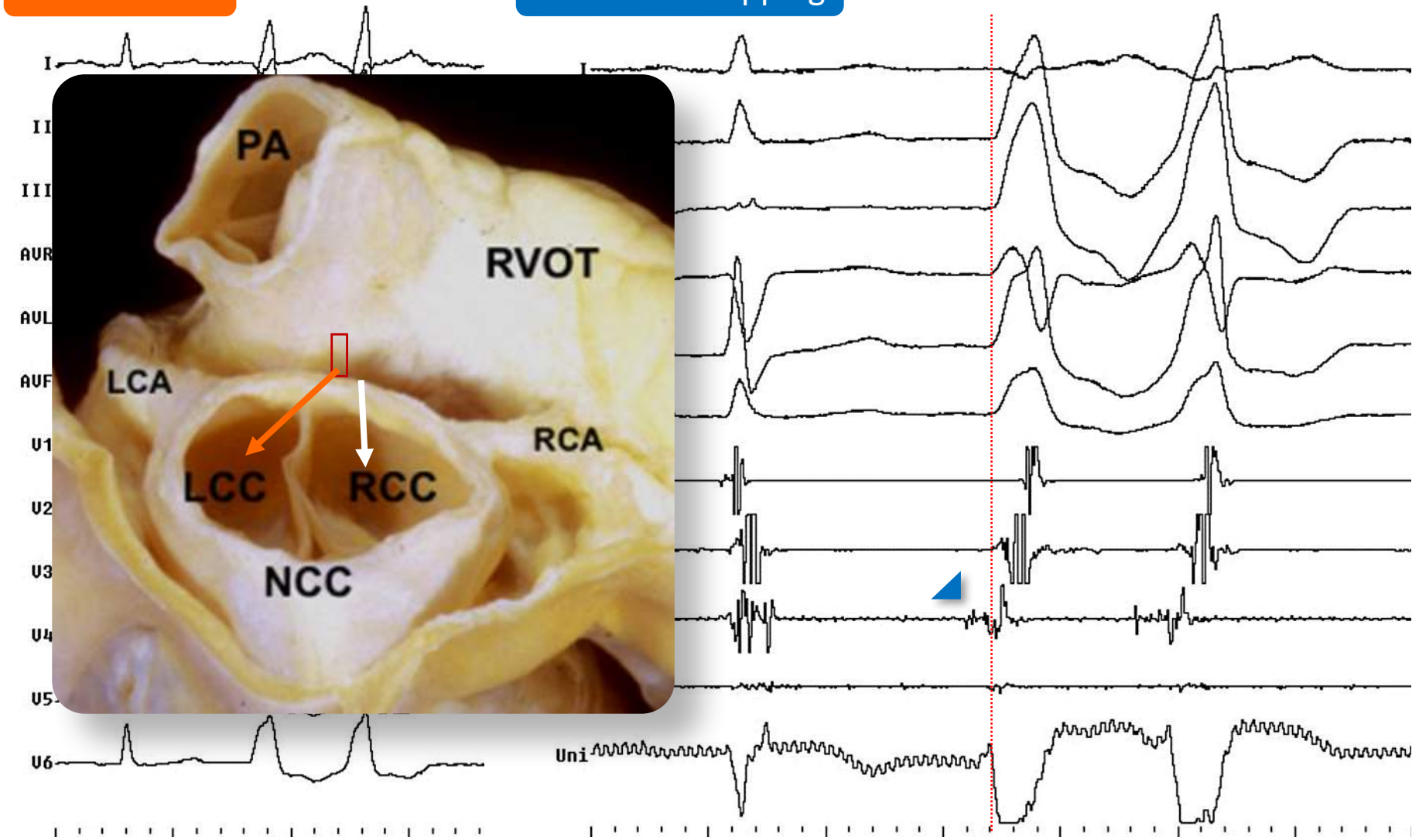


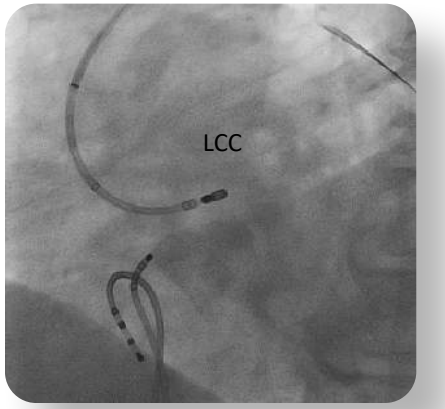
AA, m, 65 yrs

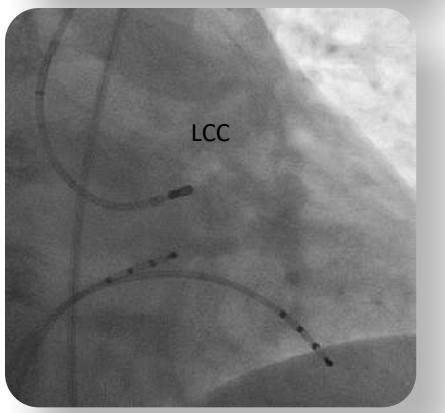
Catheter ablation of idiopathic RV arrhythmias: epicardial substrates

Baseline ECG

Activation mapping







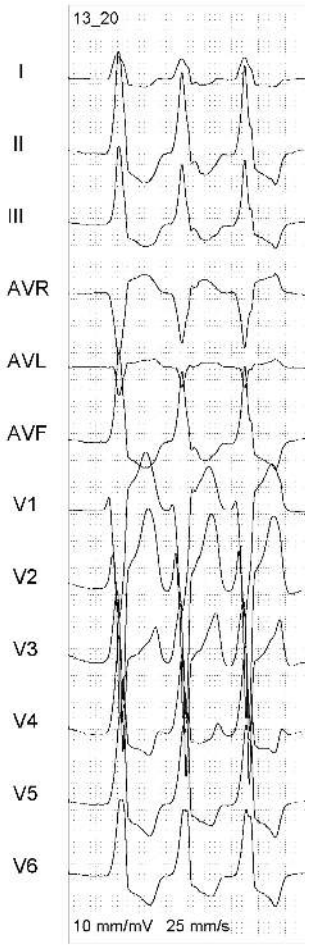
AA, m, 65 yrs

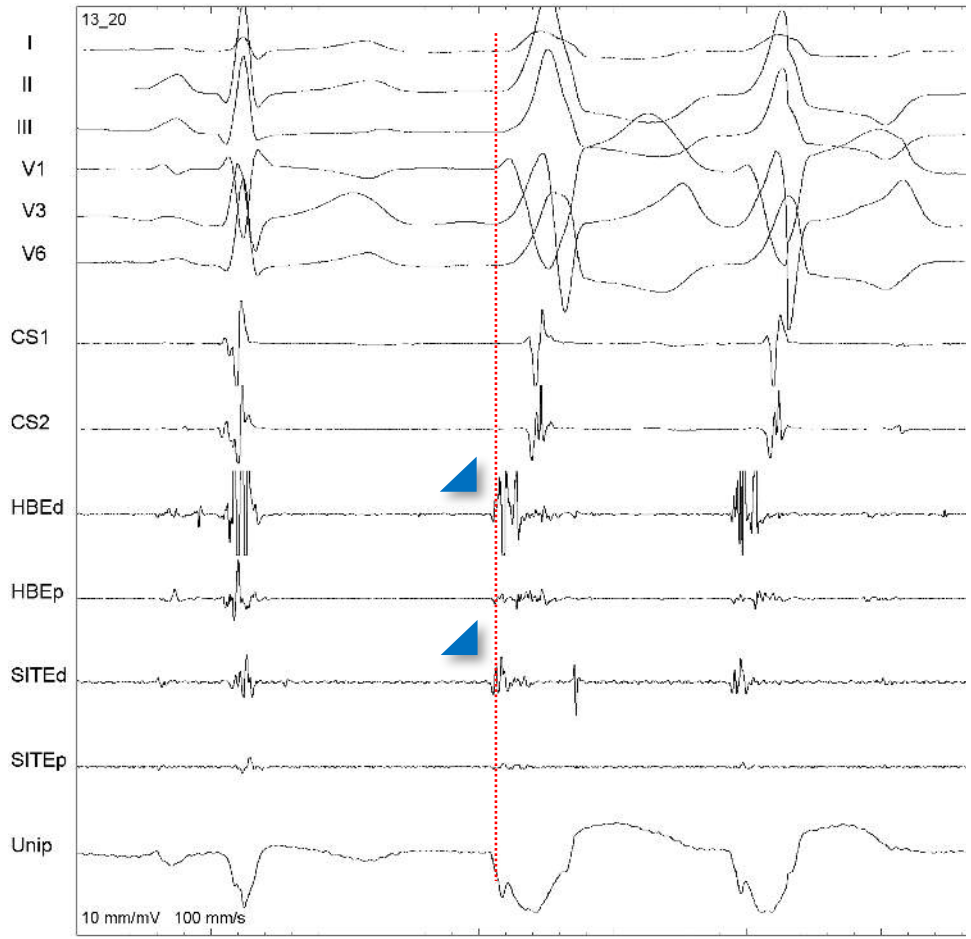
Catheter ablation of idiopathic RV arrhythmias: alternative «vantage points»

