Mapping & Ablation of VT in Non Ischemic Cardiomyopathy

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Disclosures:

- Research grant from
- Biosense webster

- Medtronic ,
- St Jude medical

Agenda

- VT : cause or the effect
- mechanism & substrate for VT
- Mapping & Catheter Ablation
- Case examples

Reversal of Cardiomyopathy in Patients With Repetitive Monomorphic Ventricular Ectopy Originating From the Right Ventricular Outflow Tract

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Background—Tachycardia-induced cardiomyopathy caused by ventricular tachycardia is a well-defined clinical entity. Less well appreciated is whether simple ventricular ectopy can result in cardiomyopathy. We sought to examine a potential causal relationship between repetitive monomorphic ventricular ectopy originating from the right ventricular outflow tract and cardiomyopathy and the role of ablation in reversing this process.

Methods and Results—The study consisted of 27 patients (11 men; age, 47±15 years) with repetitive monomorphic ventricular ectopy, including 8 patients (30%) with depressed ventricular function (ejection fraction ≤45%). All patients underwent assessment of cardiac structure and function. The burden of ectopy was quantified through 24-hour Holter monitoring. Patients then underwent ablation guided by 3D mapping. After ablation, patients underwent repeated Holter monitoring and reassessment of cardiac function. Patients with depressed ventricular function were more likely to be older than patients with normal function (58±14 versus 42±18 years; P=0.013). However, the burden of ventricular ectopy was similar in patients with (17 859±13 488 ectopic beats per 24 hours) and without (17 541±11 479 ectopic beats per 24 hours; P=0.800) preserved ventricular function. Successful ablation was performed in 23 patients (85%), including 7 of 8 patients with depressed ventricular function. In this latter group, ventricular function improved in all patients (from 39±6% to 62±6%; P=0.017).

Conclusions—Repetitive monomorphic ventricular ectopy (in the absence of sustained ventricular tachycardia) originating from the right ventricular outflow tract is an underappreciated cause of unexplained cardiomyopathy. Successful ablation of the focal source of ventricular ectopy results in normalization of left ventricular function. Patients with ectopy-induced cardiomyopathy are significantly older than patients with preserved ventricular function, which suggests either that older patients are more susceptible to the development of a cardiomyopathy or that the cardiomyopathy has had a longer period of time in which to evolve. (***Circulation*. 2005;112:1092-1097.**)

Key Words: ablation ■ cardiomyopathy ■ ventricular premature complexes

Reversal of Cardiomyopathy in Patients With Repetitive Monomorphic Ventricular Ectopy Originating From the Right Ventricular Outflow Tract

BLE 2. Patients With Repetitive Monomorphic Ventricular Ectopy and Depressed Left Ventricular Function

ient	Age, y	Sex	Presenting Symptom	Origin of CMP	Cardiac Medications	PVC Origin in RVOT	RFA Success	Initial Holter, PVCs/24 h	F/U Holter, (PVCs/24 h	Initial EF, %	F/U EF, %
	37	F	Dyspnea	Idiopathic	β-Blockers	Anterior	Yes	5502	44	38	65
	51	M	None	Idiopathic	β-Blockers	Posteroseptal	Yes	26 491	1893	45	60
	80	M	Presyncope	Idiopathic	None	Anteroseptal	Yes	35 664	1100	35	55
	51	M	Palpitations	Idiopathic	None	Anterior	Yes	9791	5	35	60
	71	F	Palpitations	Idiopathic	β-Blockers	Posteroseptal	Yes	23 352	117	43	65
	62	M	Presyncope	Idiopathic	β-Blockers	Posterolateral	Yes	...*	332	45	65
	47	M	Palpitations	Idiopathic	None	Posterolateral	Yes	16 362	55	45	65
	68	F	Palpitations	Idiopathic	β-Blockers	Posterior	No	5626	12 883	30	35

CMP indicates cardiomyopathy; PVCs, premature ventricular contractions; RFA, radiofrequency ablation; F/U, follow-up; and EF, ejection fraction.
*Patient did not have a 24-hour Holter; however, frequent ambient ectopy was noted on in-patient telemetry.

from the right ventricular outflow tract is an underappreciated cause of unexplained cardiomyopathy. Successful ablation of the focal source of ventricular ectopy results in normalization of left ventricular function. Patients with ectopy-induced cardiomyopathy are significantly older than patients with preserved ventricular function, which suggests either that older patients are more susceptible to the development of a cardiomyopathy or that the cardiomyopathy has had a longer period of time in which to evolve. (*Circulation*. 2005;112:1092-1097.)

Key Words: ablation ■ cardiomyopathy ■ ventricular premature complexes

Relationship between burden of premature ventricular complexes and left ventricular function

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BACKGROUND Frequent idiopathic premature ventricular complexes (PVCs) can result in a reversible form of left ventricular dysfunction. The factors resulting in impaired left ventricular function are unclear. Whether a critical burden of PVCs can result in cardiomyopathy has not been determined.

OBJECTIVE The objective of this study was to determine a cutoff PVC burden that can result in PVC-induced cardiomyopathy.

METHODS In a consecutive group of 174 patients referred for ablation of frequent idiopathic PVCs, the PVC burden was determined by 24-hour Holter monitoring, and transthoracic echocardiograms were used to assess left ventricular function. Receiver-operator characteristic curves were constructed based on the PVC burden and on the presence or absence of reversible left ventricular dysfunction to determine a cutoff PVC burden that is associated with left ventricular dysfunction.

RESULTS A reduced left ventricular ejection fraction (mean 0.37 ± 0.10) was present in 57 of 174 patients (33%). Patients with a decreased ejection fraction had a mean PVC burden of 33% ± 13% as compared with those with normal left ventricular function 13% ± 12% ($P < .0001$). A PVC burden of >24% best separated the patient population with impaired as compared with preserved left ventricular function (sensitivity 79%, specificity 78%, area under curve 0.89) The lowest PVC burden resulting in a reversible cardiomyopathy was 10%. In multivariate analysis, PVC burden (hazard ratio 1.12, 95% confidence interval 1.08 to 1.16; $P < .01$) was independently associated with PVC-induced cardiomyopathy.

CONCLUSION A PVC burden of >24% was independently associated with PVC-induced cardiomyopathy.

KEYWORDS Premature ventricular complexes; Ablation; Cardiomyopathy

ABBREVIATIONS **CI** = confidence interval; **EF** = ejection fraction; **HR** = hazard ratio; **LV** = left ventricular; **PVC** = premature ventricular complexes; **ROC** = receiver operator characteristic; **RVOT** = right ventricular outflow tract

(Heart Rhythm 2010;7:865–869)

Cause vs effect?

- Short history
- Preserved wall thickness
- Absence of LGE on MRI
- High degree of ambient ectopy

Mechanisms of VT in NICM

- (1) scar-based reentry
- (2) abnormal automaticity- Focal, and
- (3) Purkinje system related .

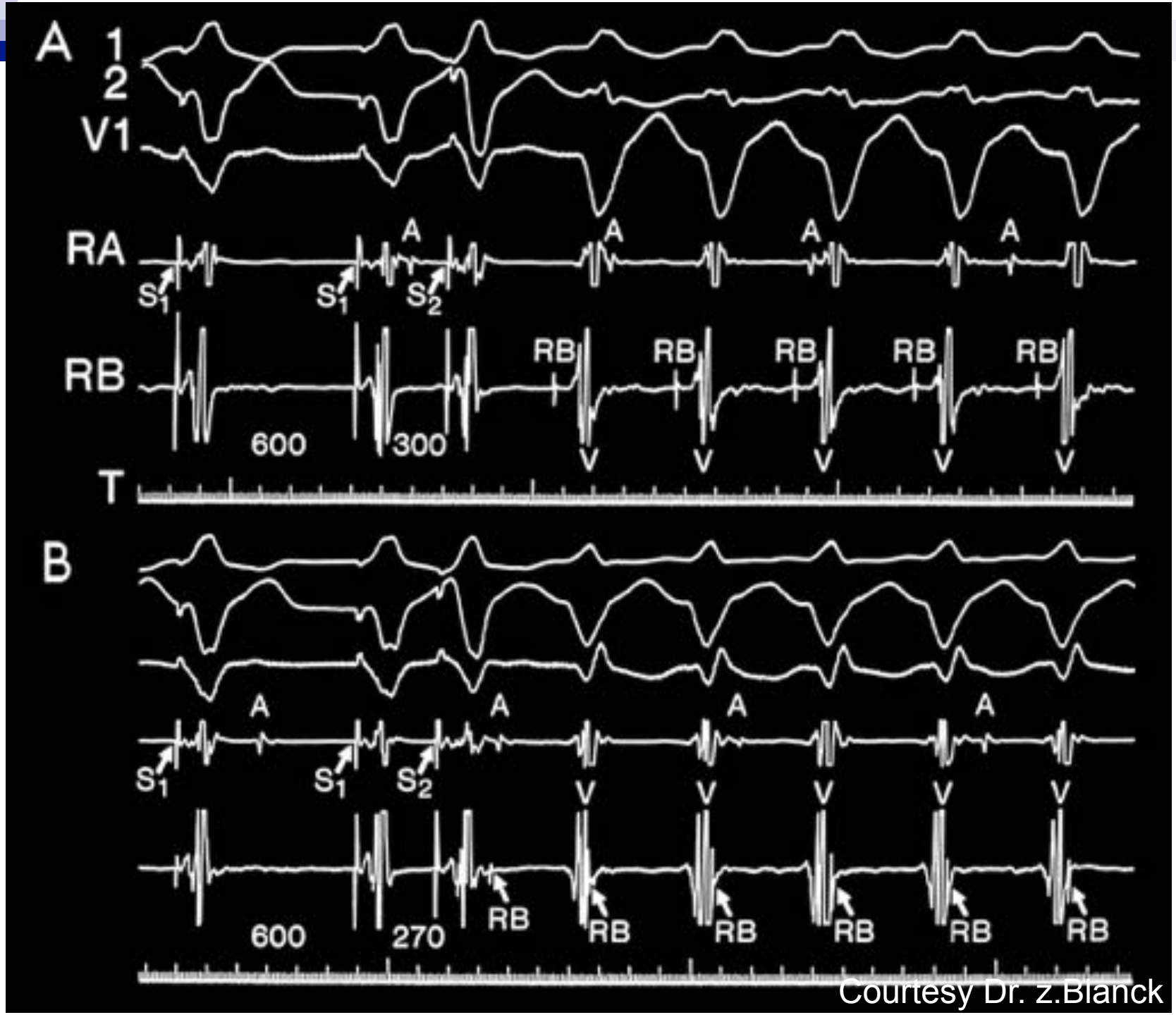
1.Hsia et al Circulation 2003
2.Pogwizd et al. Circulation 1998
3. Blank Z et al JCE 2003

Issues during mapping and Ablation

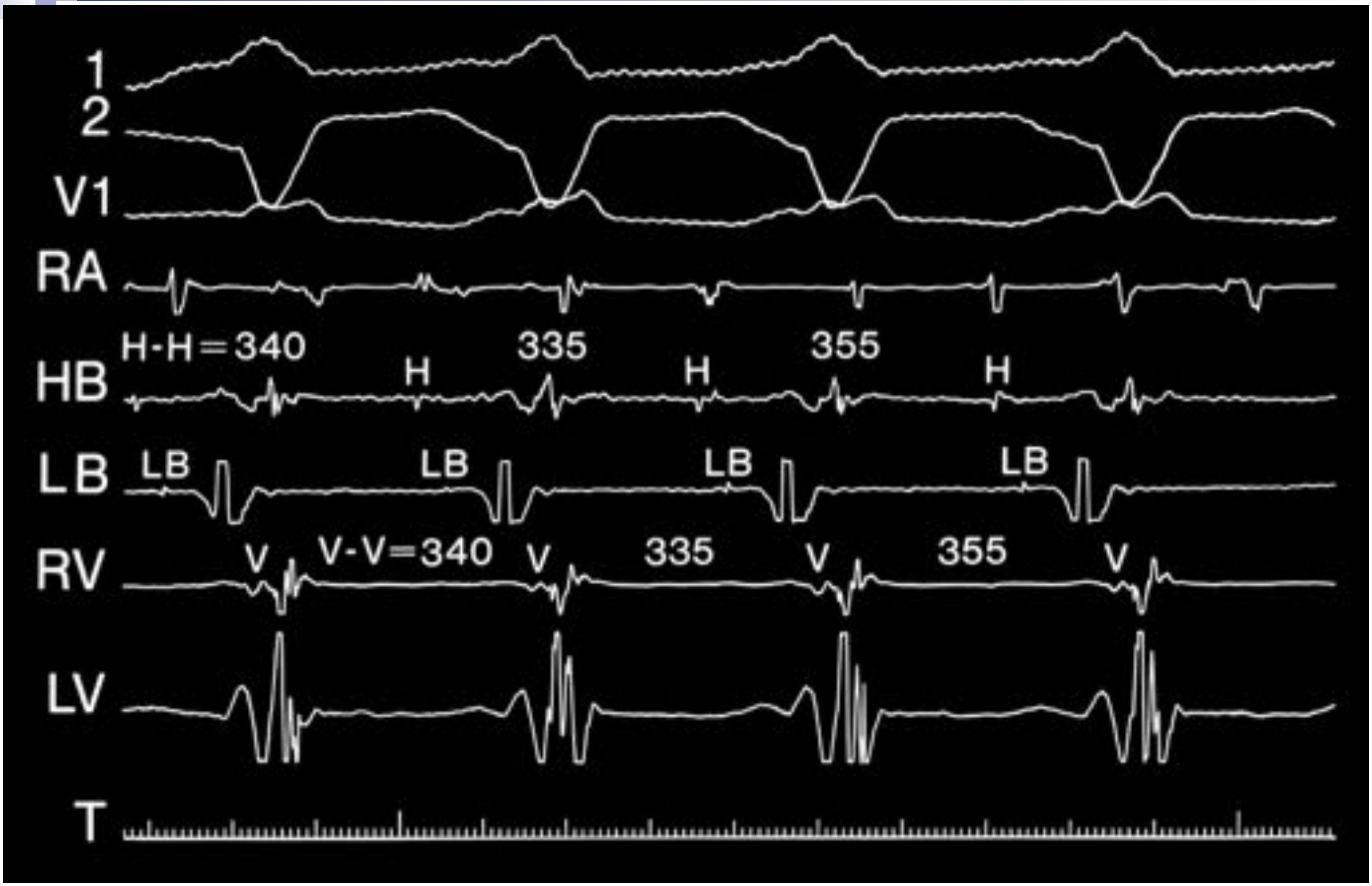
- (1)Clinical VT non inducible
- (2) multiple nontolerated VTs,
- (3) incomplete delineation of substrate,
- (4) possibility of collateral damage
- (5) Mid myocardial substrate

Induction: (pertinent issues)

- Avoid injury to RB
- RV apical pacing / LV
- short-long short sequence
- pts while on AAA: Isuprel
- rapid VA :IV procainamide

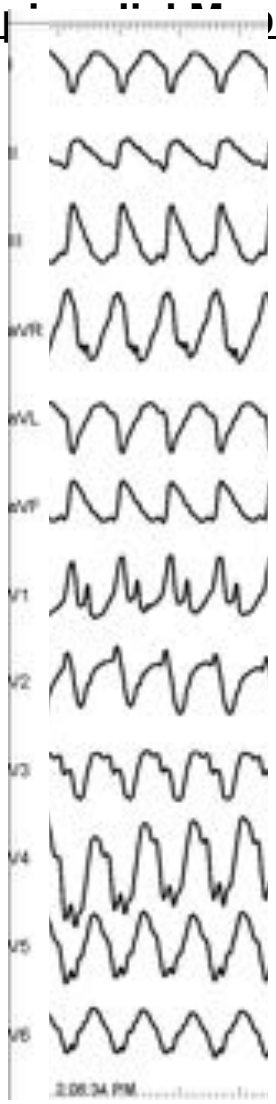


Courtesy Dr. z.Blanck

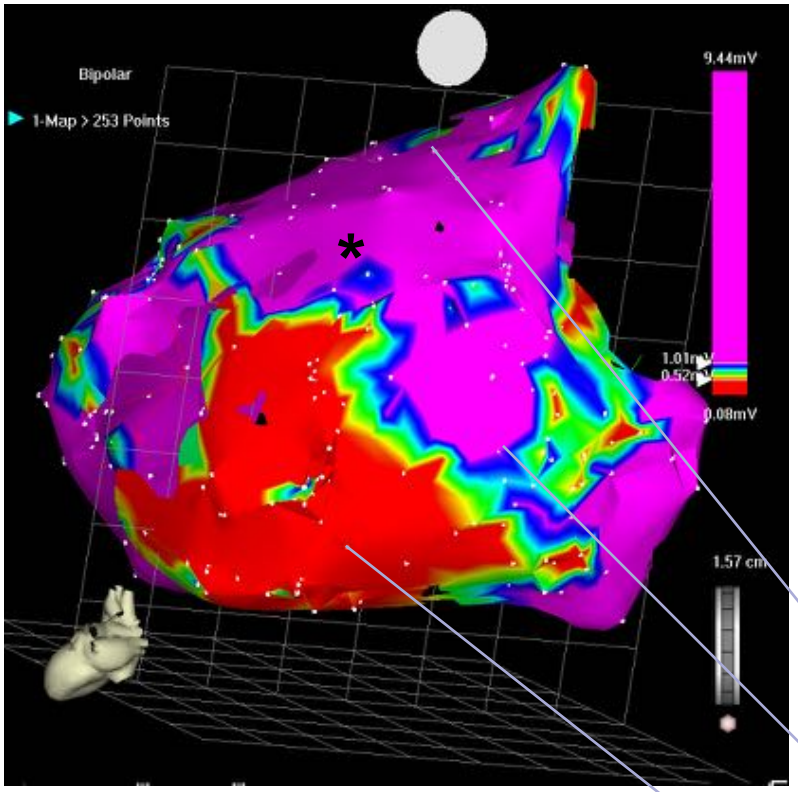


Courtesy Dr. Z..Blanck

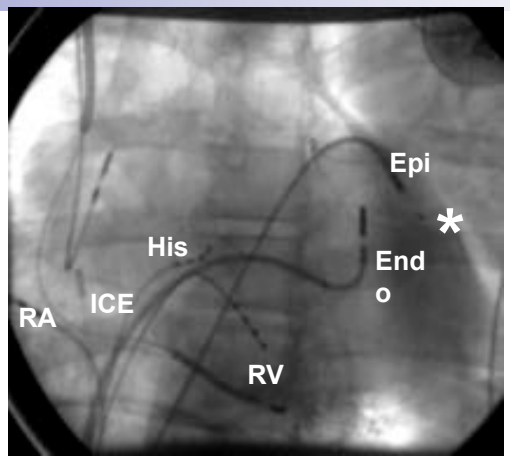
E[illegible]: Non-Ischemic Cardiomyopathy



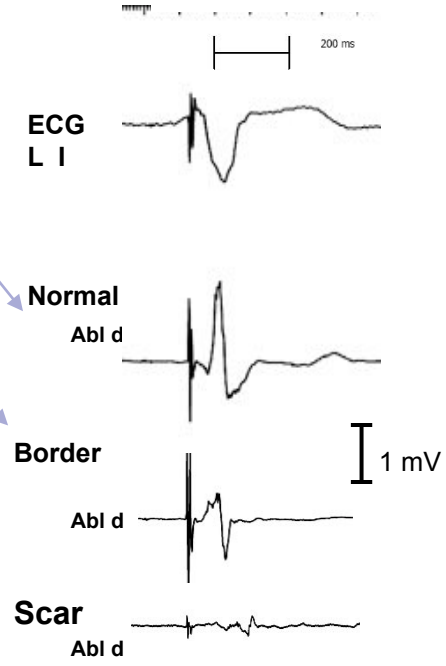
TARGETED VT



EPICARDIAL VOLTAGE MAP



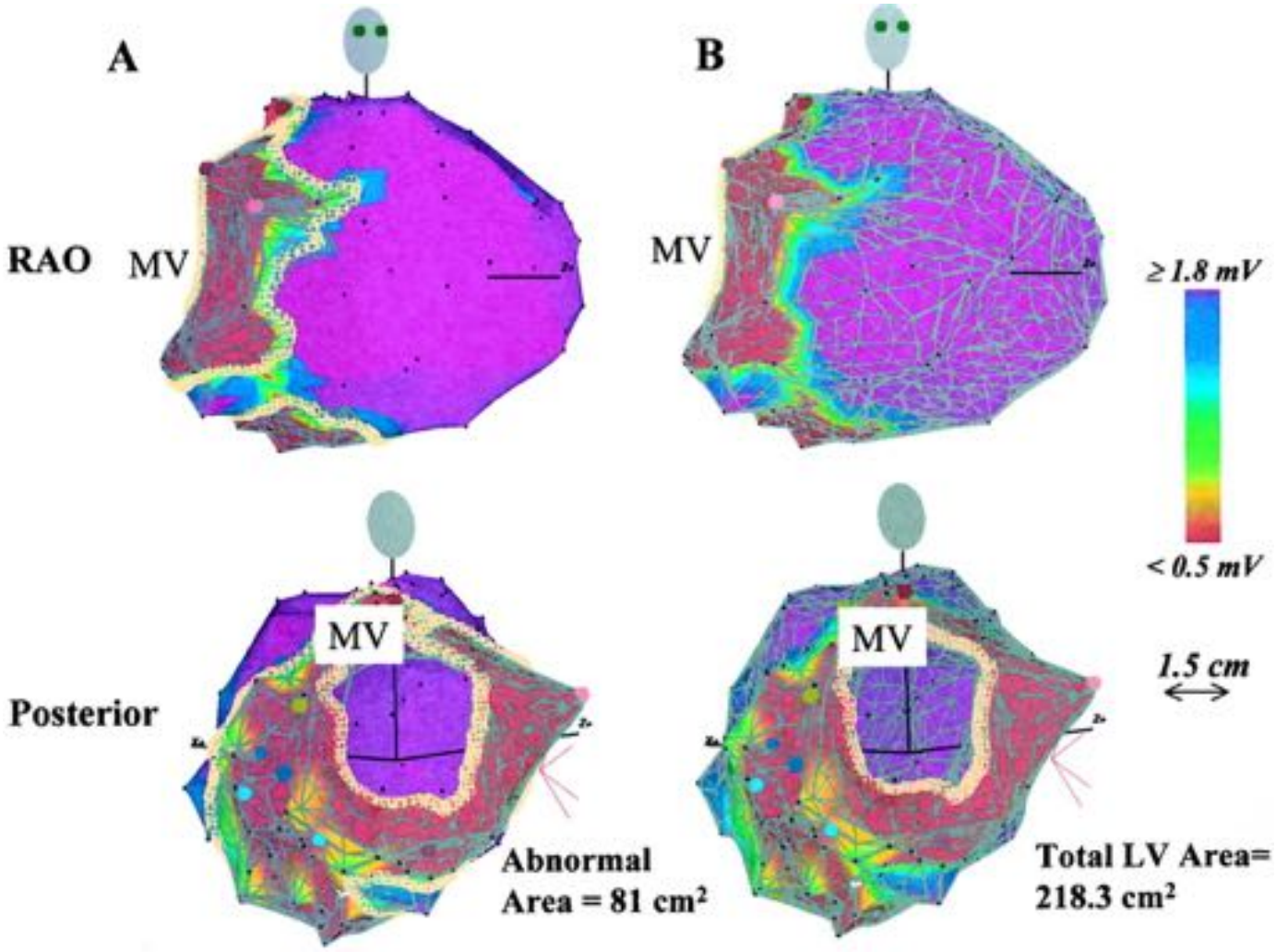
FLUOROSCOPY



ELECTROGRAMS

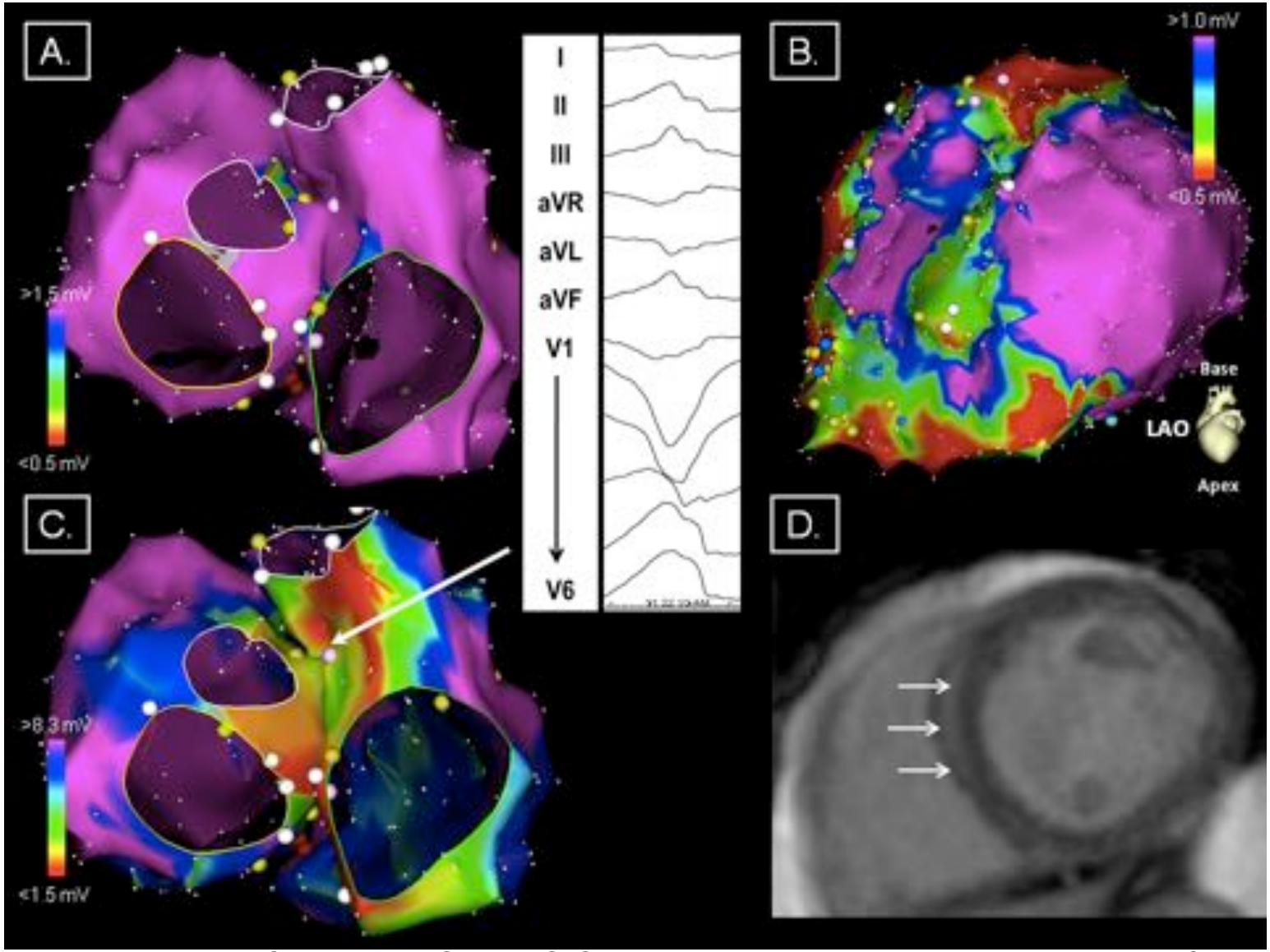
Cesario D, Vaseghi M, Boyle NG, Fishbein MC, Valderrabano M, **Narasimhan C,** Wiener I and Shivkumar K. Value of High density Epicardial and Endocardial Mapping for Catheter Ablation of Hemodynamically Unstable Ventricular Tachycardia. ***Heart Rhythm*** 2006 ;3:1-10

Figure 1. Estimation of the abnormal LV endocardium in a patient with nonischemic cardiomyopathy.



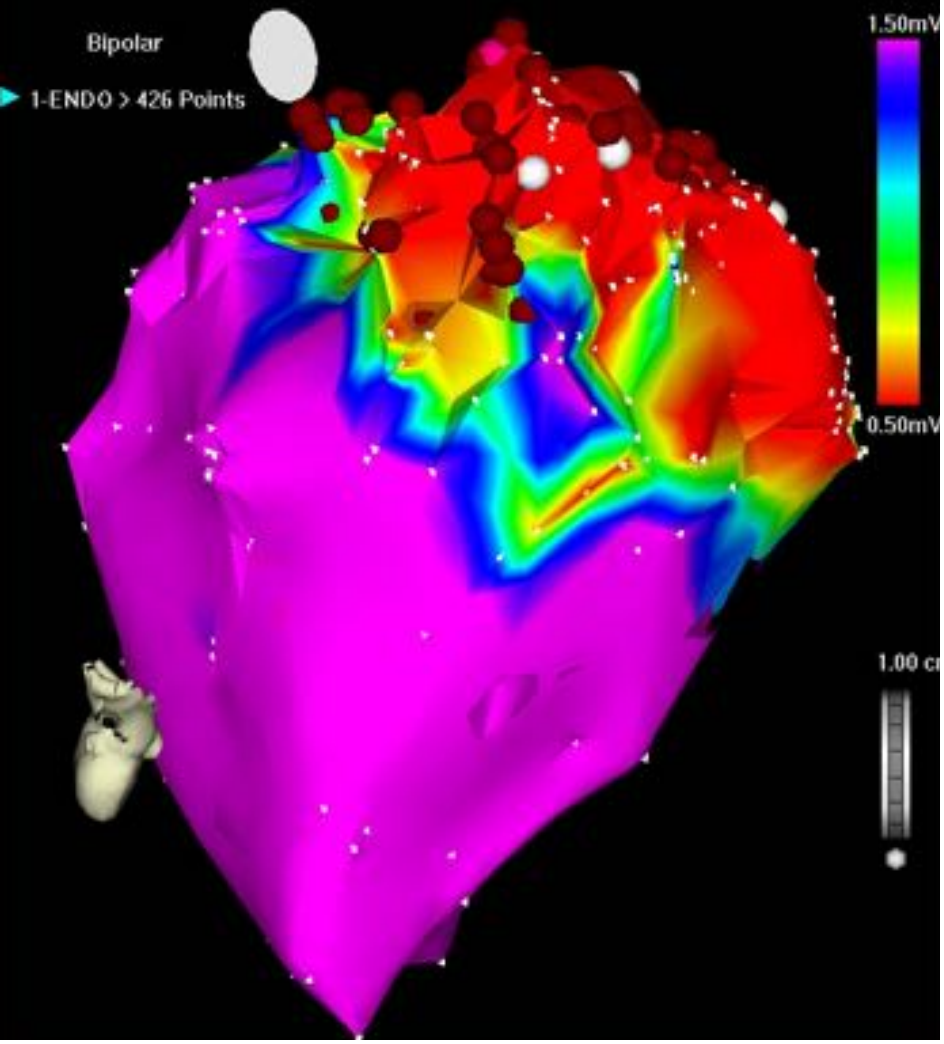
Henry H. Hsia et al. Circulation. 2003;108:704-710