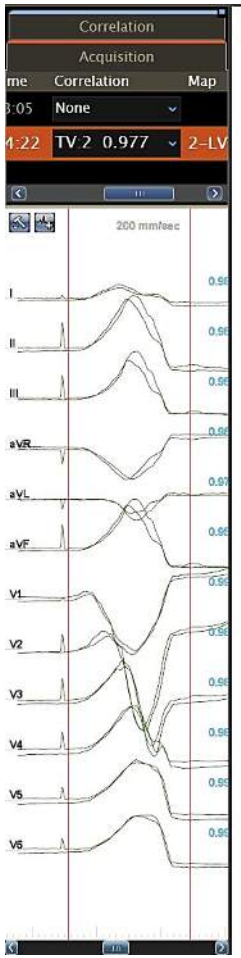
Baseline ECG

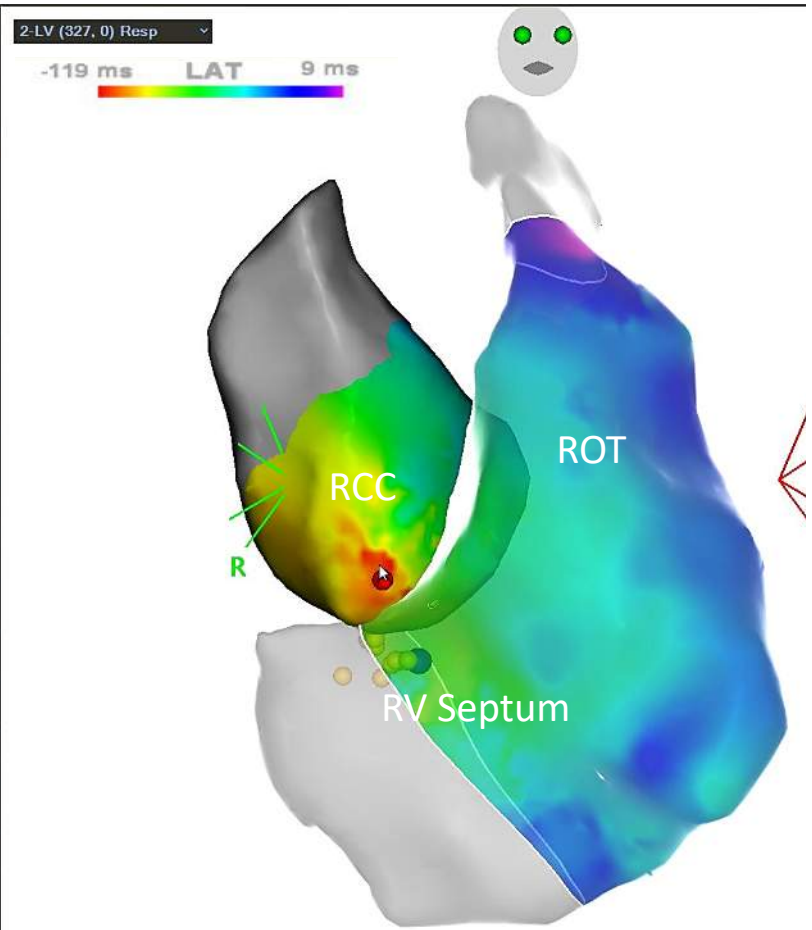
Activation mapping

Pace mapping and 3D EA mapping





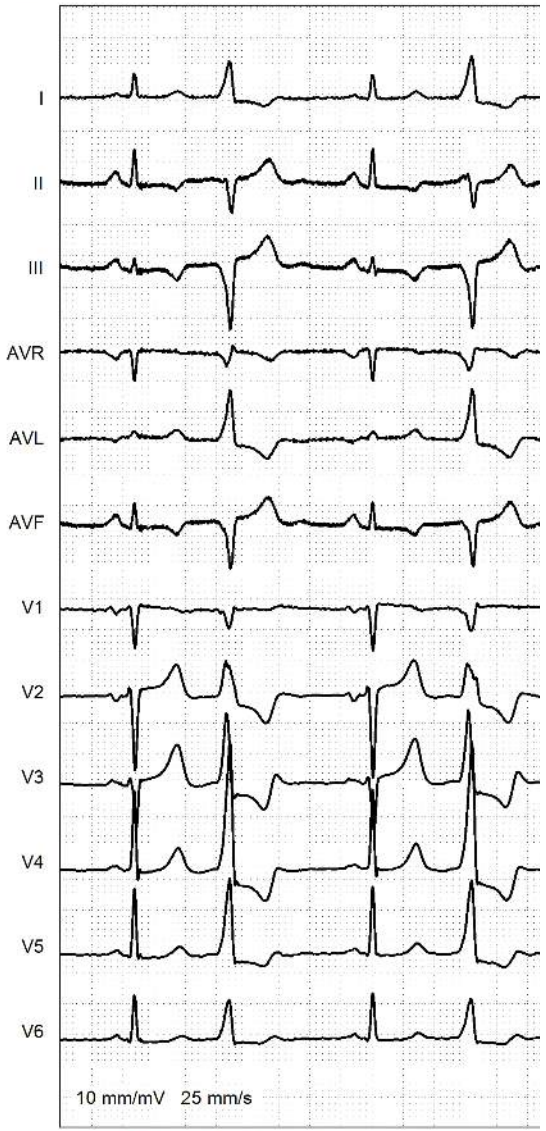


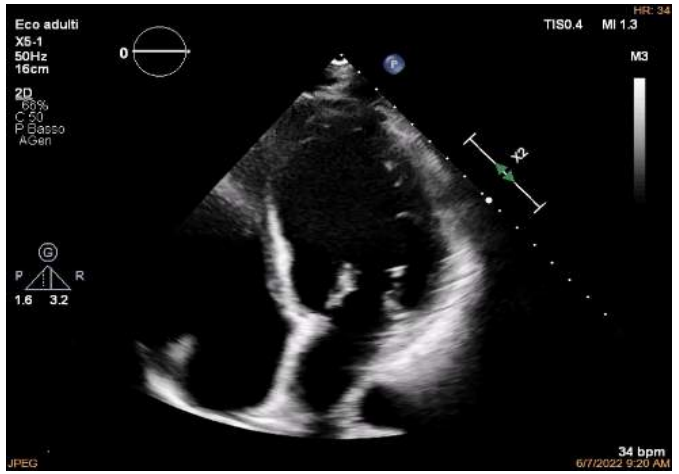


SS, m, 17 yrs

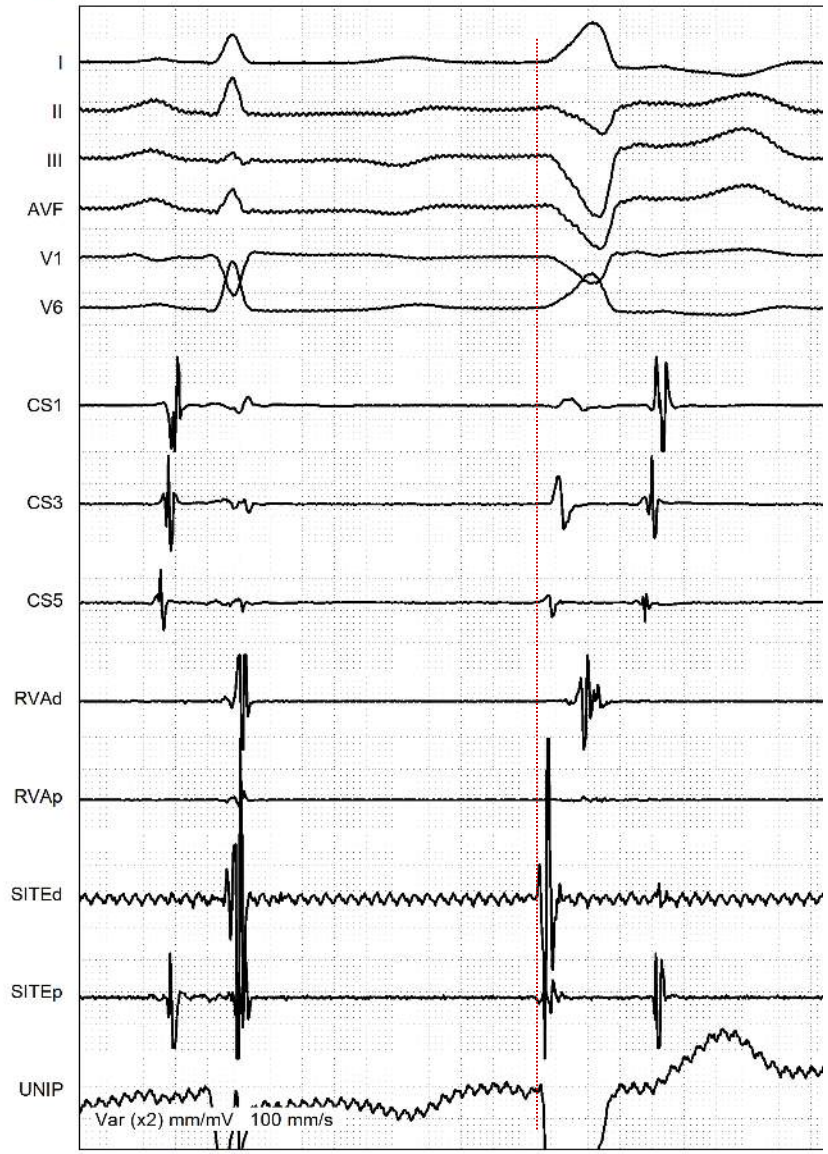
Caso clinico

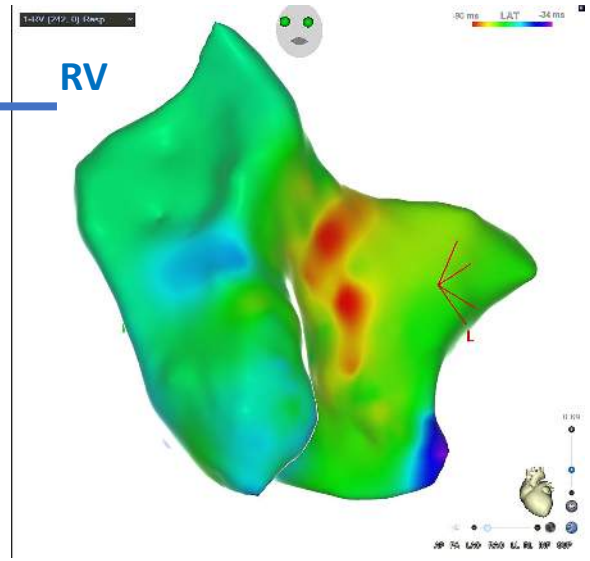
- TS, 49 a, f
- 5/2022: riscontro ECG di ExVe monomorfe (idoneità sportiva)
- Holter: 35.000 ExVe (40% battiti totali)
- Disfunzione Vsn all'ECO2D (FE: 35-40%)
- Soppressione parziale delle ExVe durante sforzo
- Coronarie normali all'esame angiografico
- Assenza di LGE alla RMN cuore
- 8/2022: ablazione transcatetere ExVe (origine dal processo posteriore-superiore del ventricolo sn – approccio bi-ventricolare)

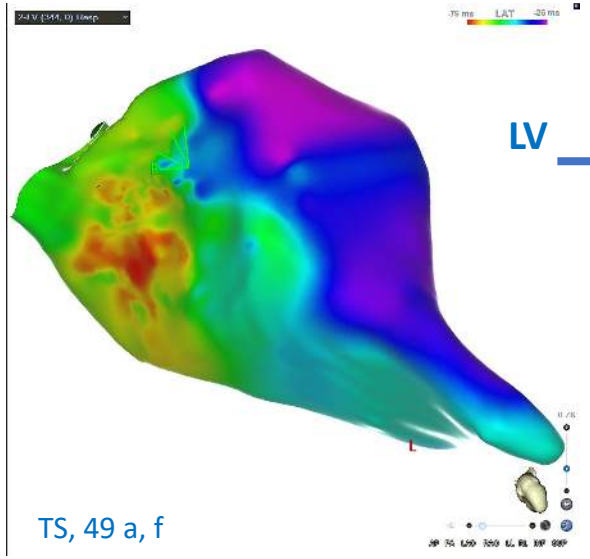




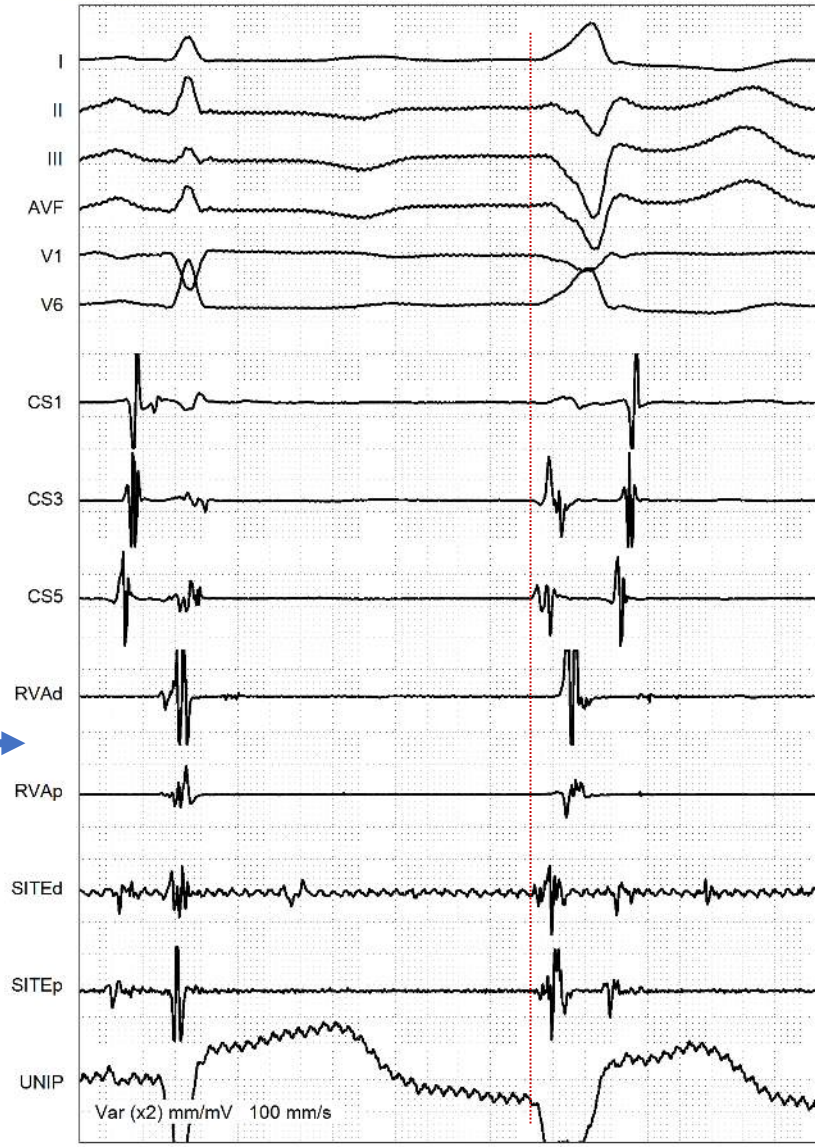
Aritmie ventricolari ad origine dal VD: ablazione transcatetere