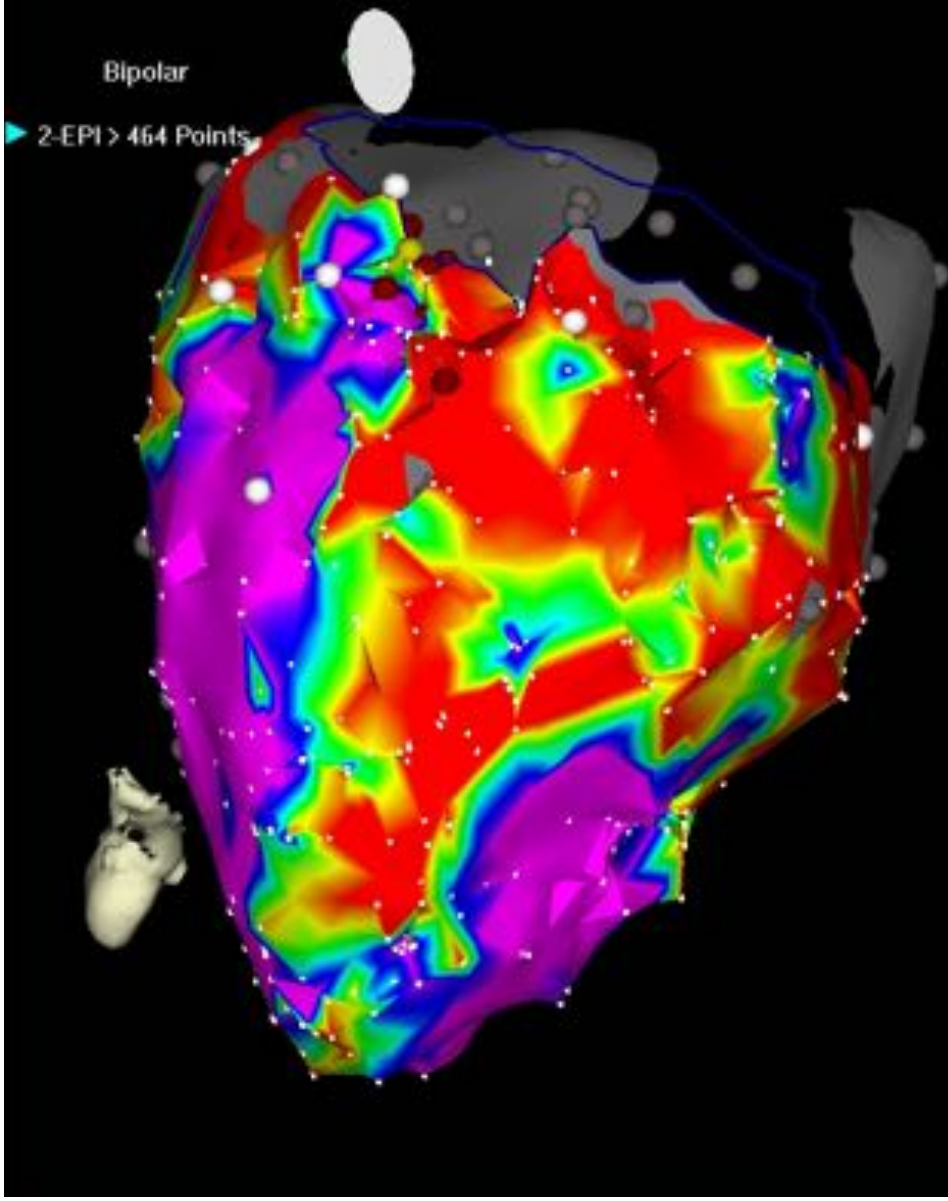
Isolated Septal Substrate for NICM

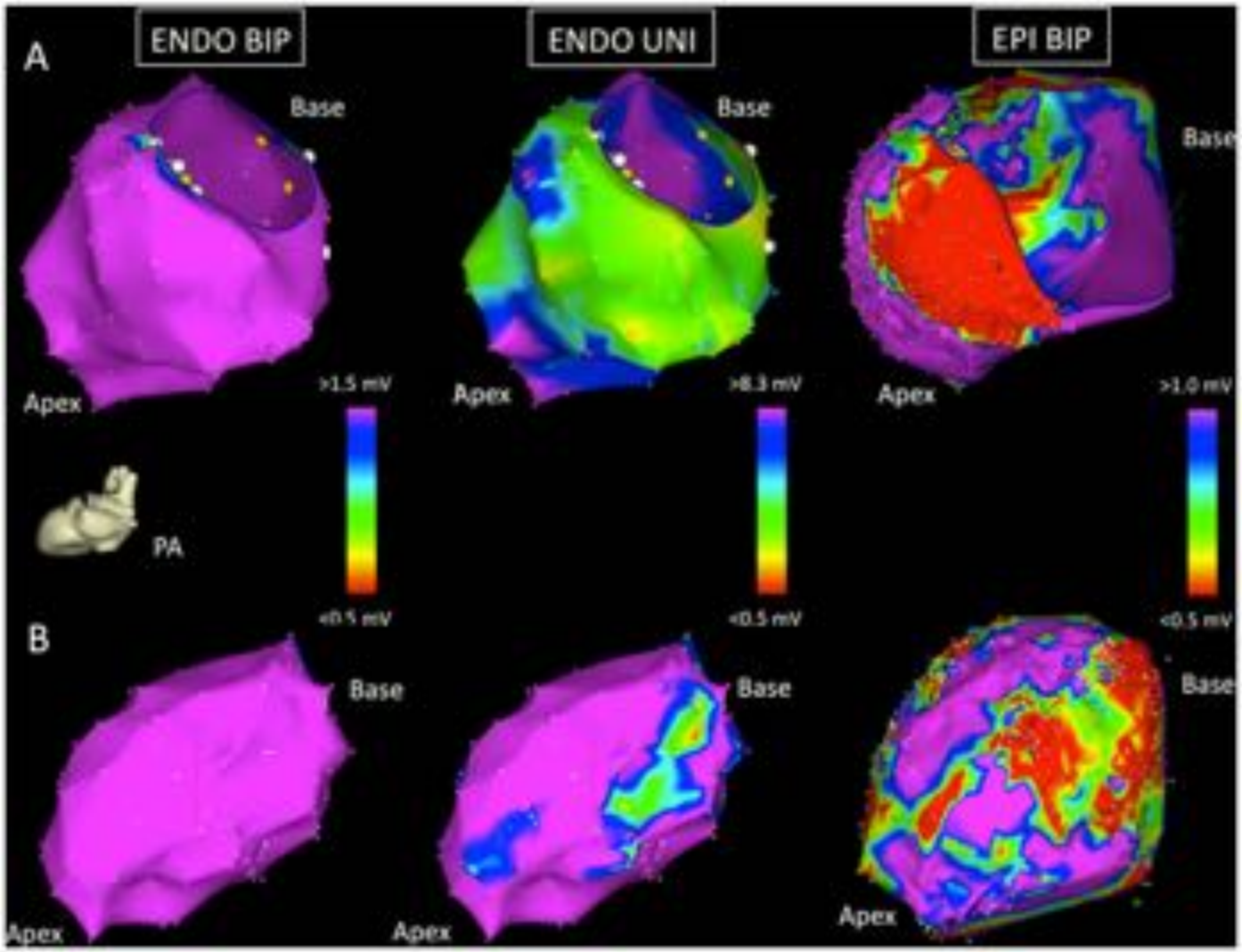


Haqqani, H.M., Tschabrunn, C.M., Tzou, W.S., Dixit, S.,Cooper, J.M., Riley, M.P., Lin, D., Hutchinson, M.D., Garcia, F.C., Bala, R., Verdino,R.J., Callans, D.J., Gerstenfeld, E.P., Zado, E.S., Marchlinski, F.E., Isolated Septal Substrate for Ventricular Tachycardia in Nonischemic Dilated Cardiomyopathy: Incidence, Characterization and Implications, ***Heart Rhythm (2011), doi: 10.1016/j.hrthm. 2011.03.008.***

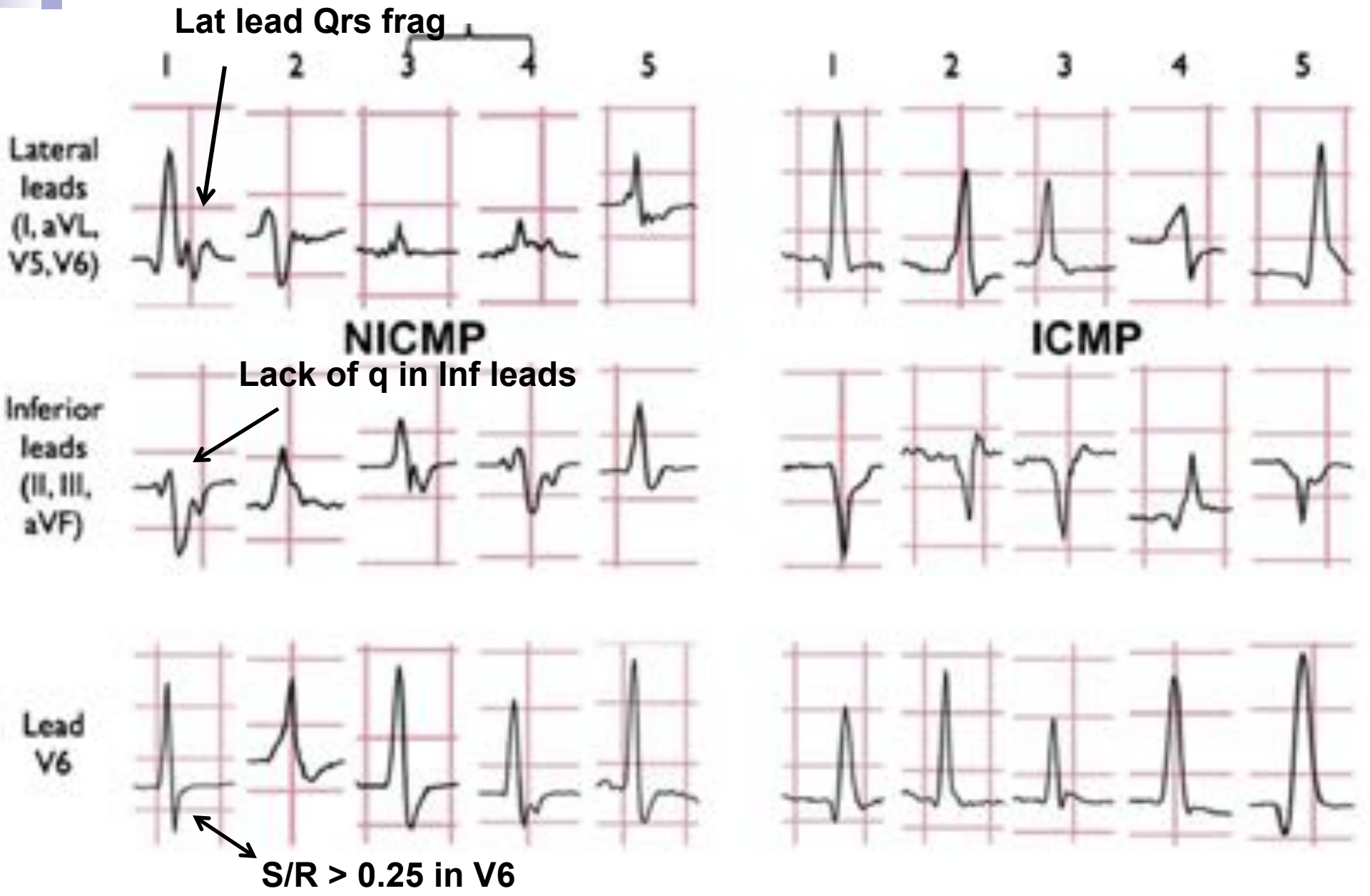
Idiopathic Nonischemic CM



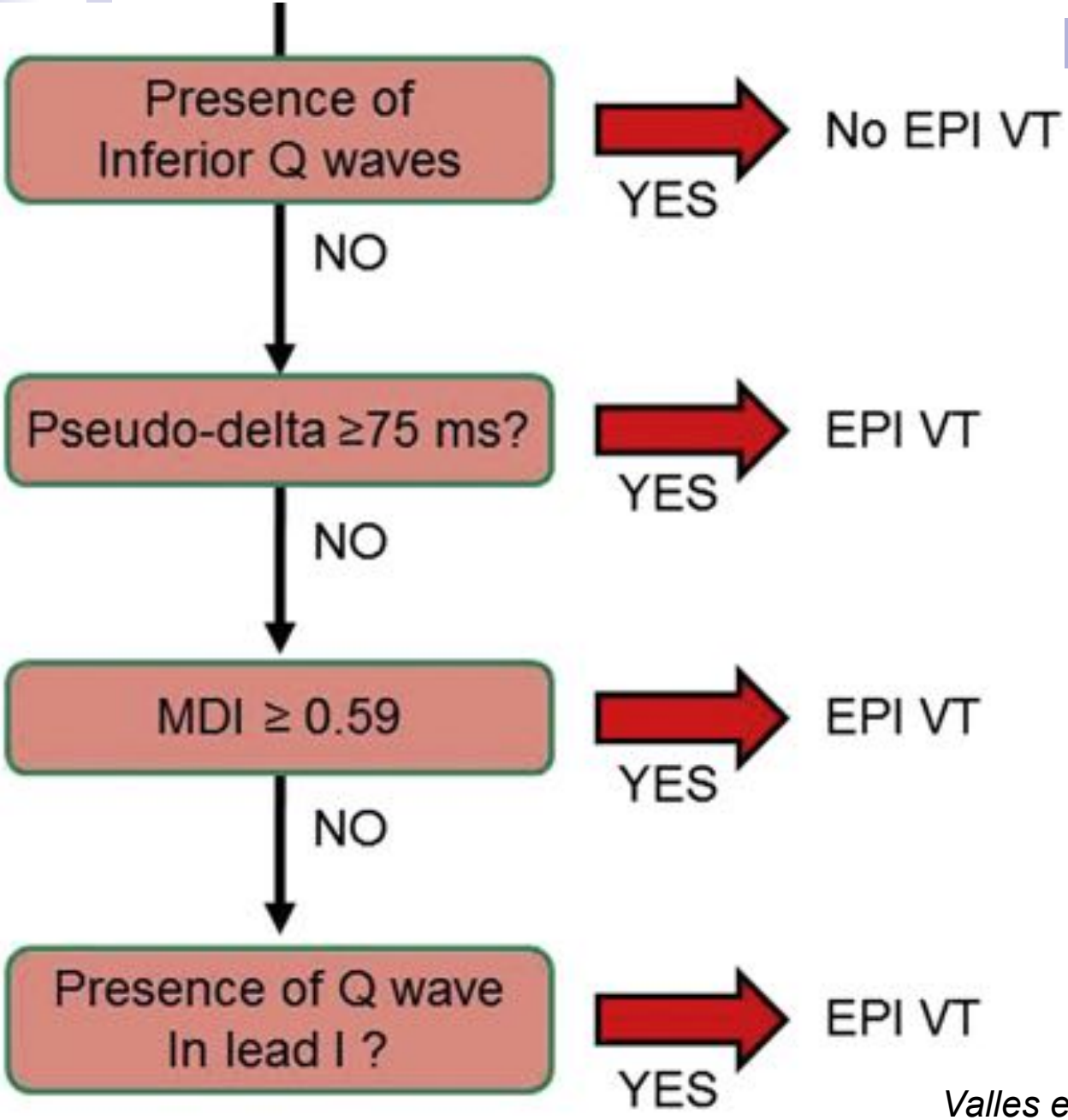




Hutchinson et al *Circ Arrhythm Electrophysiol 2011*



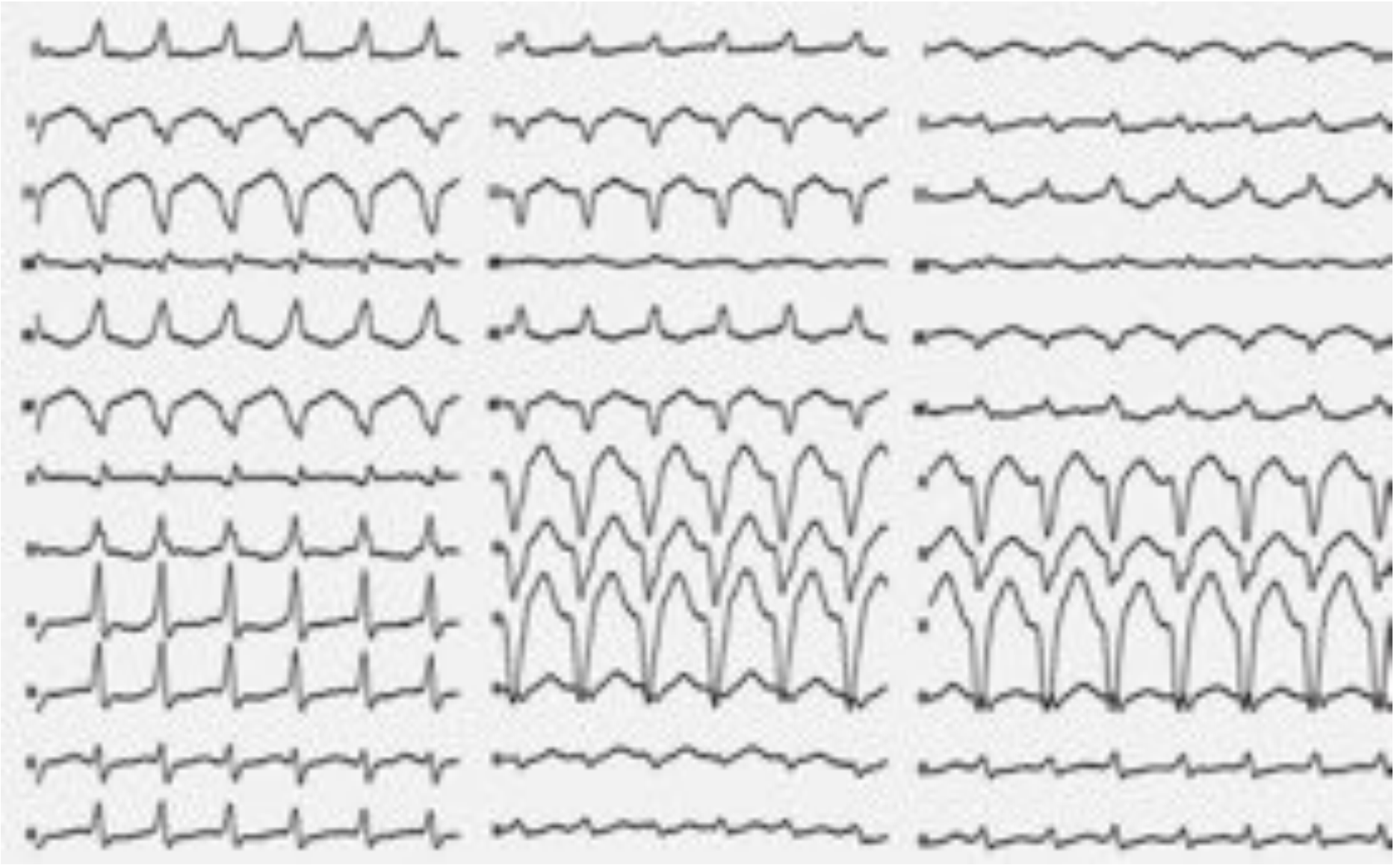
Betensky et al : J Interv Card Electrophysiol 2012;35:311–21;

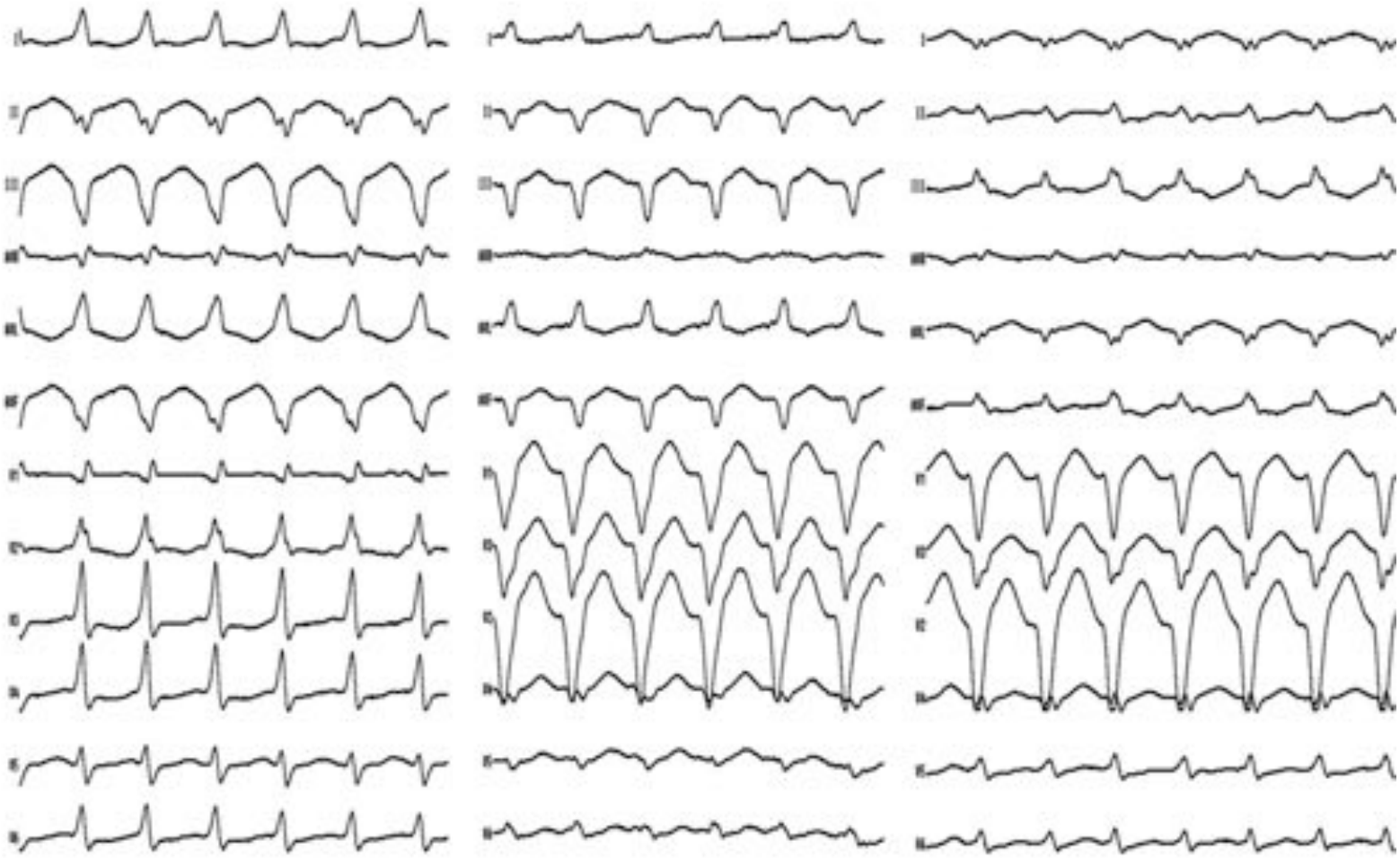


Valles et alCirc Arrhythm Electrophysiol 2010;3:63–71

Sensitivity = 96% Specificity= 93%

42 yr old male with Non ischemic Cardiomyopathy with recurrent episodes of VT-onBB+ Amio