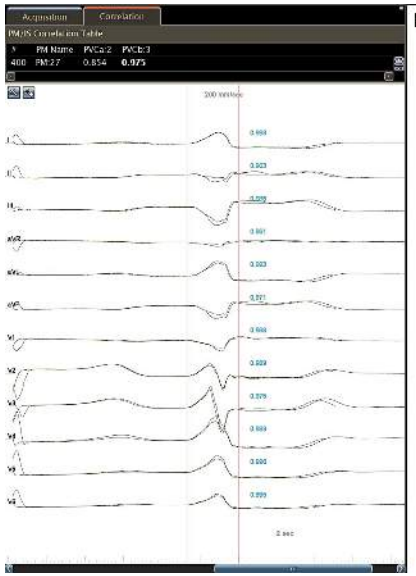


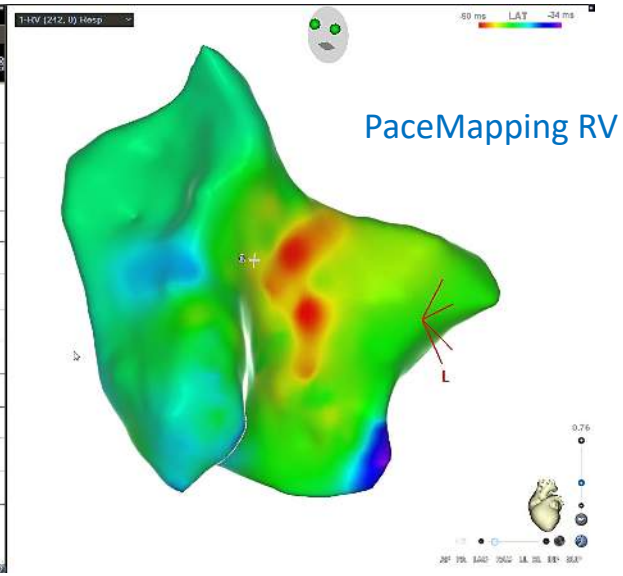


TS, 49 a, f

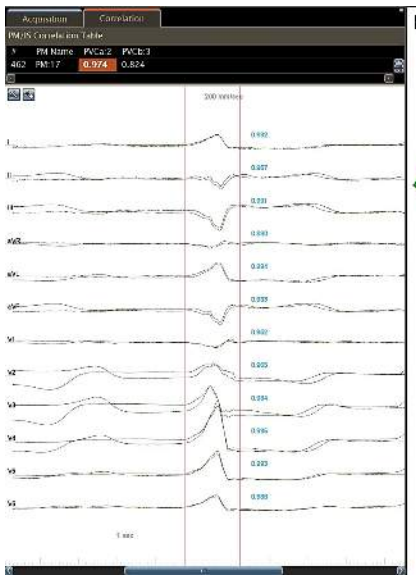


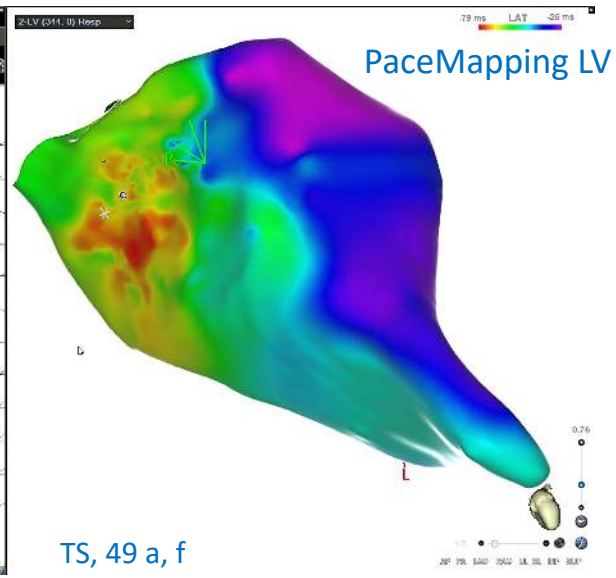
Aritmie ventricolari ad origine dal VD: ablazione transcatetere





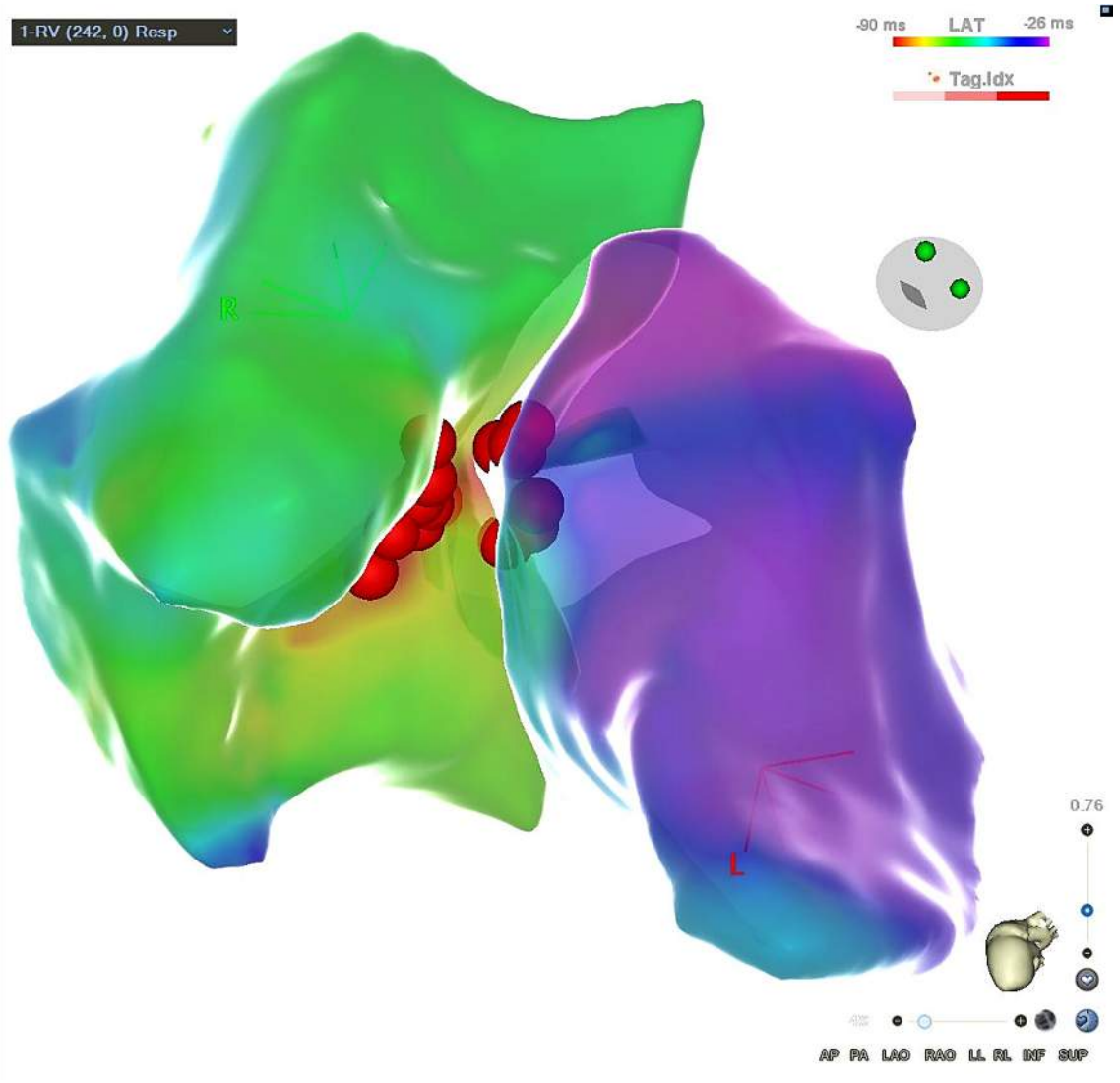
PaceMapping RV



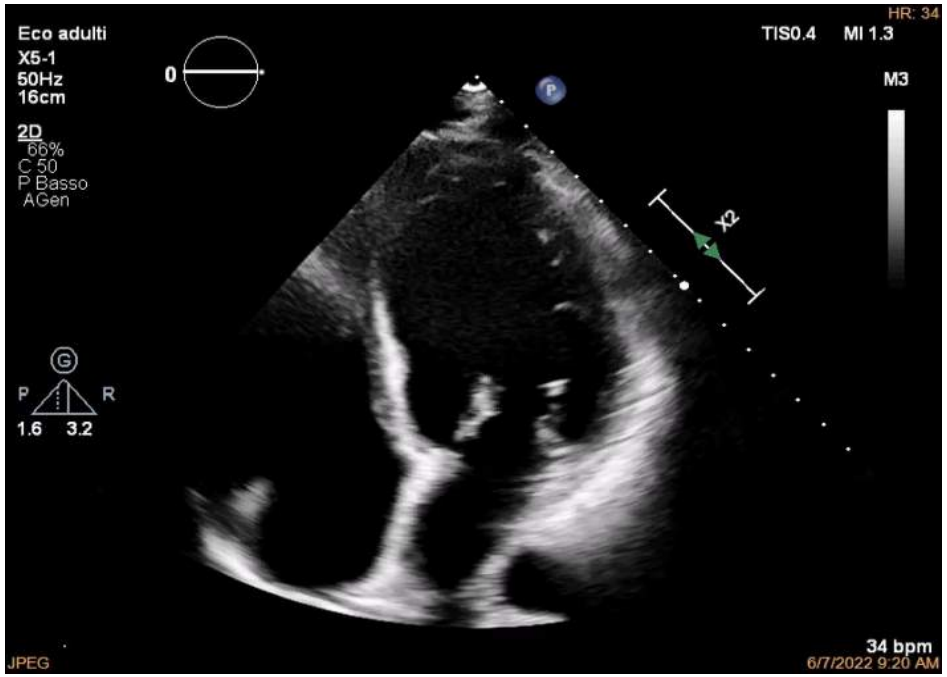


PaceMapping LV

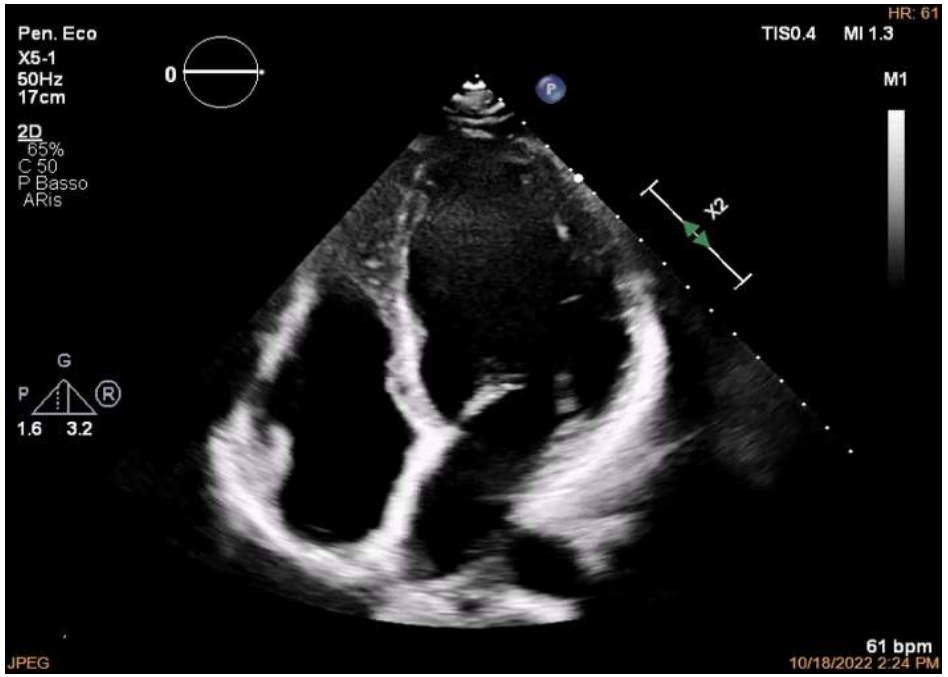
TS, 49 a, f



Pre ablazione



Post ablazione



TS, 49 a, f

Aritmie ventricolari ad origine dal VD: ablazione transcatetere

Multicenter Outcomes for Catheter Ablation of Idiopathic Premature Ventricular Complexes

Rakesh Latchamsetty, MD,* Miki Yokokawa, MD,* Fred Morady, MD,* Hyungjin Myra Kim, ScD,† Shibu Mathew, MD,† Roland Tilz, MD,† Karl-Heinz Kuck, MD,† Koichi Nagashima, MD, PhD,§ Usha Tedrow, MD,§ William Gregory Stevenson, MD,§ Ricky Yu, MD,‖ Roderick Tung, MD,‖ Kalyanam Shivkumar, MD,‖ Jean-Francois Sarrazin, MD,¶ Arash Arya, MD,# Gerhard Hindricks, MD,# Rama Vunnam, MD,** Timm Dickfeld, MD,** Emile G. Daoud, MD,†† Nishaki M. Oza, MD,†† Frank Bogun, MD*

OBJECTIVES This study reports multicenter outcomes and complications for catheter ablation of premature ventricular complexes (PVCs) and investigates predictors of procedural success, as well as development of PVC-induced cardiomyopathy.