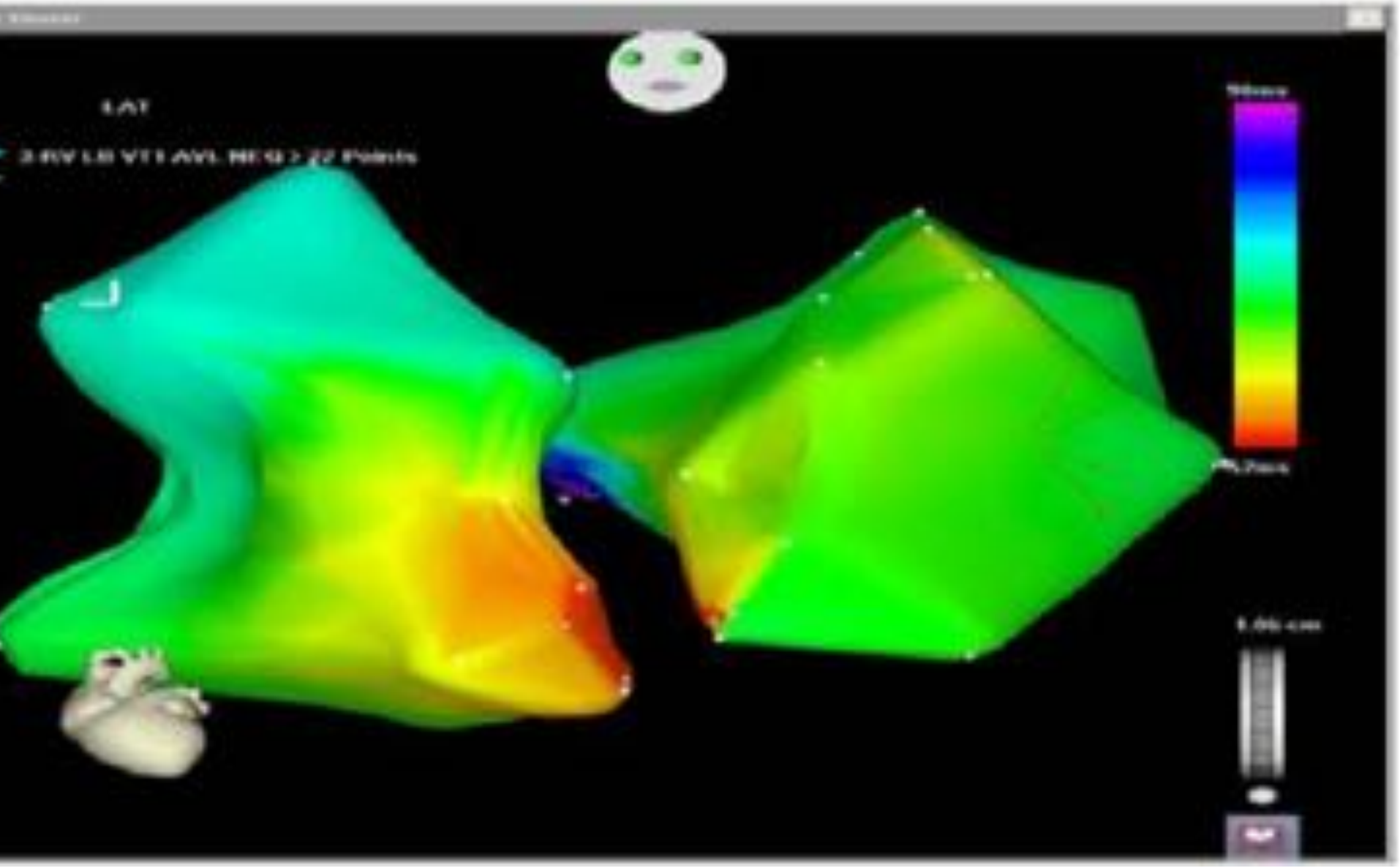


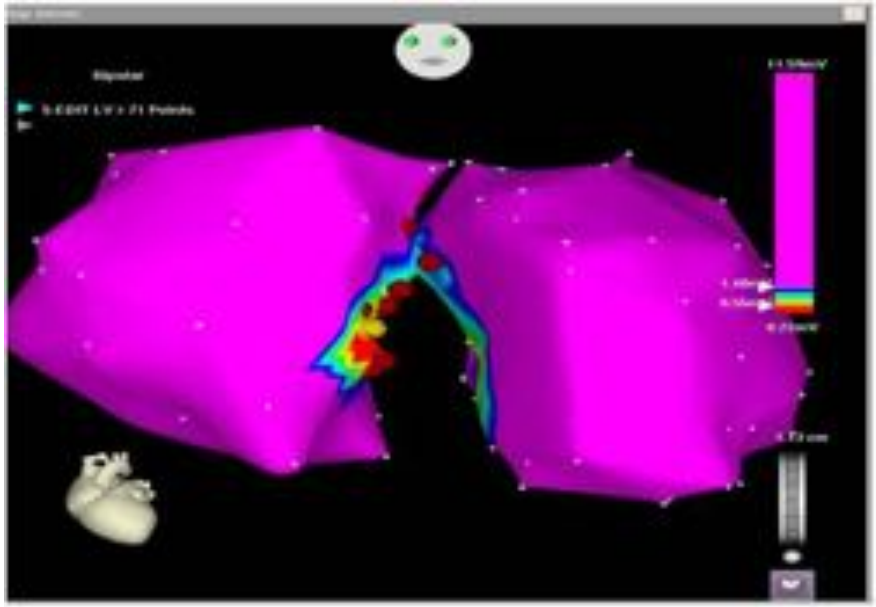
•RB LA

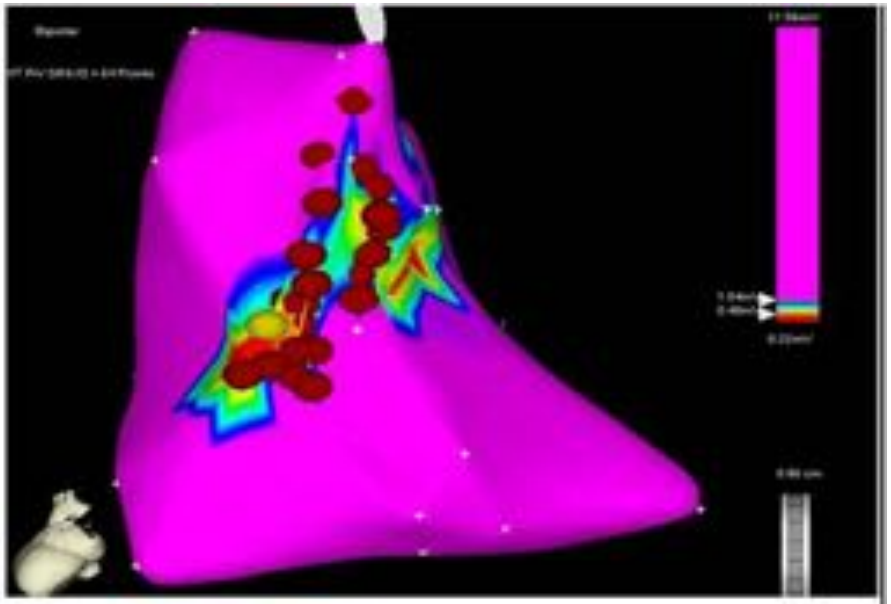
•LB LA

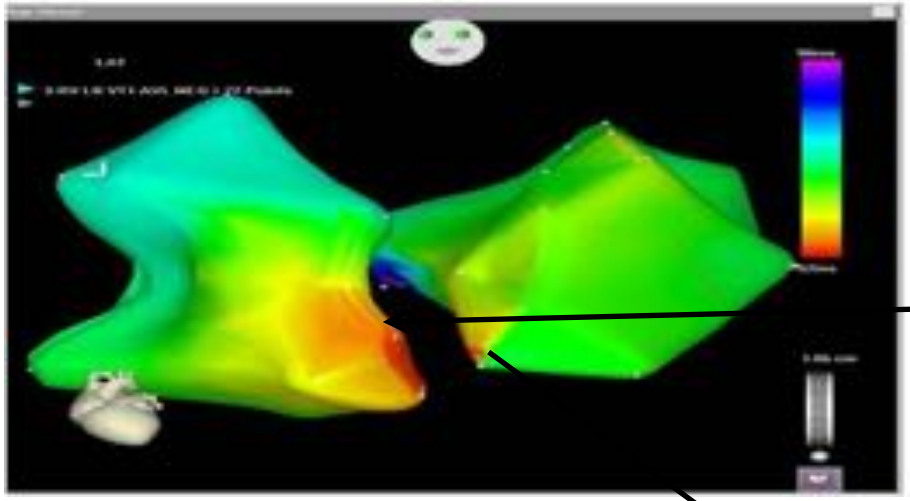
•LB NA

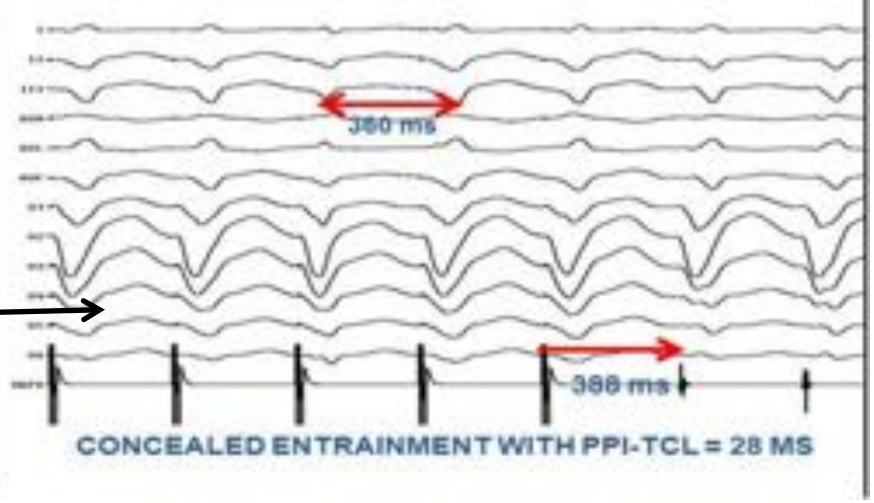
•Activation Map from RV and LV





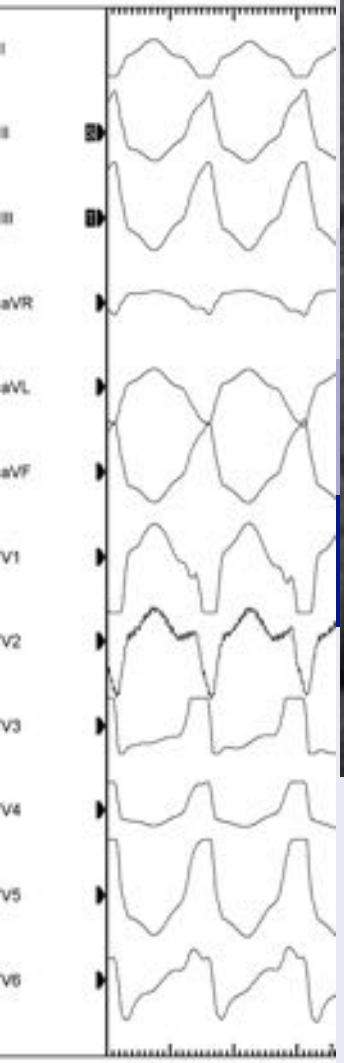


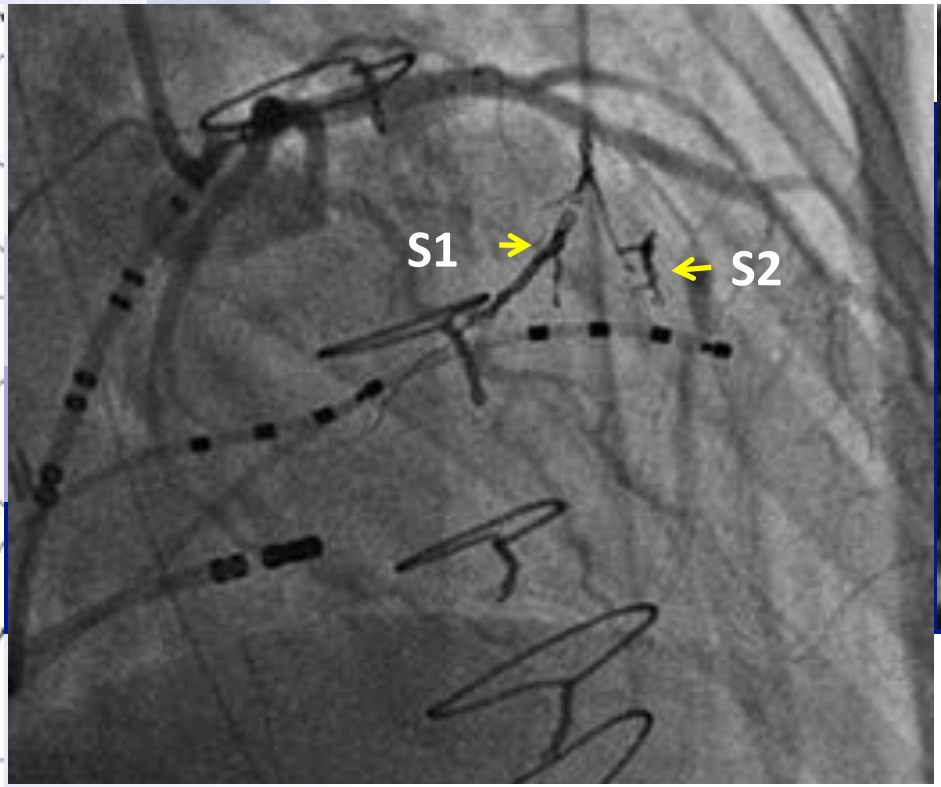




•Manifest entrainment from septal surface of LV

INTRA-SEPTAL VT : MAPPING AND ABLATION

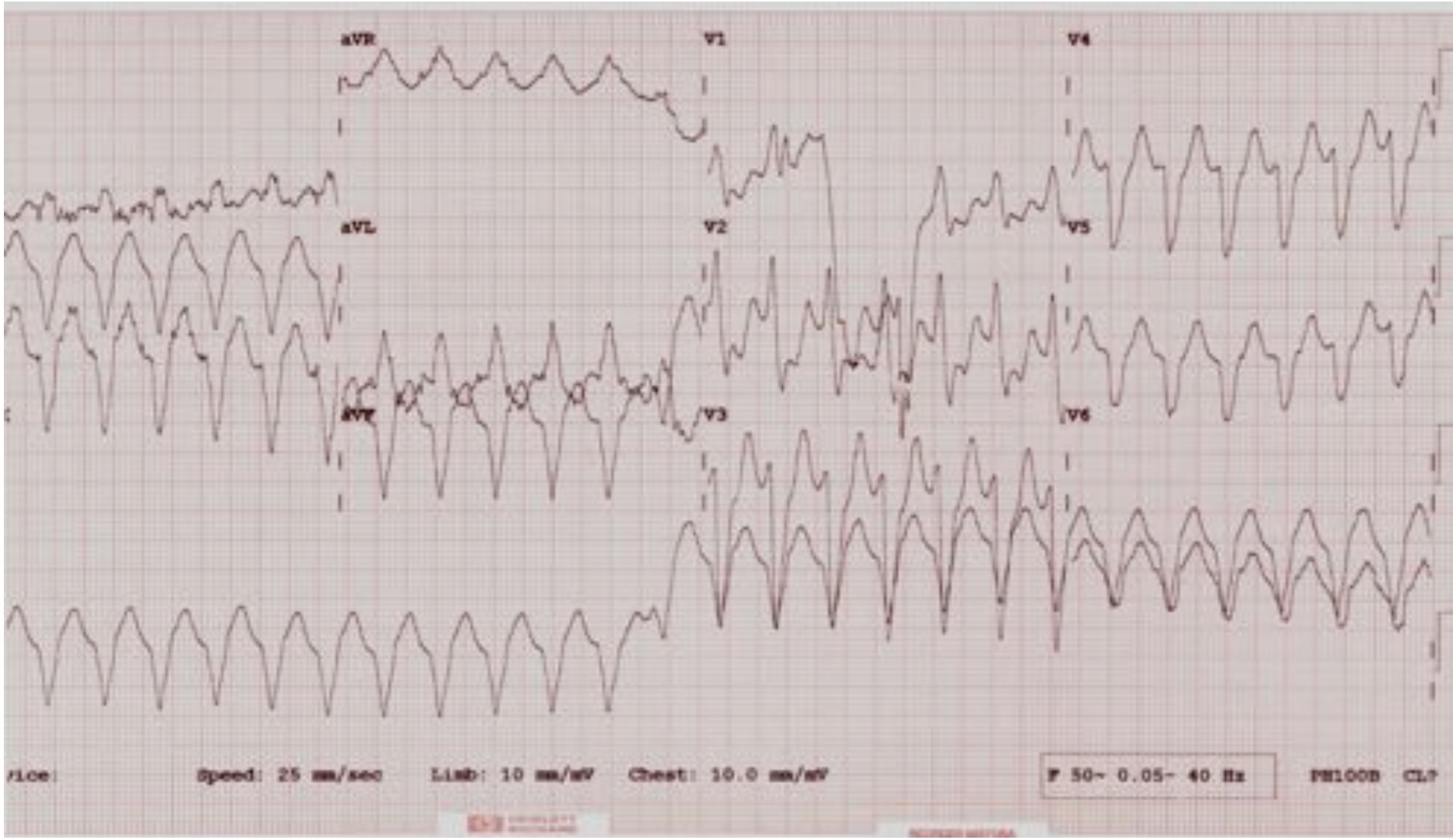




Tholakanahalli V, Bertog S, Roukoz H & Shivkumar K: Catheter Ablation Of Ventricular Tachycardia Using Intracoronary Wire Mapping And Coil Embolization: Description Of A New Technique. 2012

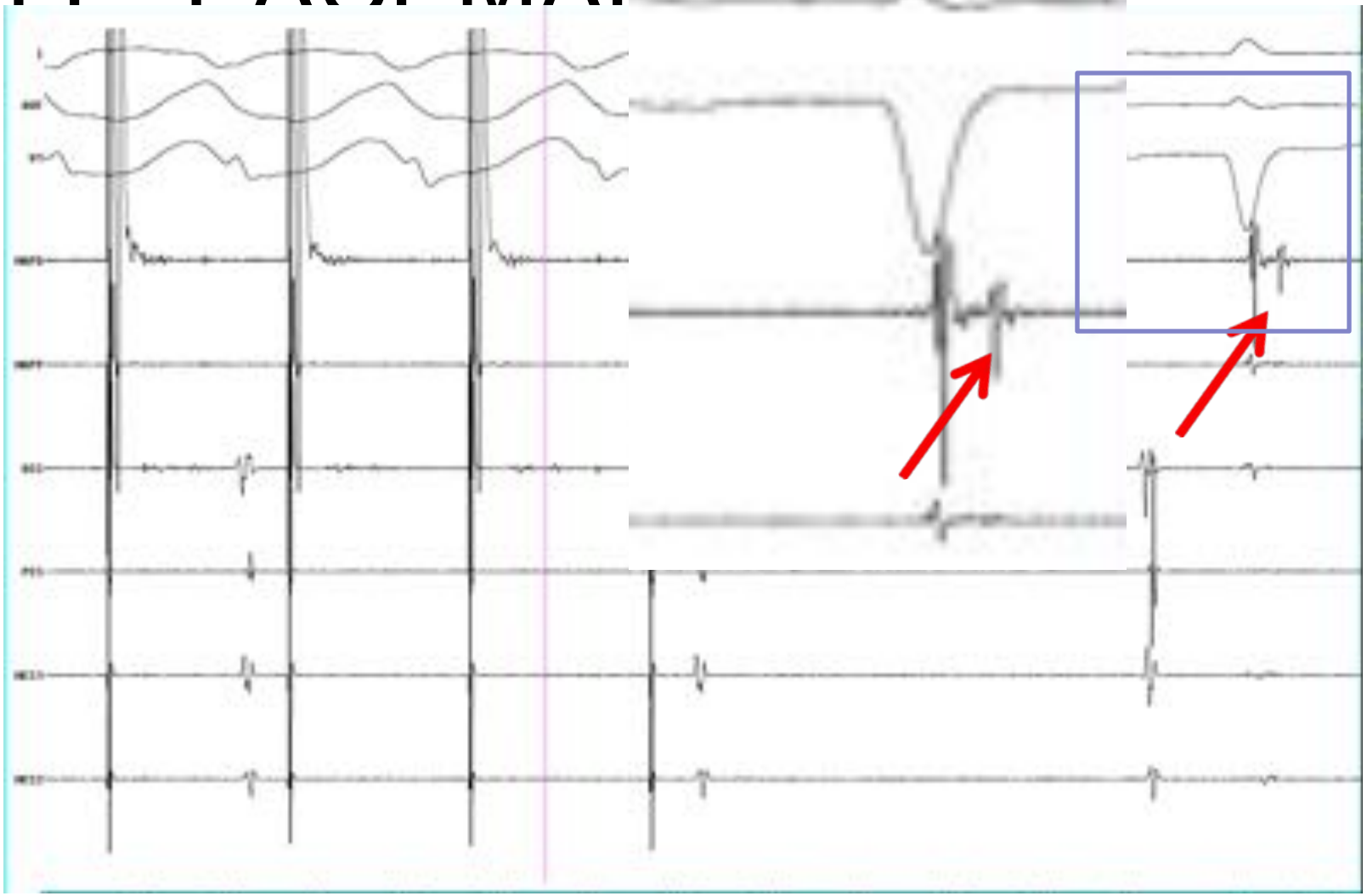
72 M, DCM Recurrent AICD shocks

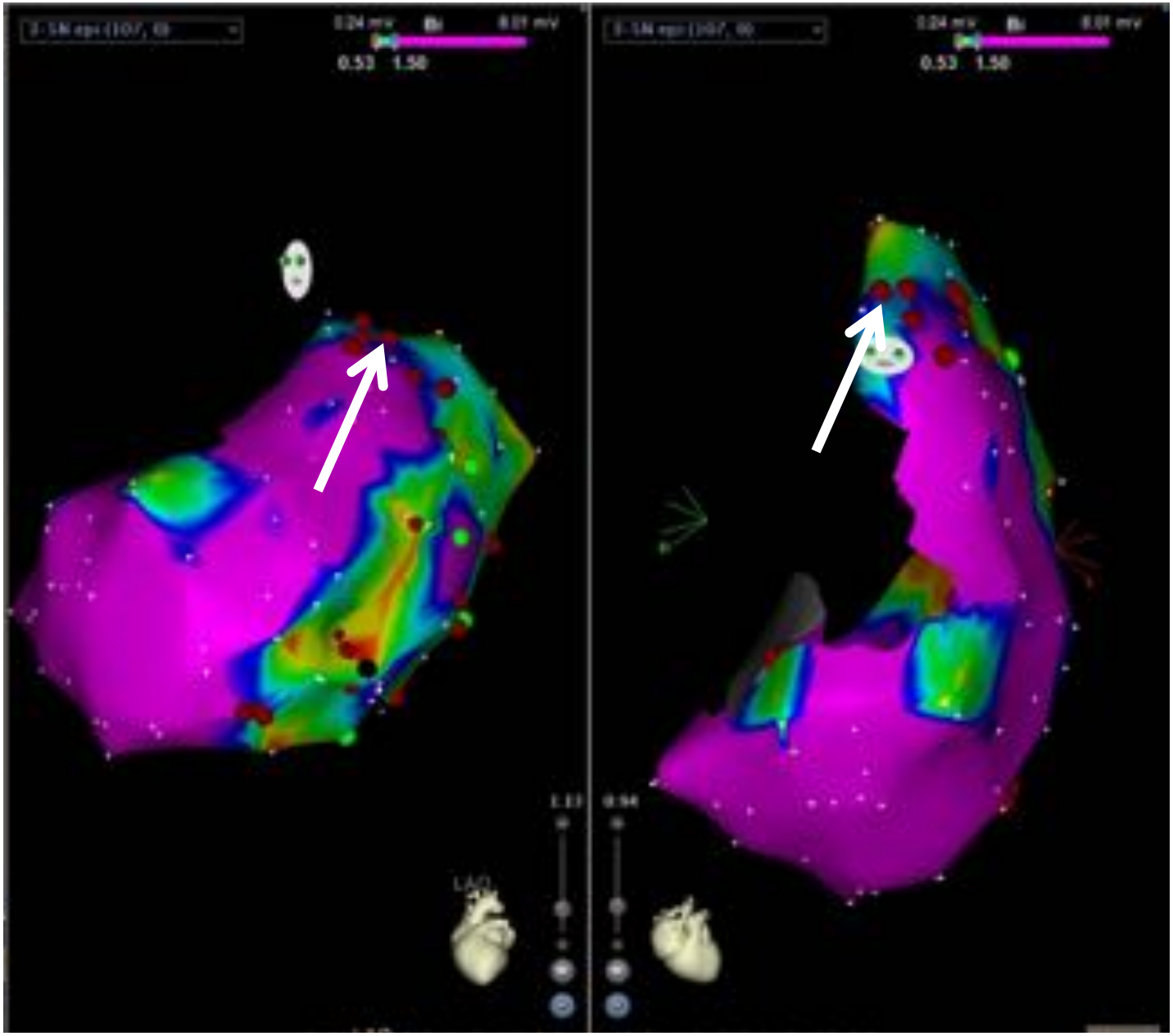
On Sotalol ,metoprolol and Mexiletine

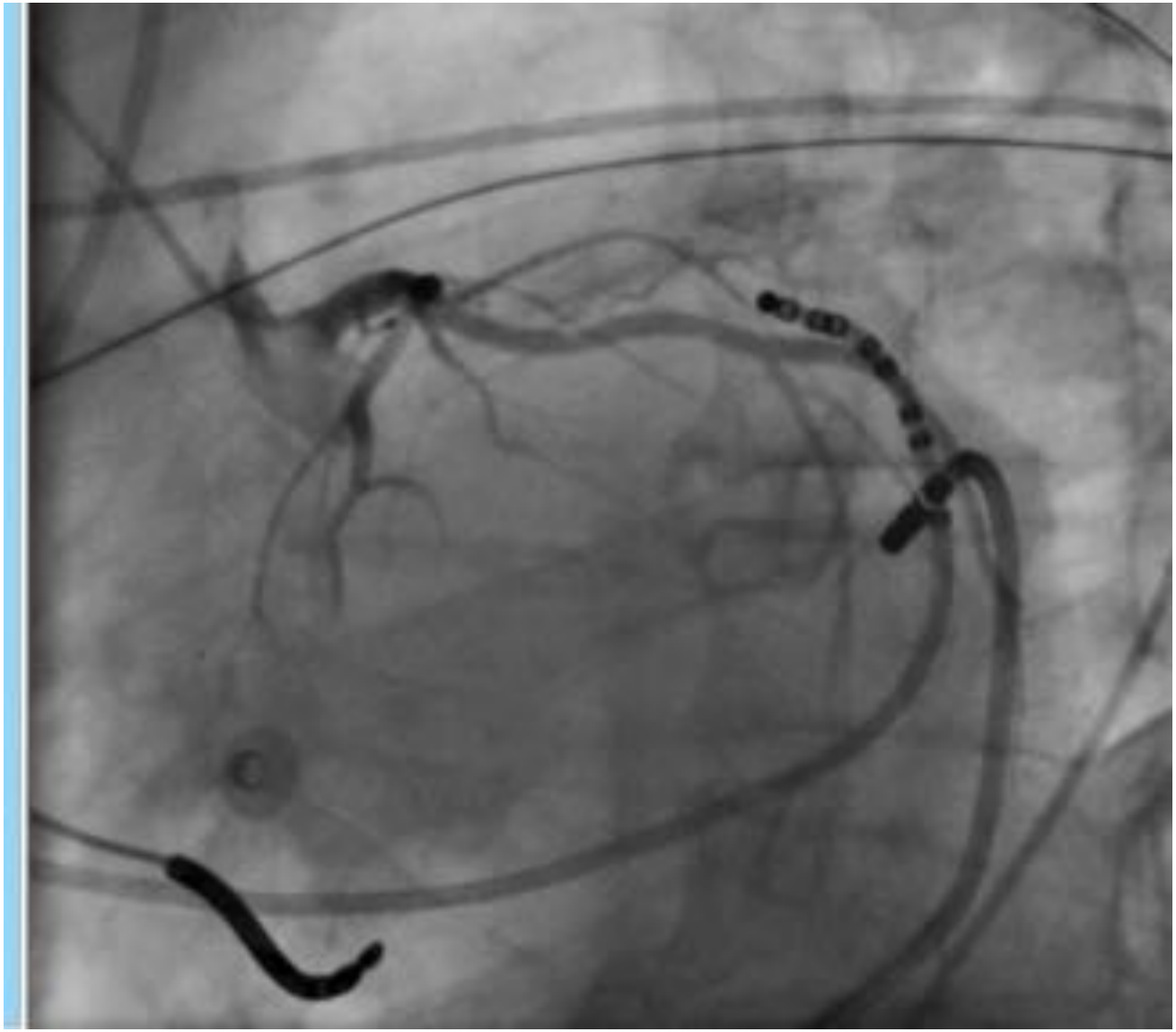


VT1 induced with V1V2

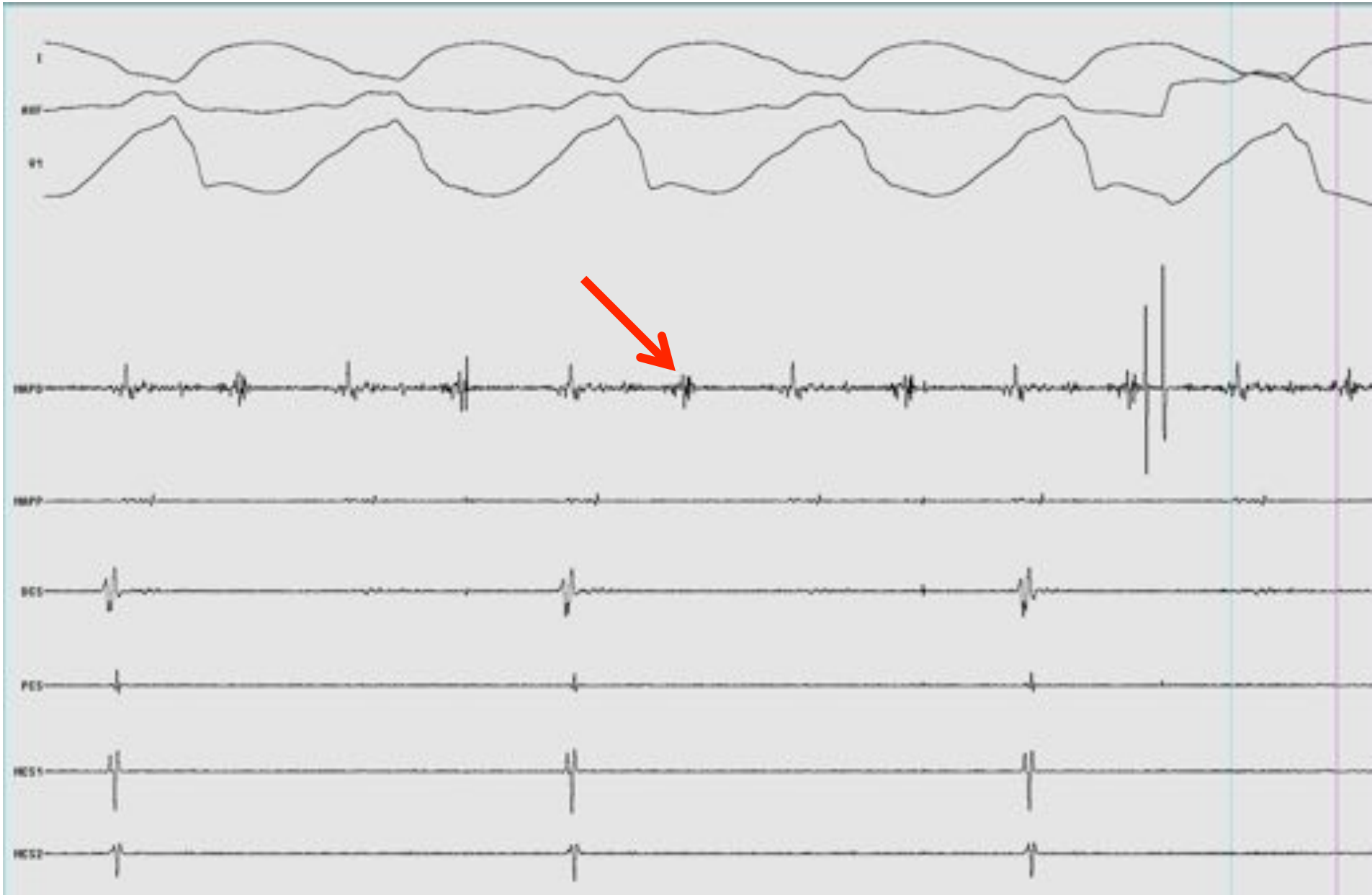
II P PACEMAP



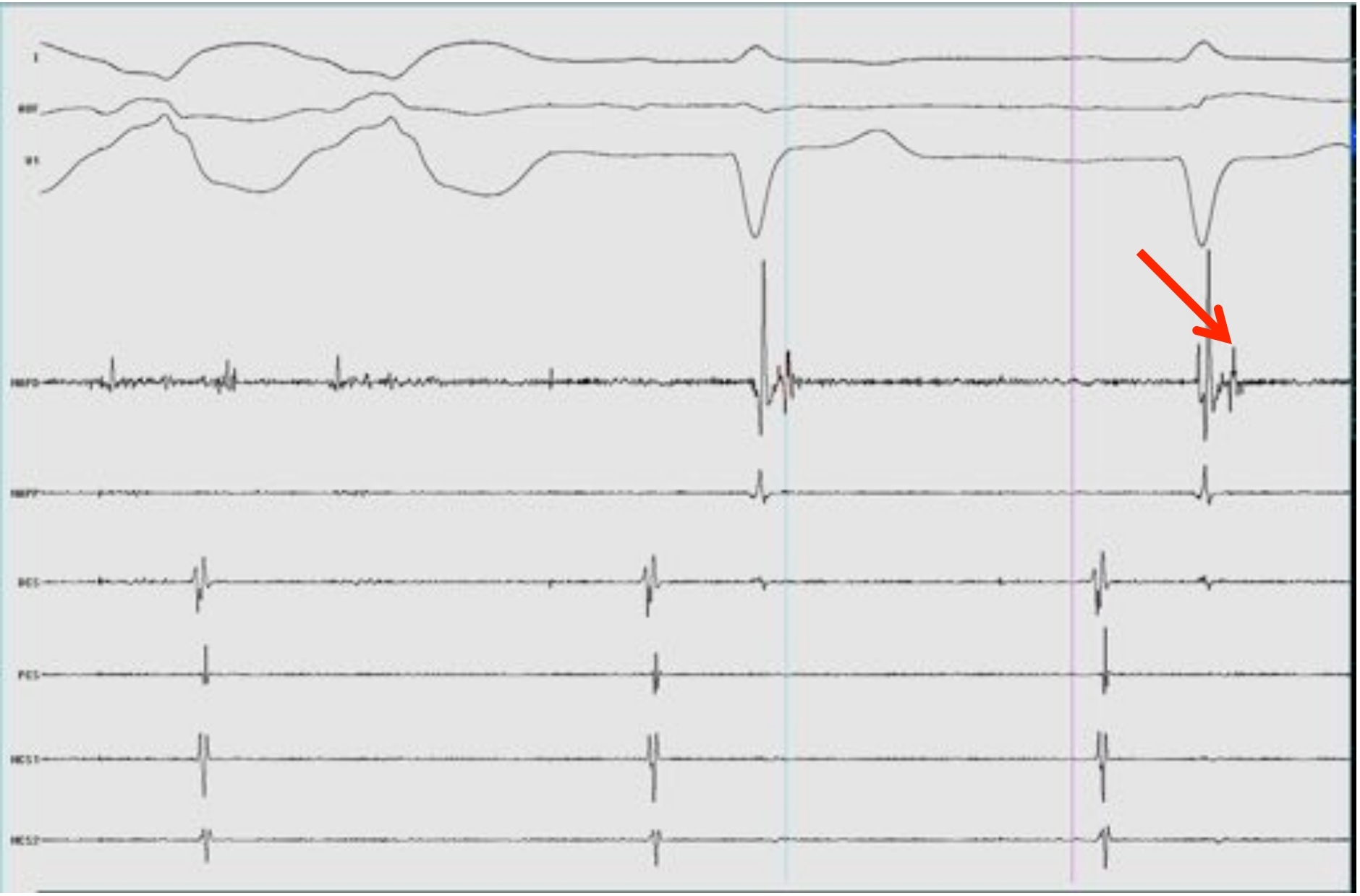

0.53 1.50
0.53 1.50