BACKGROUND Catheter ablation of frequent idiopathic PVCs is used to eliminate symptoms and treat PVC-induced cardiomyopathy. Large-scale multicenter outcomes and complication rates have not been reported.

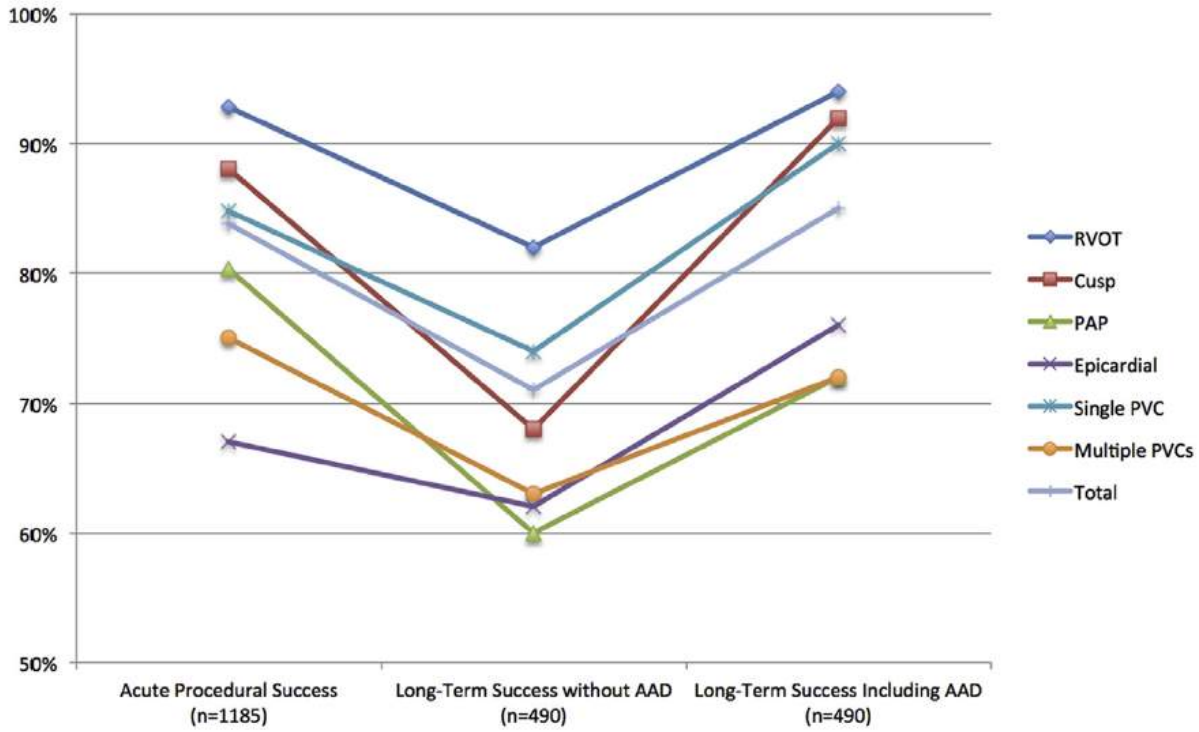
METHODS This retrospective cohort study included 1,185 patients (55% female; mean age 52 ± 15 years; mean ejection fraction 55 ± 10%; mean PVC burden 20 ± 13%) who underwent catheter ablation for idiopathic PVCs at 8 centers between 2004 and 2013. The following factors were evaluated: patient demographics, procedural characteristics, complication rates, and clinical outcomes.

RESULTS Acute procedural success was achieved in 84% of patients. In centers at which patients were followed up routinely with post-ablation Holter monitoring, continued success at clinical follow-up without use of antiarrhythmic drugs was 71%. Including the use of antiarrhythmic medications, the success rate at a mean of 1.9 years of follow-up was 85%. In a multivariate analysis, the significant predictors of acute success were PVC location and number of distinct PVC configurations ($p < 0.03$). The only significant predictor of continued success at clinical follow-up was a right ventricular outflow tract PVC location ($p < 0.01$). In 245 patients (21%) with PVC-induced cardiomyopathy, the mean ejection fraction improved from 38% to 50% ($p < 0.01$) after ablation. Independent predictors for development of PVC-induced cardiomyopathy were male gender, PVC burden, lack of symptoms, and epicardial PVC origin ($p < 0.05$). The overall complication rate was 5.2% (2.4% major complications and 2.8% minor complications), and complications were most commonly related to vascular access (2.8%). There was no procedure-related mortality.

CONCLUSIONS Catheter ablation of frequent PVCs is a low-risk and often effective treatment strategy to eliminate PVCs and associated symptoms. In patients with PVC-induced cardiomyopathy, cardiac function is frequently restored after successful ablation. (J Am Coll Cardiol EP 2015;1:116-23) © 2015 by the American College of Cardiology Foundation.

TABLE 2 Procedure Times

PVC Location	RVOT	Cusp	PAP	Epicardial	Single PVC	Multiple PVCs	Total	p Value
Procedure duration, min	157 ± 97	178 ± 96	294 ± 109	249 ± 117	180 ± 107	278 ± 125	198 ± 115	<0.001*
Fluoroscopy time, min	20 ± 17	27 ± 21	40 ± 21	48 ± 27	27 ± 23	44 ± 27	30 ± 24	<0.001†
Radiofrequency time, min	9 ± 9	11 ± 7	26 ± 19	13 ± 10	10 ± 8	20 ± 16	12 ± 11	<0.001‡



Aritmie ventricolari ad origine dal VD: ablazione transcatetere

ESC European Society of Cardiology
European Heart Journal (2022) 43, 3997–4126
https://doi.org/10.1093/eurheartj/ehac262
ESC GUIDELINES

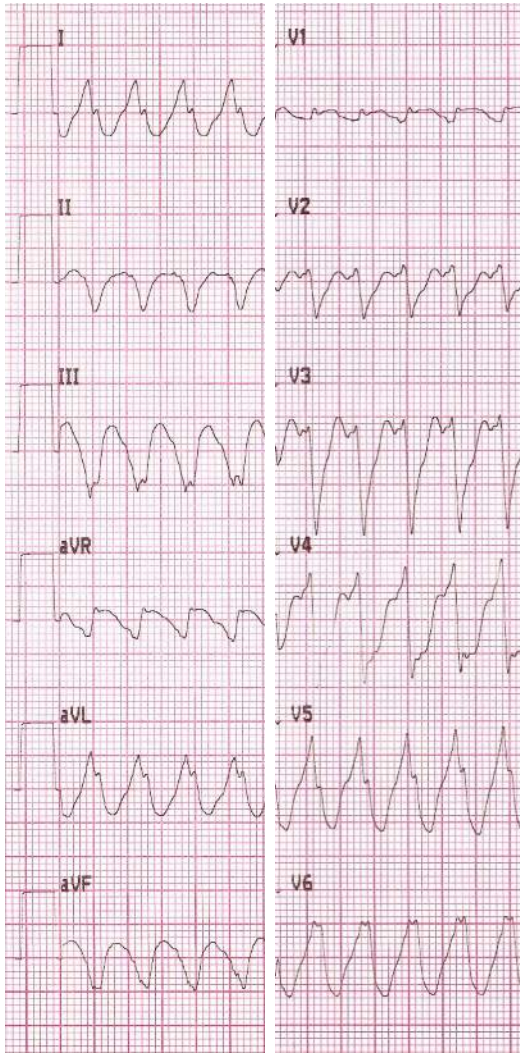
2022 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death

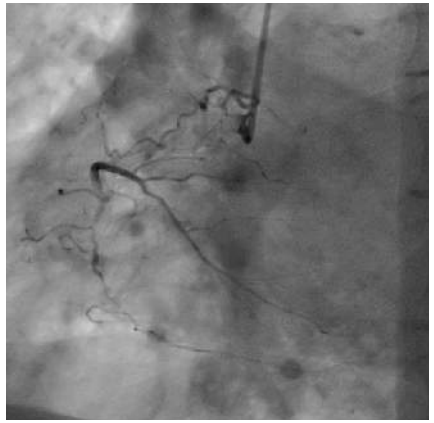
Developed by the task force for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death of the European Society of Cardiology (ESC)

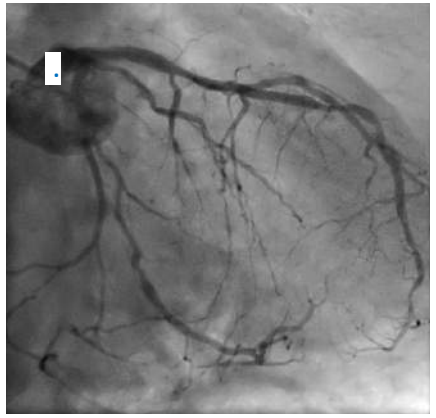
	Ablation	Beta-blocker	CCB	Flecainide	Amiodarone
RVOT/fascicular PVC/VT: Symptomatic, normal LV function	Class I	Class IIa	Class IIa	Class IIa	Class III
PVC/VT other than RVOT/fascicular: Symptomatic, normal LV function	Class IIa	Class I	Class I	Class IIa	Class III
RVOT/fascicular PVC/VT: LV dysfunction	Class I	Class IIa	Class III[a]	Class IIa[b]	Class IIa
PVC/VT other than RVOT/fascicular: LV dysfunction	Class I	Class IIa	Class III[a]	Class IIa[b]	Class IIa
PVC: Burden >20%, asymptomatic, normal LV function	Class IIb				Class III

Catheter ablation of structural RV arrhythmias: post-AMI

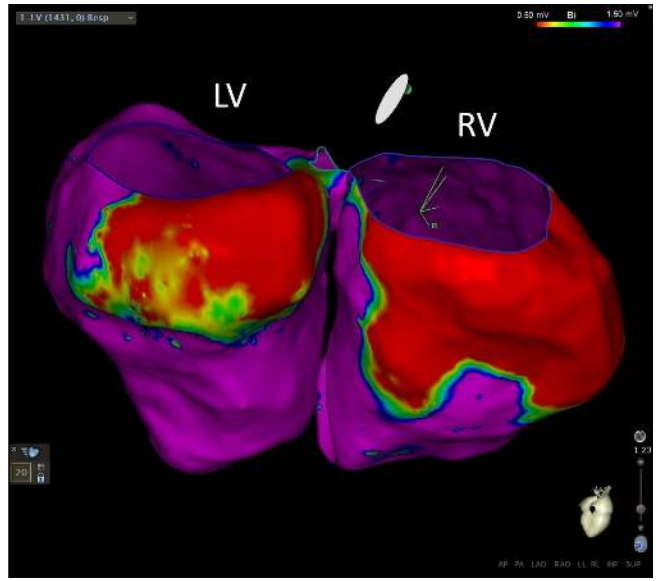
Post RV AMI VT



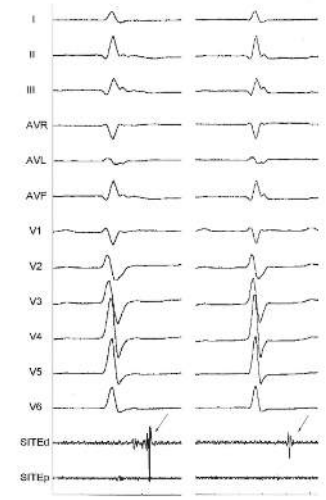




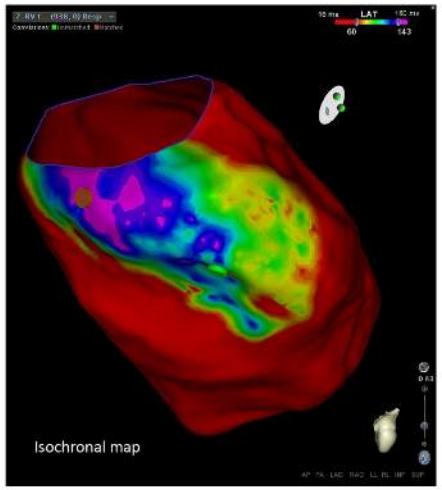
Time	Events
25 years before presentation	Acute, ST elevation, infero-posterolateral myocardial infarction treated with thrombolysis i.v.
At presentation	Emergency Department admission for haemodynamically stable (arterial blood pressure: 90/60 mmHg; O^2 saturation: 94%) sustained ventricular tachycardia (VT) at 210 b.p.m.
8 days after presentation	Cardioverter/defibrillator implantation
11 days after presentation	Electrical storm (three effective cardioverter/defibrillator shocks, 10 ineffective, and one effective antitachycardia pacing attempt) due to recurrent episodes of the clinical VT. Amiodarone administration (i.v.) started.



A

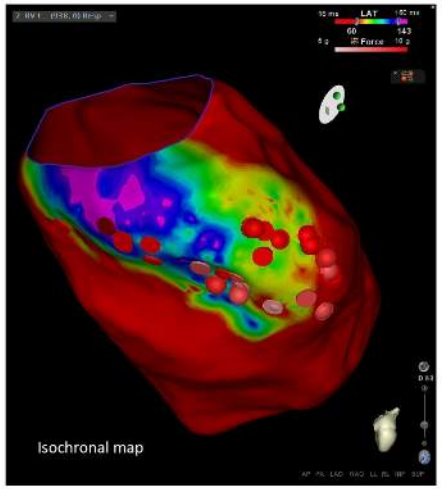


B



C

D

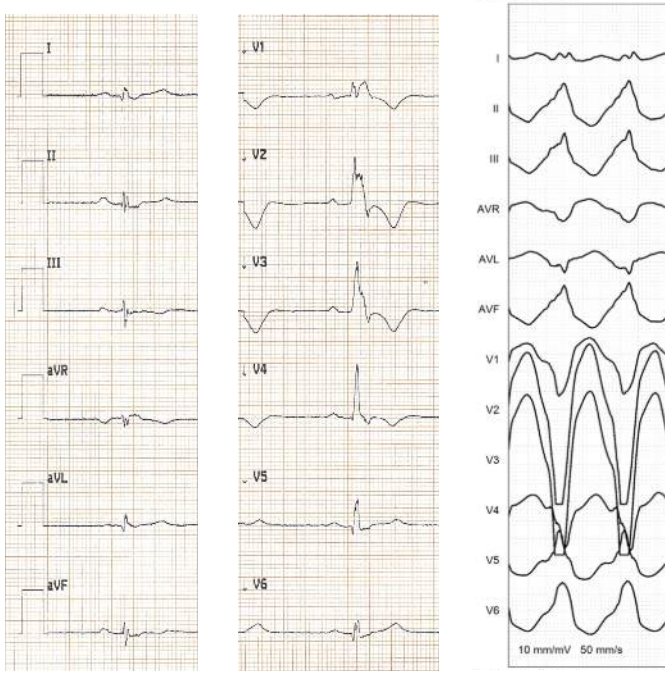


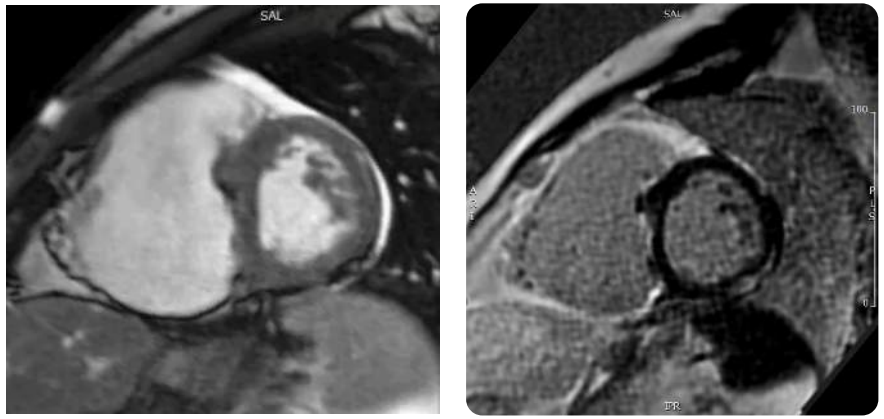
M. Tritto et al Eur Heart J CR 2019 doi: 10.1093/ehjcr/ytz067

Arrhythmogenic (RV) CM

- Inherited disease mostly linked to desmosomal mutations
- Prevalence: 1/1000 – 1/5000
- Males > females
- Fibro-fatty replacement (right ventricle, left ventricle or both) moving from the epicardium to the endocardium
- Distinctive ECG and MRI features
- Arrhythmic presentation (LBBB pattern)
  - ☑ SCD: 4.6 – 6.1%
  - ☑ SM VT: 23% (8-11 yr FU)

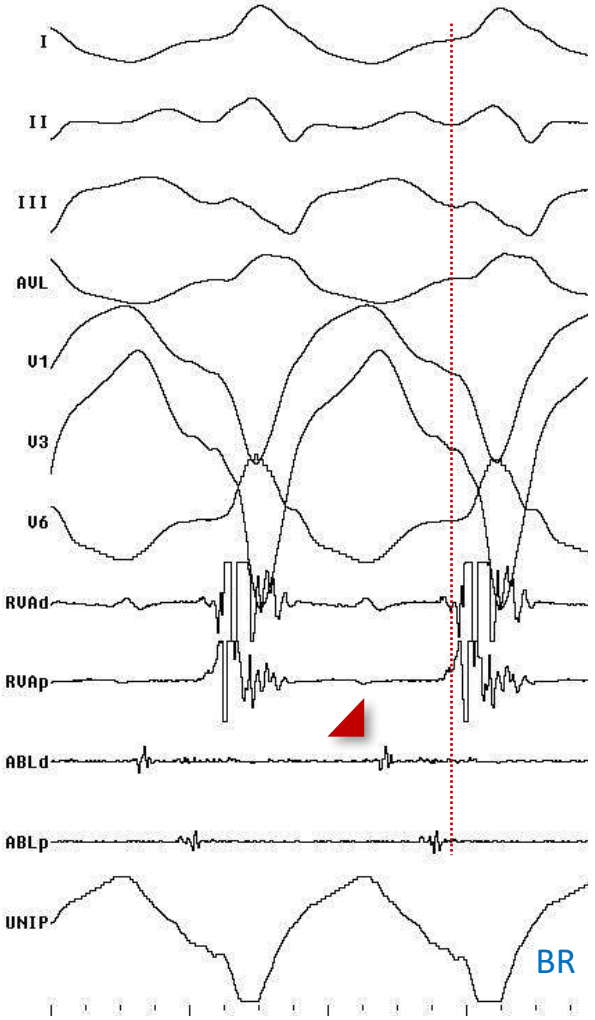
Gene	Encoded Protein	Subcellular Localization
JUP	Junction plakoglobin	Desmosome
DSP	Desmoplakin	Desmosome
PKP2	Plakophilin-2	Desmosome
DSG2	Desmoglein-2	Desmosome
DSC2	Desmocollin-2	Desmosome
TMEM43	Transmembrane protein 43 (luma)	Nuclear envelope
LMNA	Lamin A/C	Nuclear envelope
DES	Desmin	Intermediate filament
CTNNA3	Alpha-T-catenin	Area composita
PLN	Phospholamban	SERCA
TGFB3	Transforming growth factor-3	Growth factor
TTN	Titin	Sarcomere
SCN5A	Sodium voltage-gated channel alpha subunit 5 ($Na_v1.5$)	Sodium channel
CDH2	Cadherin C	Area composita