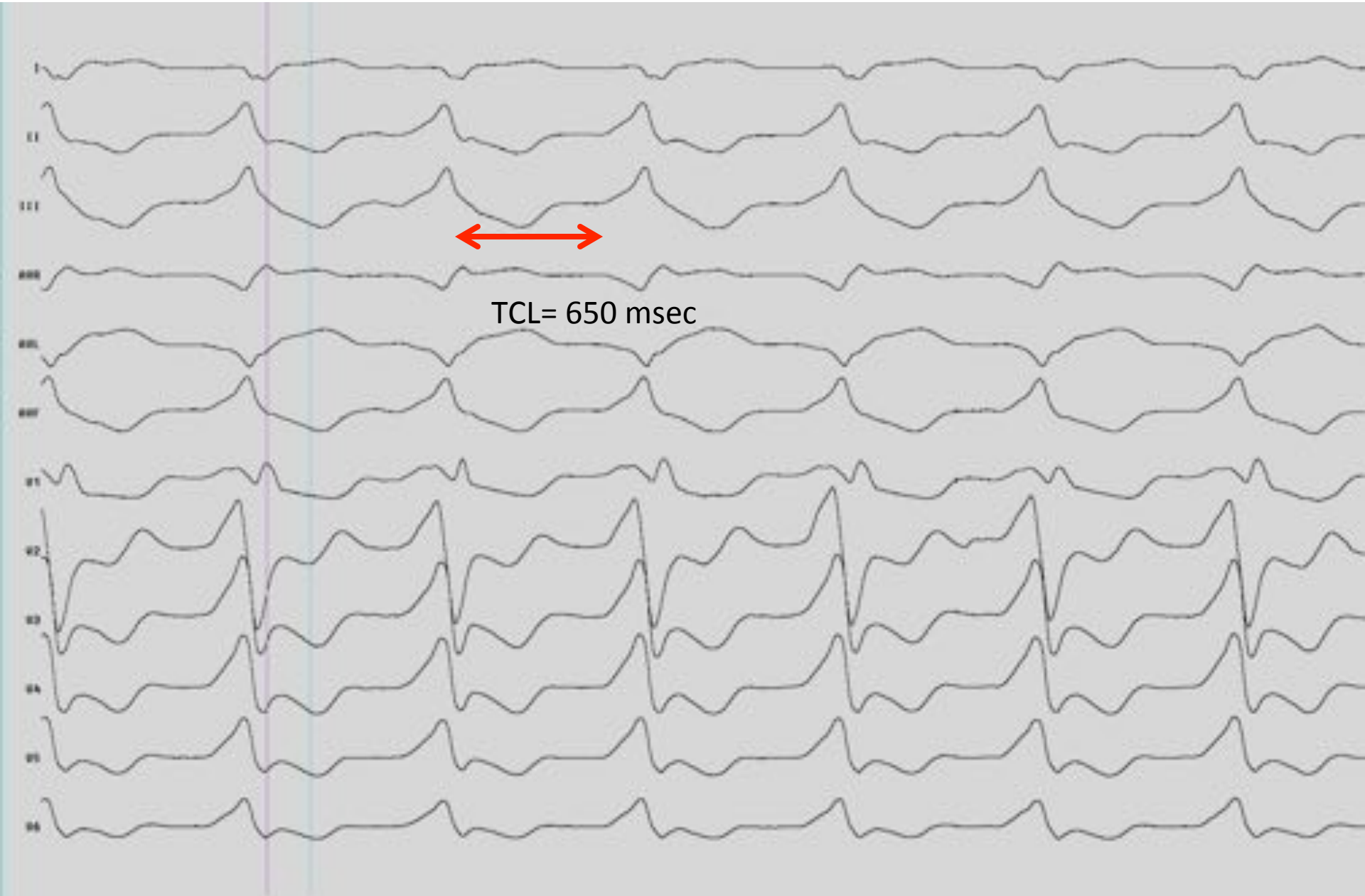
Tachy induced during pace mapping MDP

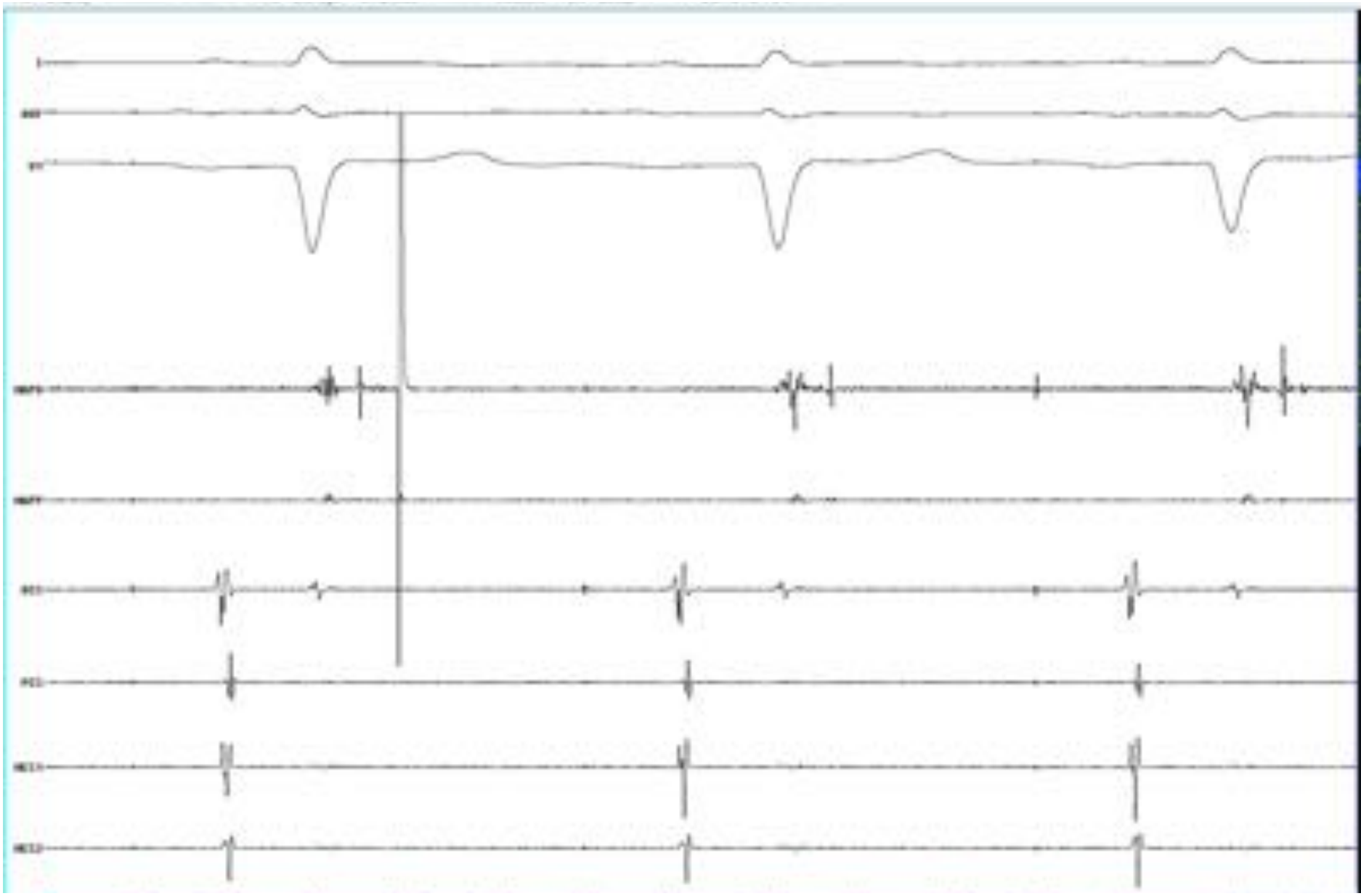


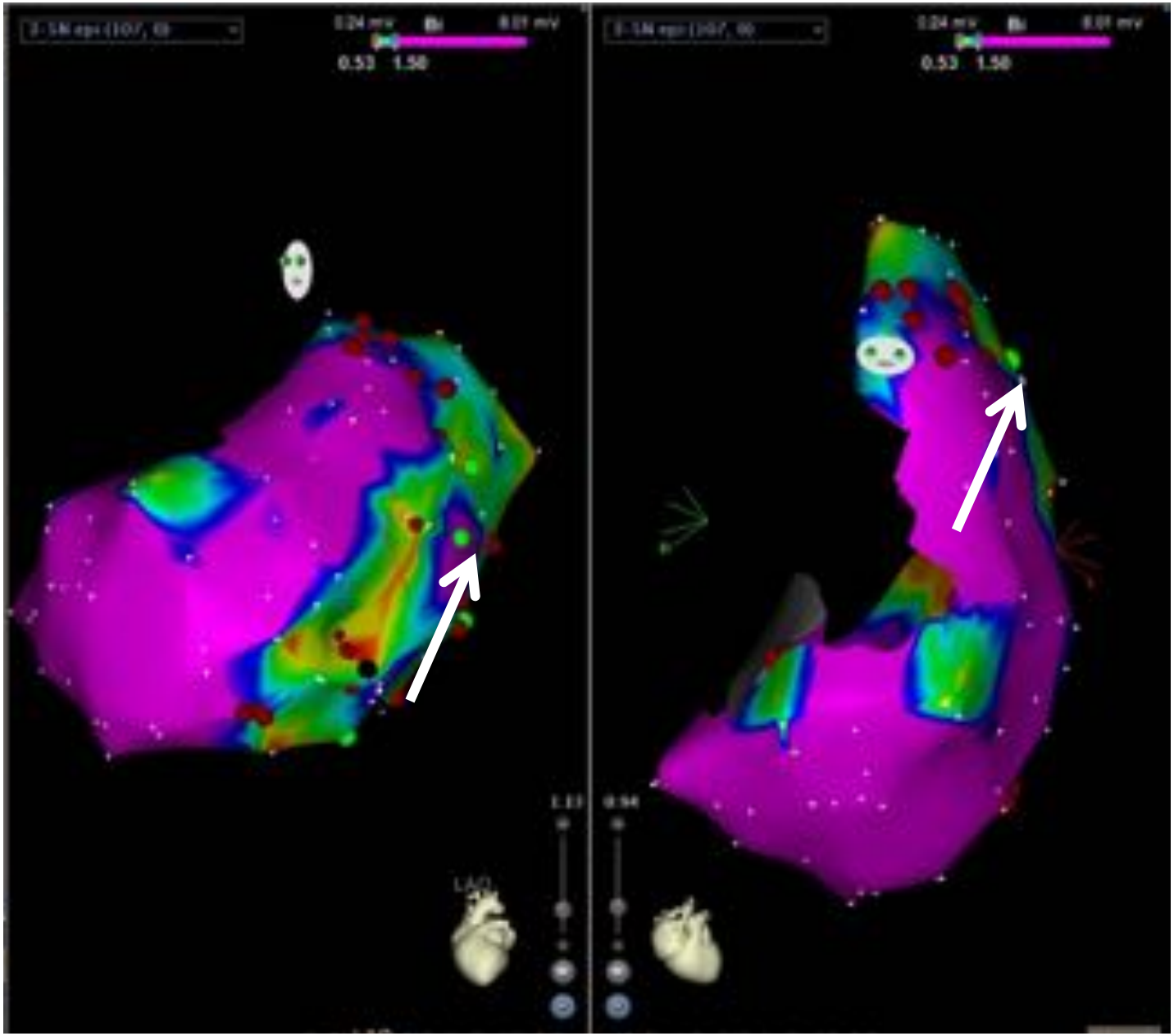
8 SEC OF RFA TACHY TERM NOTE ILP



VT3






0.53 1.50
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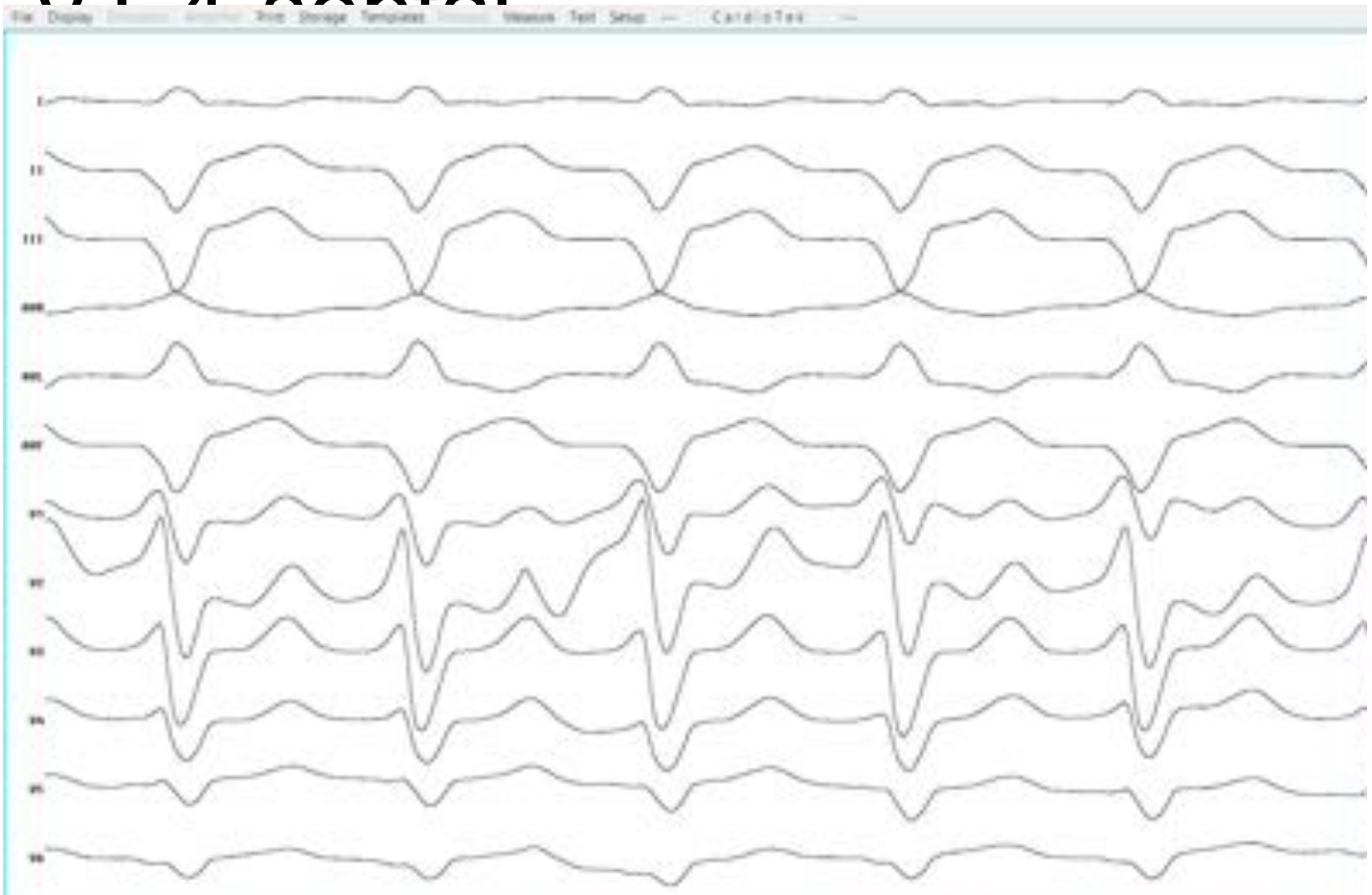
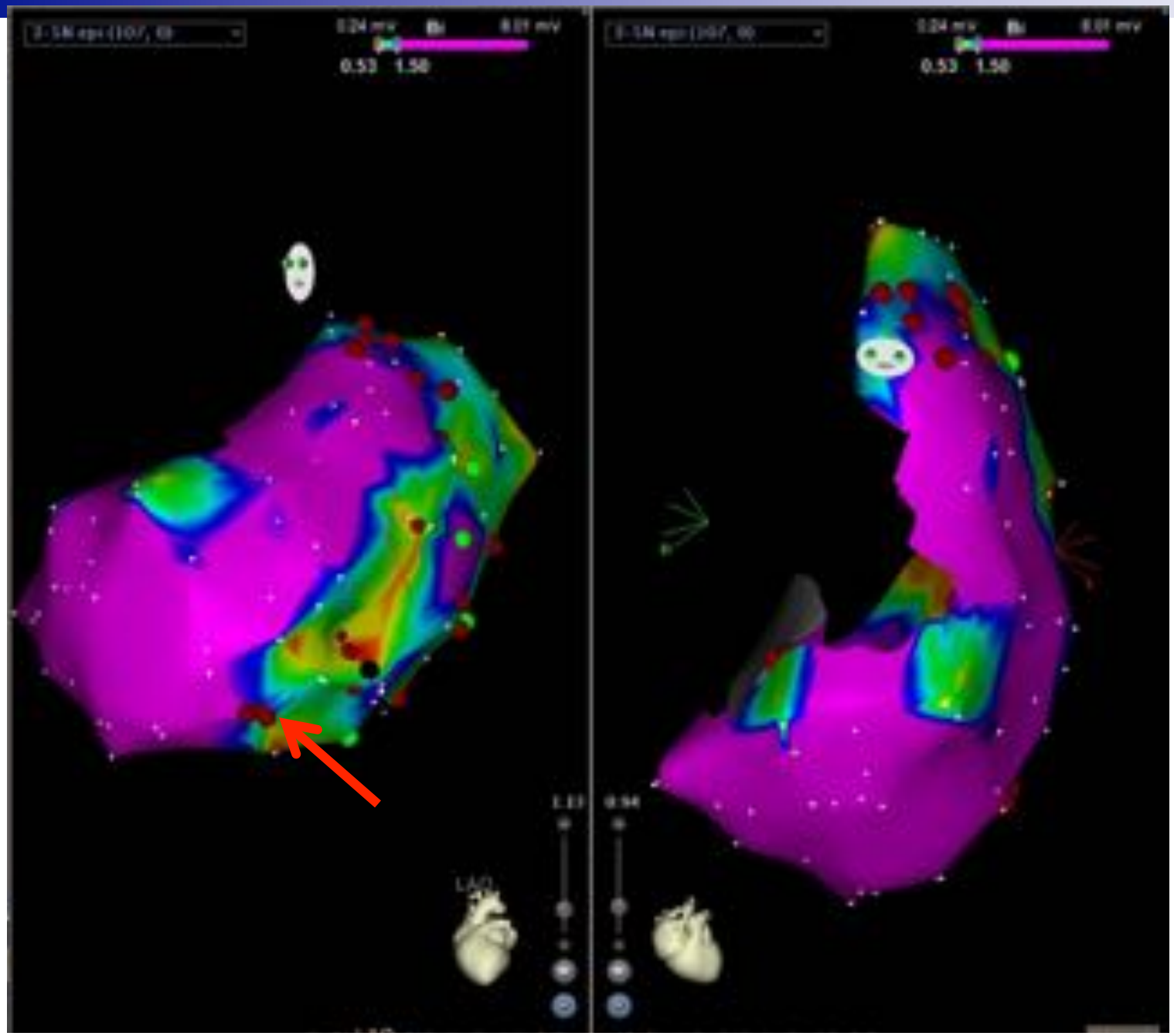
VT terminated during RFA