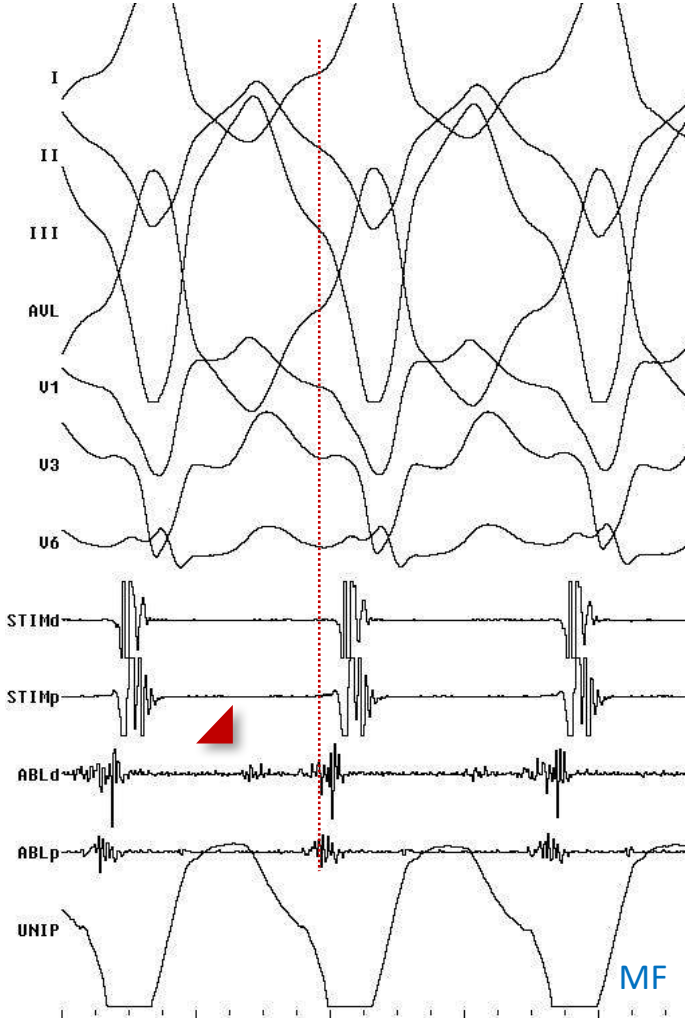


Catheter ablation of structural RV arrhythmias: ARVC

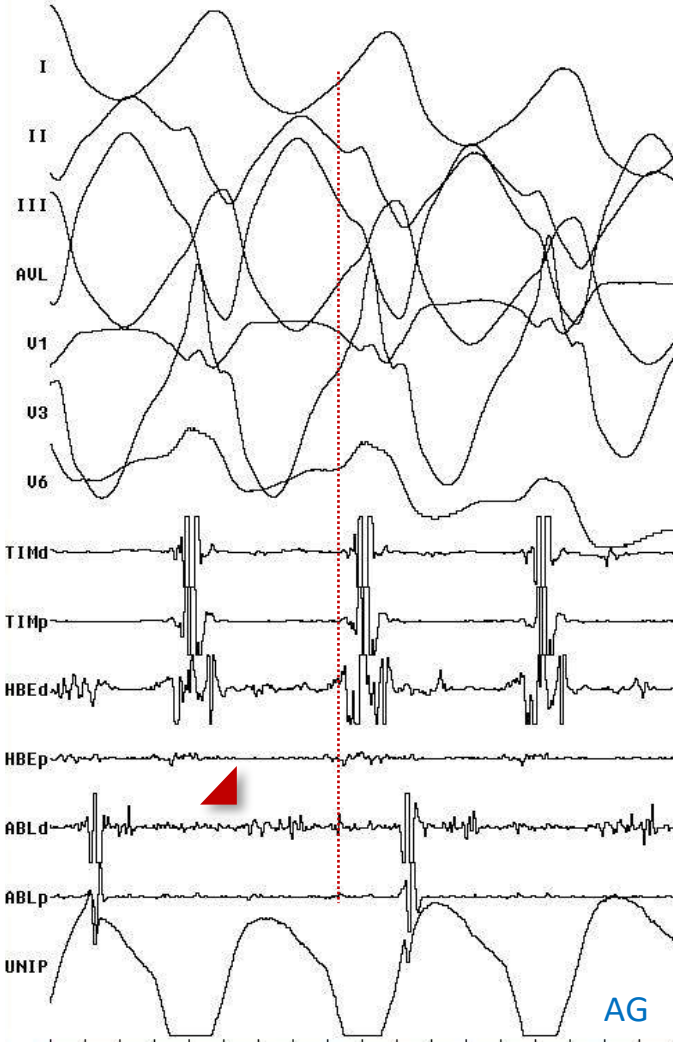
Isolated potential



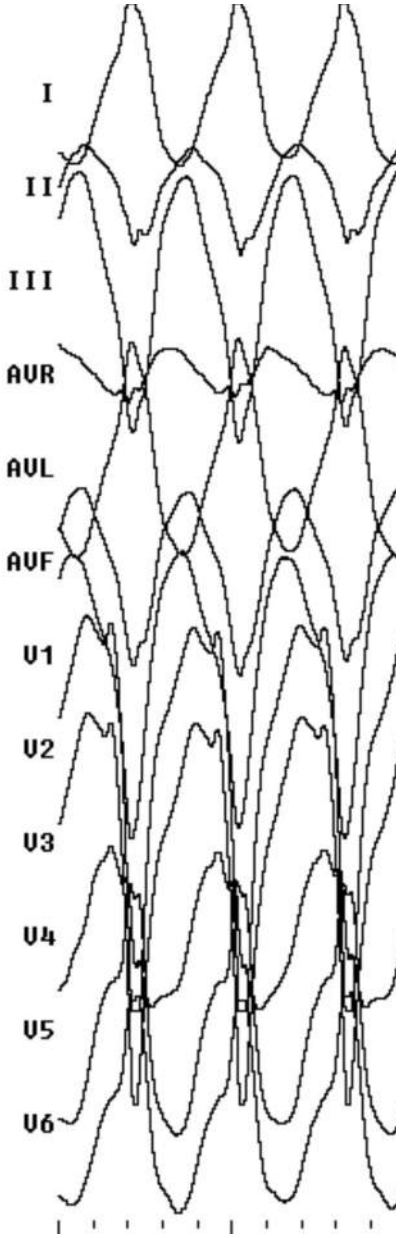
Fragmented potential

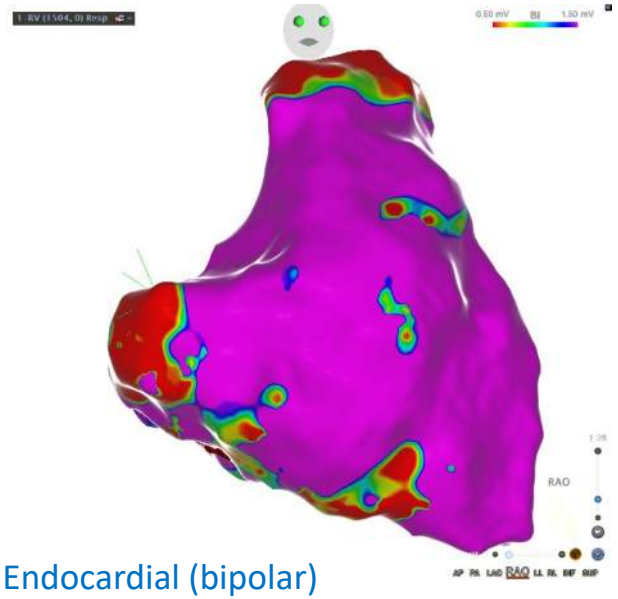


Continuous electrical activity

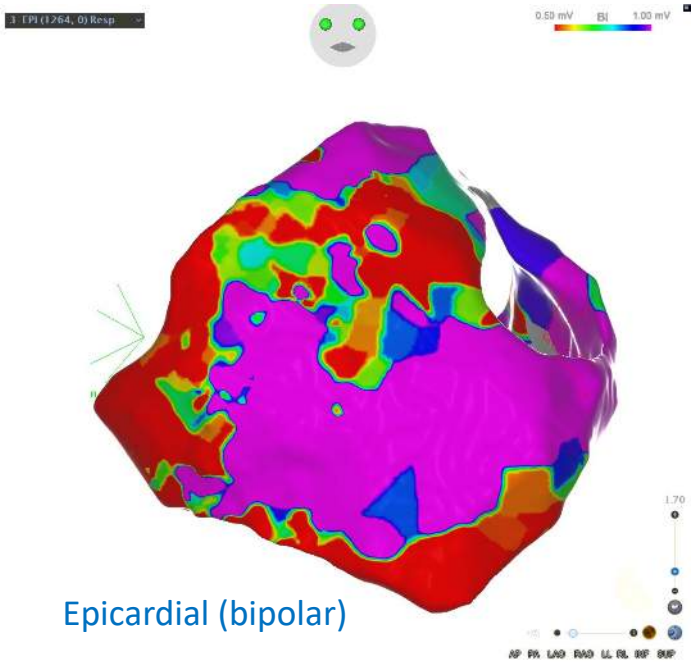


FP, m, 46 yrs, ARVC (DSG2 mutation)

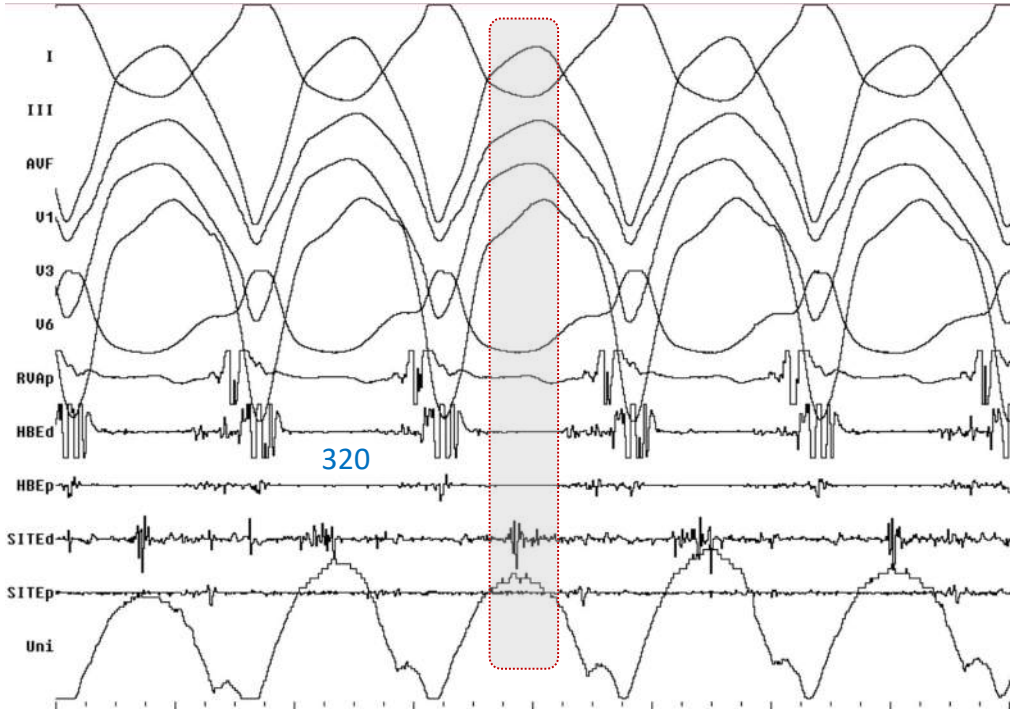


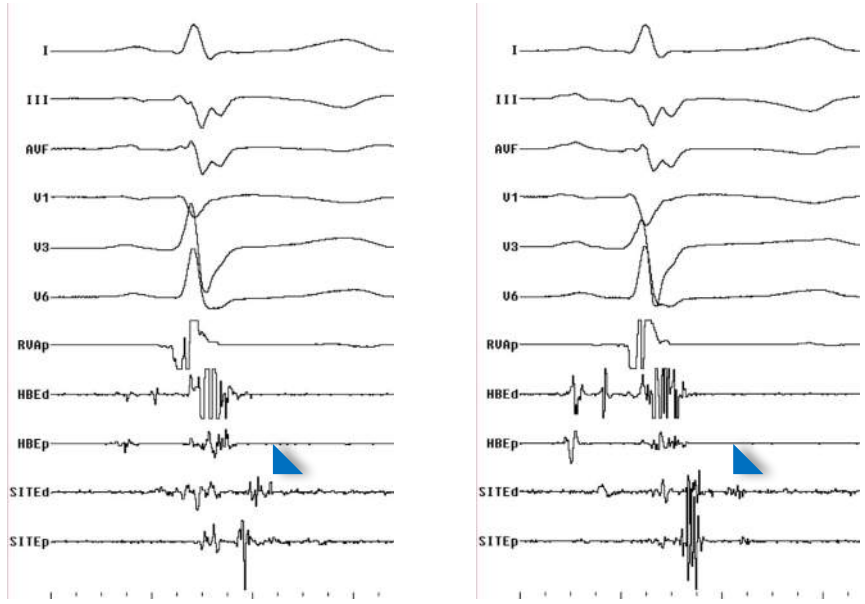


Endocardial (bipolar)



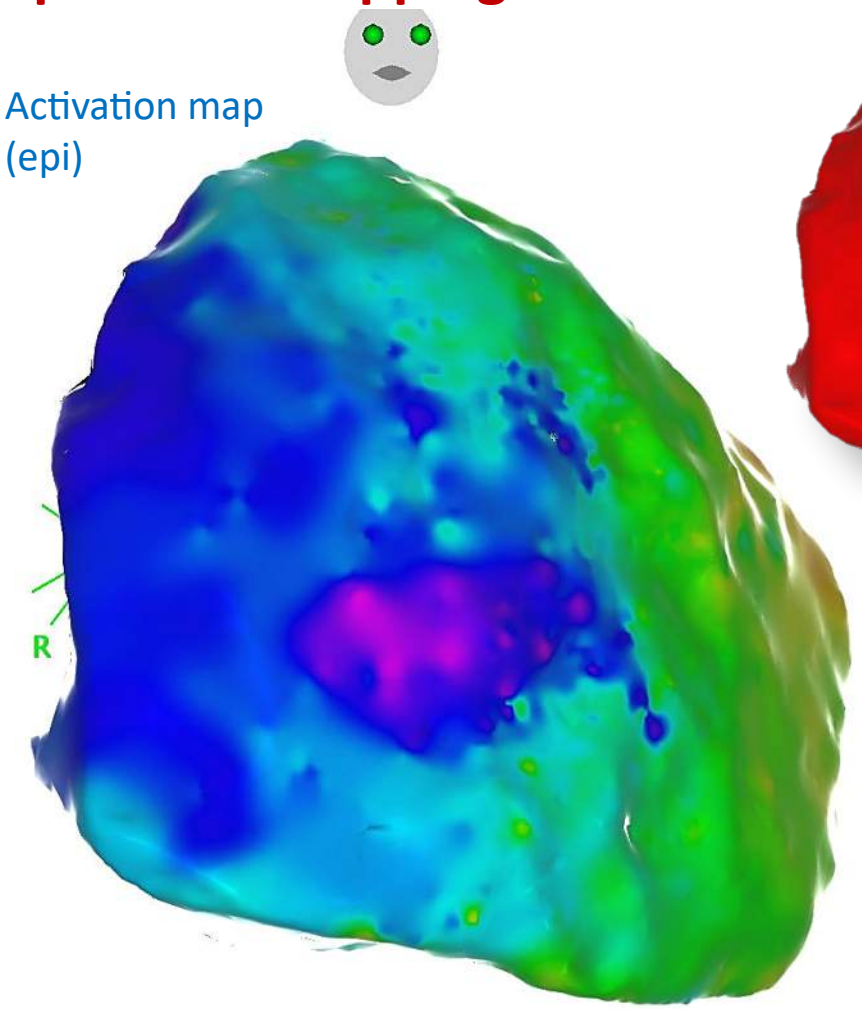
Epicardial (bipolar)

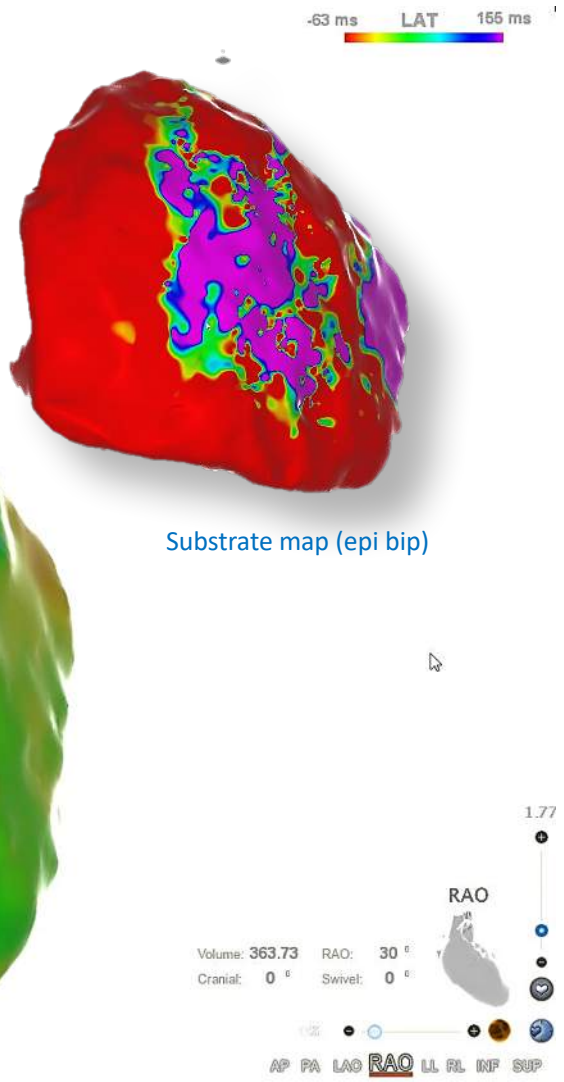




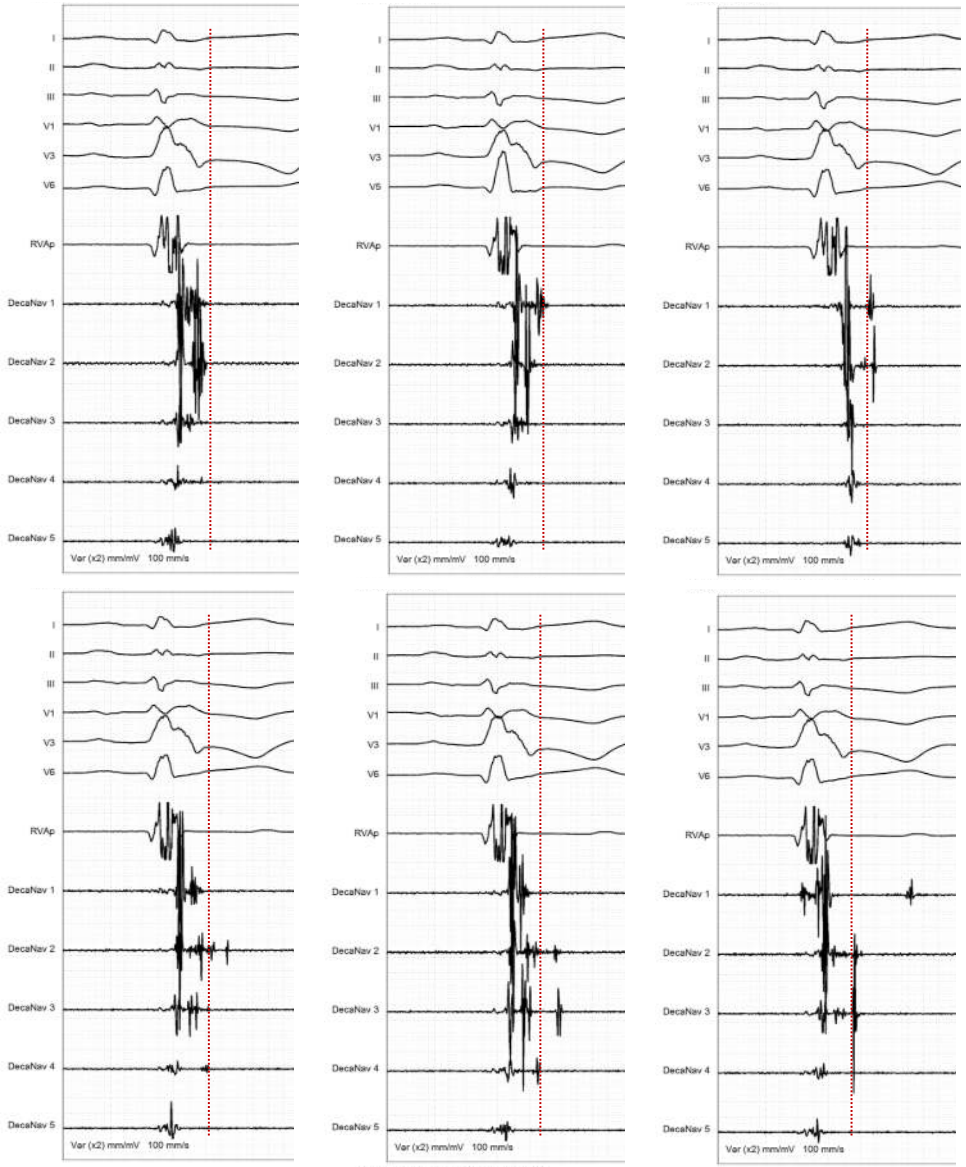
Epicardial mapping

Activation map (epi)





Substrate map (epi bip)



Circulation
Arrhythmia and Electrophysiology
JOURNAL OF THE AMERICAN HEART ASSOCIATION
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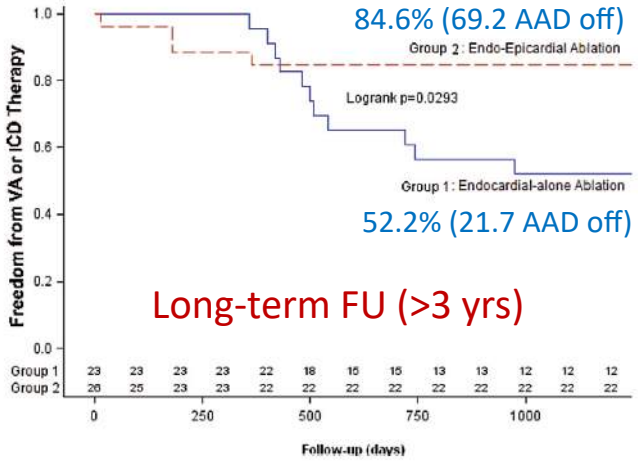
Ablation of Ventricular Arrhythmias in Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy : Arrhythmia-Free Survival After Endo-Epicardial Substrate Based Mapping and Ablation

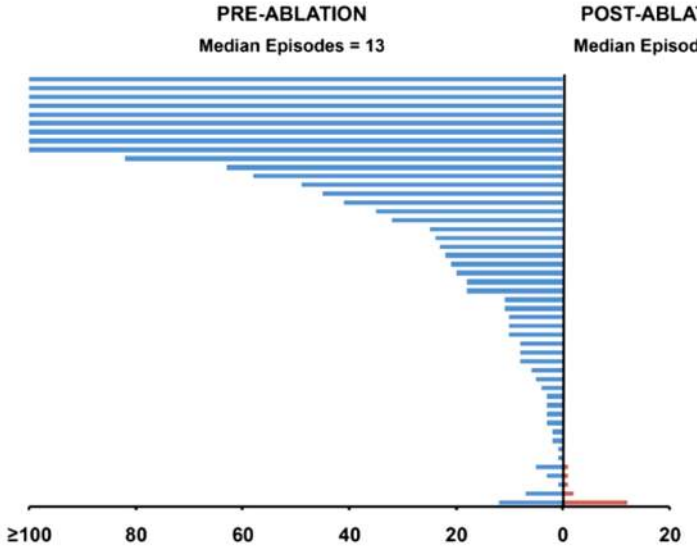
Rong Bai, Luigi Di Biase, Kalyanam Shivkumar, Prasant Mohanty, Roderick Tung, Pasquale Santangeli, Luis Carlos Saenz, Miguel Vacca, Atul Verma, Yariv Khaykin, Sanghamitra Mohanty, J. David Burkhardt, Richard Hongo, Salwa Beheiry, Antonio Dello Russo, Michela Casella, Gemma Pelargonio, Pietro Santarelli, Javier Sanchez, Claudio Tondo and Andrea Natale

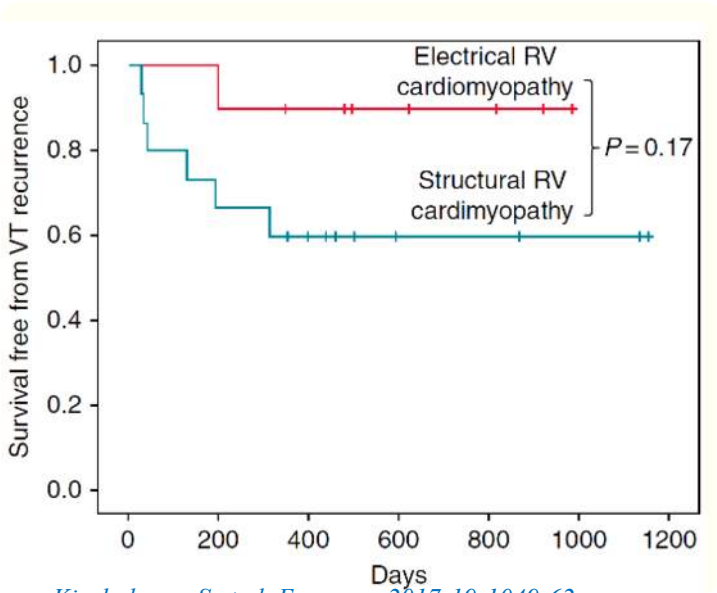
Circ Arrhythm Electrophysiol 2011;4:478-485; originally published online June 10, 2011;

Results of VT catheter ablation in ARVC (endo/epi)

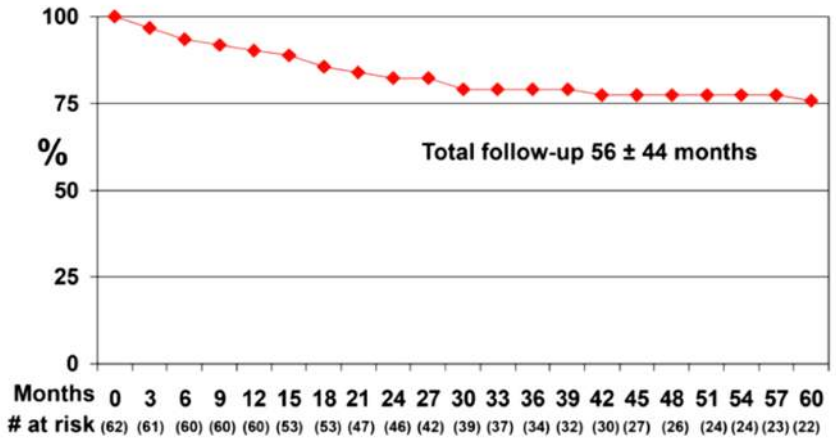


Long-term FU (>3 yrs)





Kirubakaran S et al. Europace 2017;19:1049-62



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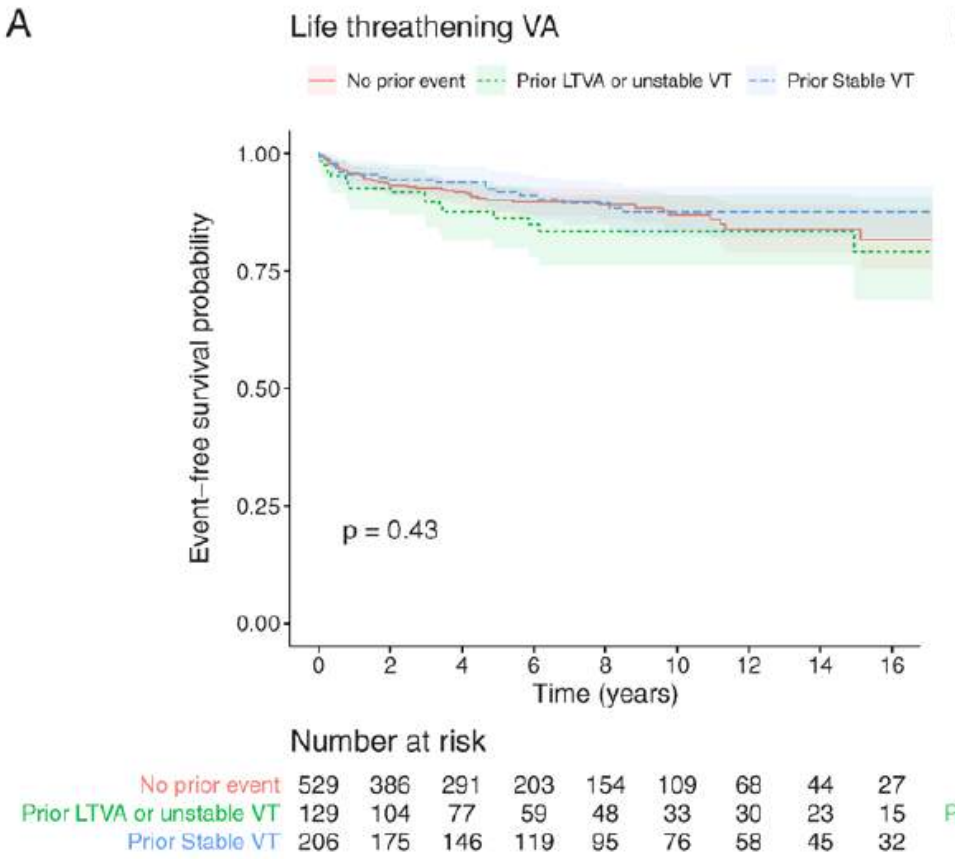
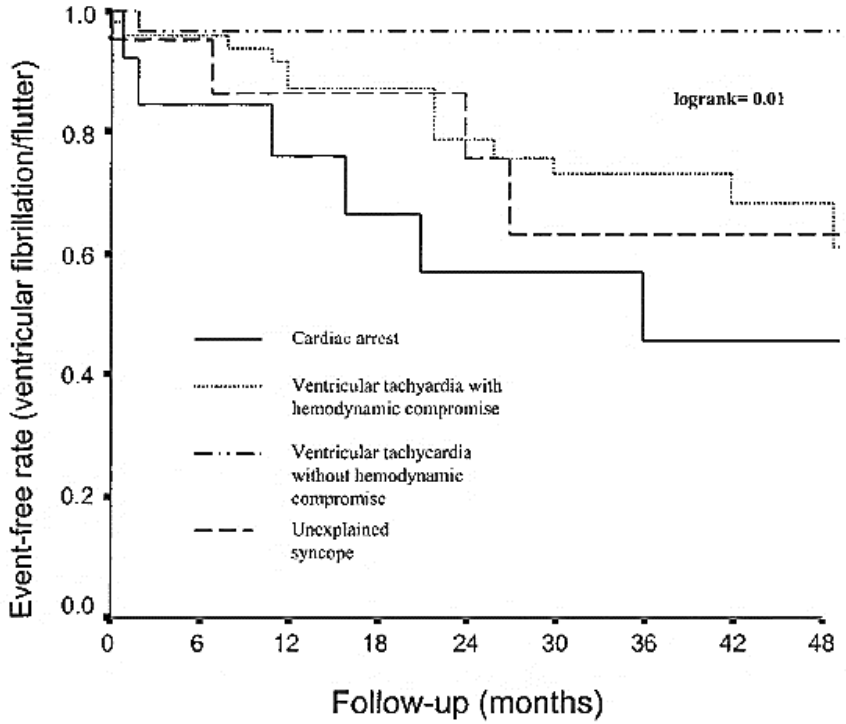
Risk of VFl/VF in ARVC pts implanted with an ICD



Cadrin-Tourigny J et al. Circ AEP 2021;14:e008509.



Corrado D et al. Circulation 2003;108:3084-91

Catheter ablation of structural RV arrhythmias: ARVC

Circulation: Arrhythmia and Electrophysiology

ORIGINAL ARTICLE

Characterization of Structural Changes in Arrhythmogenic Right Ventricular Cardiomyopathy With Recurrent Ventricular Tachycardia After Ablation

Insights From Repeat Electroanatomic Voltage Mapping

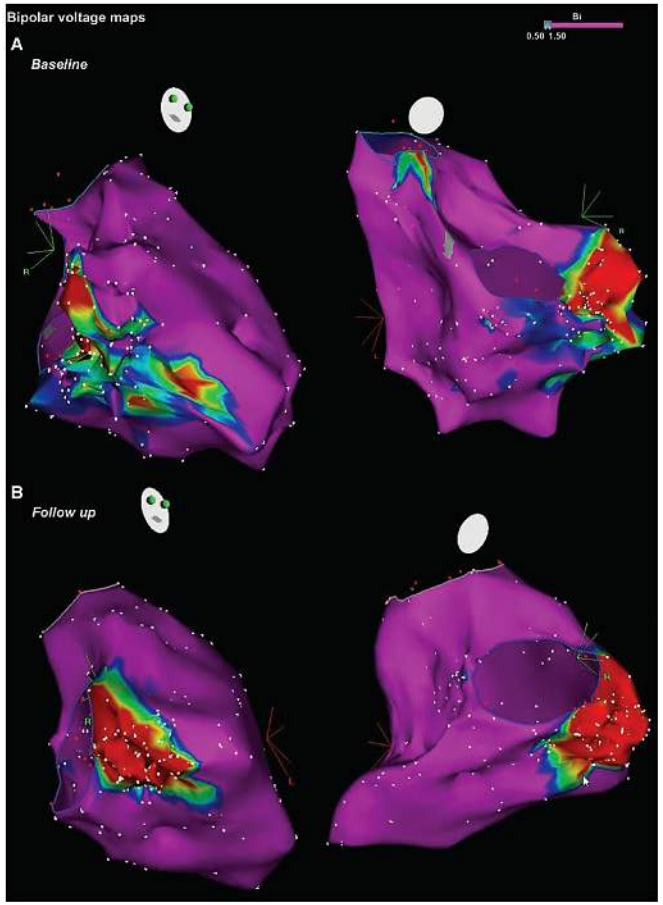
David F. Briceño, MD; Jackson J. Liang, DO; Yasuhiro Shirai, MD; Timothy M. Markman, MD; Anwar Chahal, MD, PhD; Cory Tschabrunn, PhD; Erica Zado, PA-C; Mathew C. Hyman, MD, PhD; Ramanan Kumareswaran, MD; Jeffery S. Arkles, MD; Pasquale Santangeli, MD, PhD; Robert D. Schaller, DO; Gregory E. Supple, MD; David S. Frankel, MD; Rajat Deo, MD, PhD; Michael P. Riley, MD, PhD; Saman Nazarian, MD, PhD; David Lin, DO; Andrew E. Epstein, MD; Fermin C. Garcia, MD; Sanjay Dixit, MD; David J. Callans, MD; Francis E. Marchlinski, MD

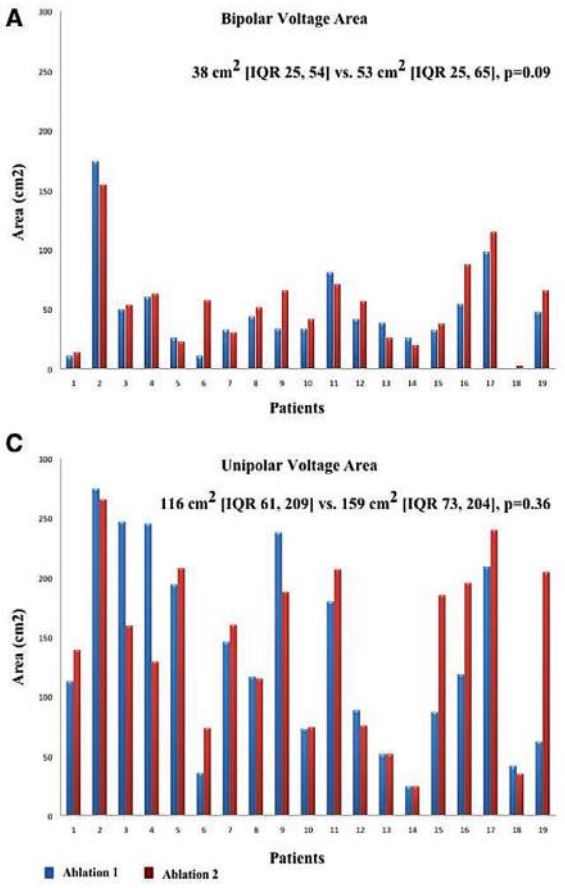
BACKGROUND: Data characterizing structural changes of arrhythmogenic right ventricular (RV) cardiomyopathy are limited.

METHODS: Patients presenting with left bundle branch block ventricular tachycardia in the setting of arrhythmogenic RV cardiomyopathy with procedures separated by at least 9 months were included.

RESULTS: Nineteen consecutive patients (84% males; mean age 39±15 years [range, 20–76 years]) were included. All 19 patients underwent 2 detailed sinus rhythm electroanatomic endocardial voltage maps (average 385±177 points per map; range, 93–847 points). Time interval between the initial and repeat ablation procedures was mean 50±37 months (range, 9–162). No significant progression of voltage was observed (bipolar: 38 cm^2 [interquartile range (IQR), 25–54] versus 53 cm^2 [IQR, 25–65], P=0.09; unipolar: 116 cm^2 [IQR, 61–209] versus 159 cm^2 [IQR, 73–204], P=0.36) for the entire study group. There was a significant increase in RV volumes (percentage increase, 28%; 206 mL [IQR, 170–253] versus 263 mL [IQR, 204–294], P<0.001) for the entire study population. Larger scars at baseline but not changes over time were associated with a significant increase in RV volume (bipolar: Spearman ρ, 0.6965, P=0.006; unipolar: Spearman ρ, 0.5743, P=0.03). Most patients with progressive RV dilatation (8/14, 57%) had moderate (2 patients) or severe (6 patients) tricuspid regurgitation recorded at either initial or repeat ablation procedure.

CONCLUSIONS: In patients with arrhythmogenic RV cardiomyopathy presenting with recurrent ventricular tachycardia, >10% increase in RV endocardial surface area of bipolar voltage consistent with scar is uncommon during the intermediate term. Most recurrent ventricular tachycardias are localized to regions of prior defined scar. Voltage indexed scar area at baseline but not changes in scar over time is associated with progressive increase in RV size and is consistent with adverse remodeling but not scar progression. Marked tricuspid regurgitation is frequently present in patients with arrhythmogenic RV cardiomyopathy who have progressive RV dilation.





Circ Arrhythm Electrophysiol. 2020;13:e007611. DOI: 10.1161/CIRCEP.119.007611

Outcome of ARVC pts not implanted with an ICD after VT catheter ablation

	# Pts	Epi CA	Abl Success	FU	# Deaths
Santangeli P[1]	32	72%	81%	46 [26-65]	None
Gandjbakhch E[2]	65	29%	72%	52 [12-171]	None
Pers experience	6	50%	83%	137 [85-200]	None

1. Santangeli P et al. JACC EP 2019;5:55-65
2. Gandjbakhch E et al. Europace 2021;23:1428-36