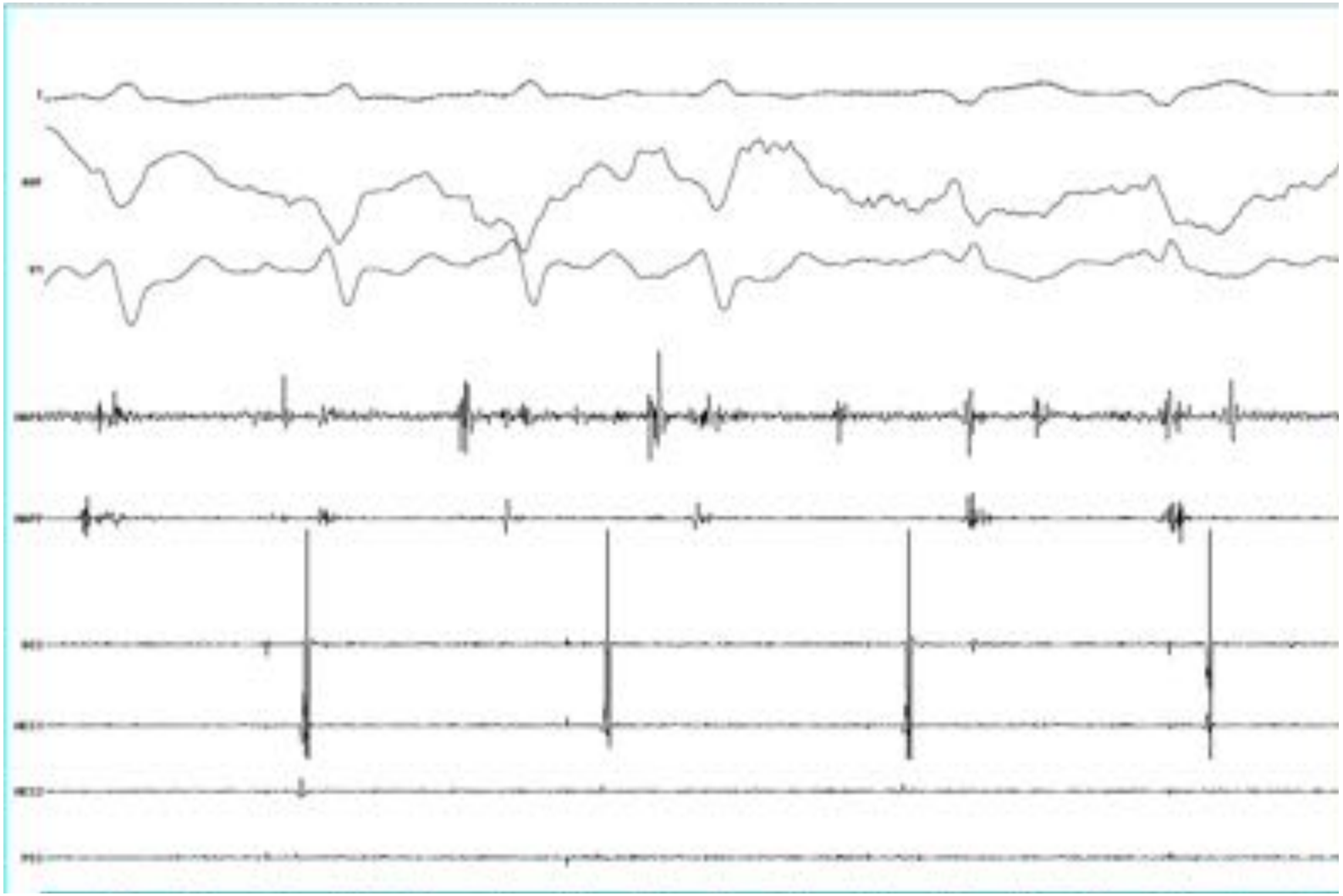
DURING RFA DISAPPEARNCE OF LP

VT 4 septal

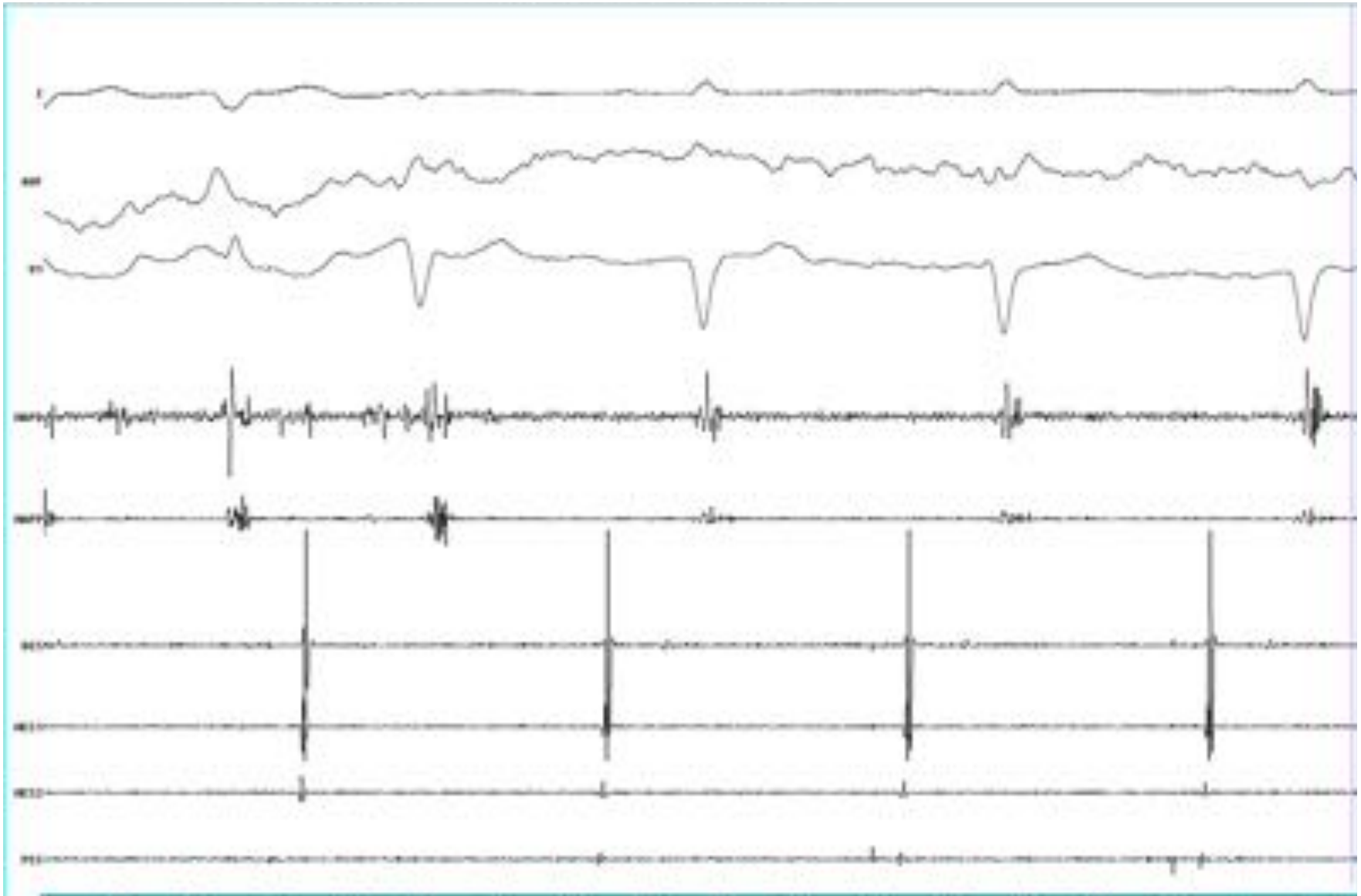



0.24 mv
8.01 mv
0.53
1.50
LAO

5 sec RFA changed to other vt



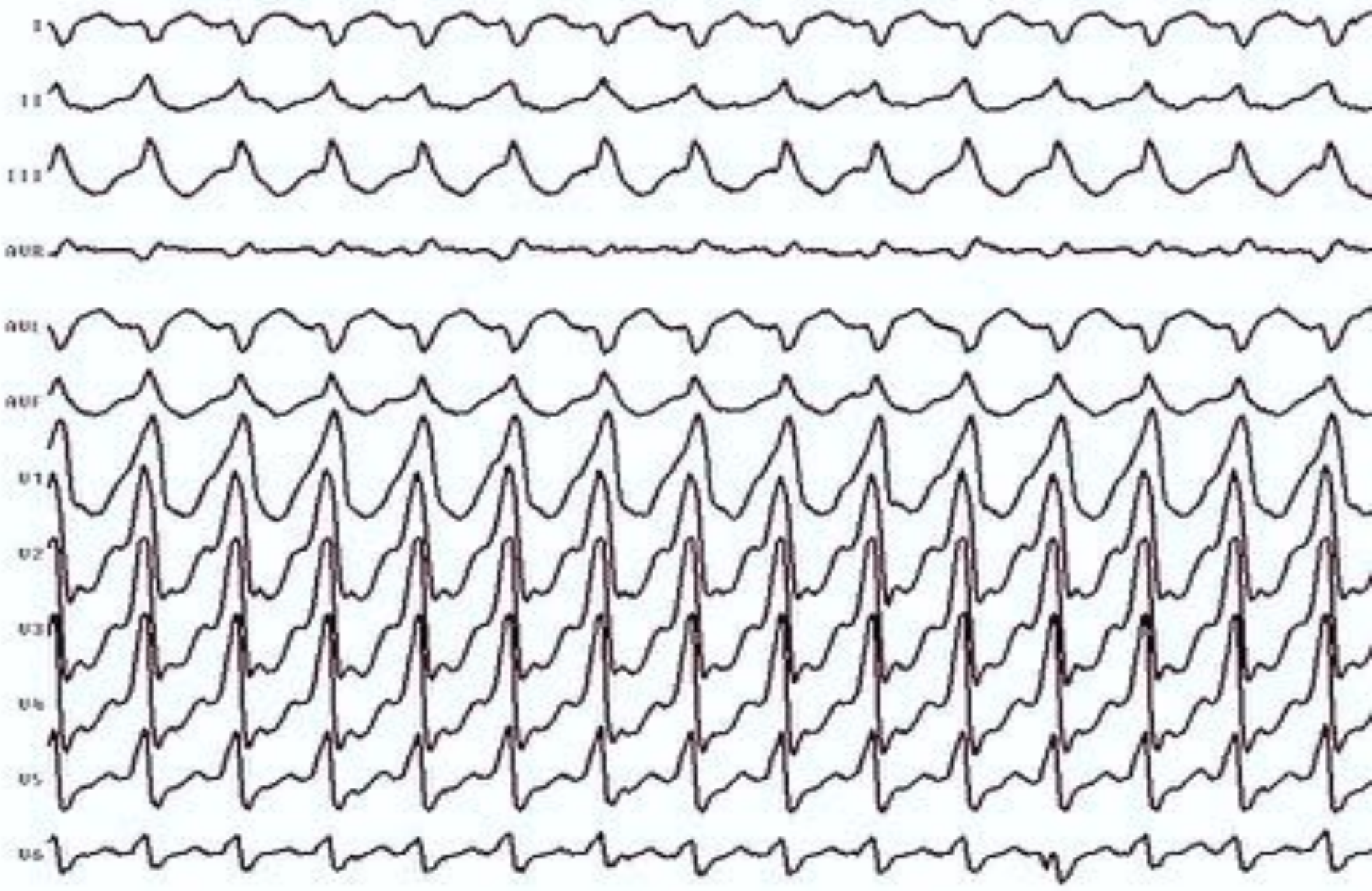
Terminated during RFA 3 sec

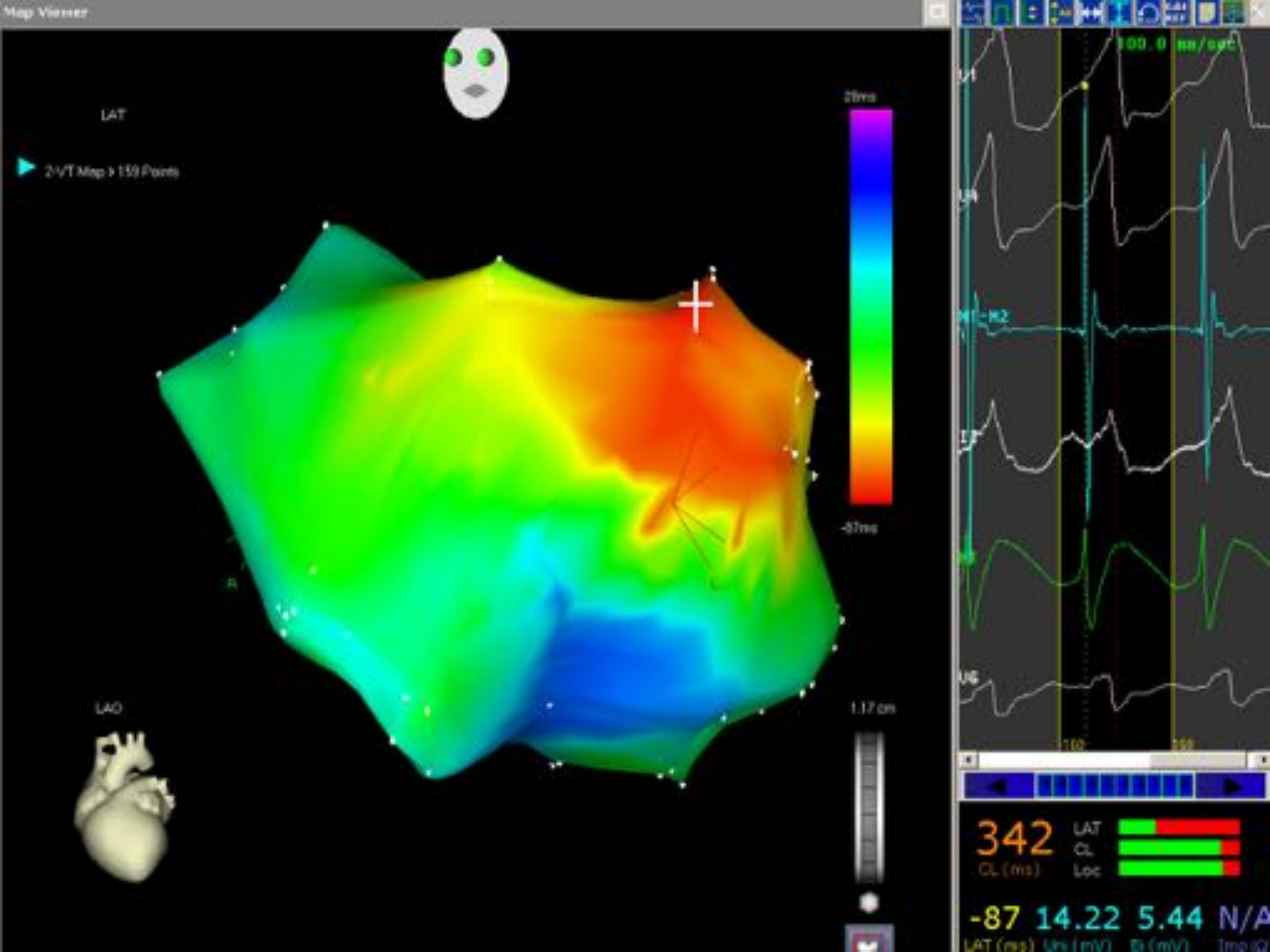


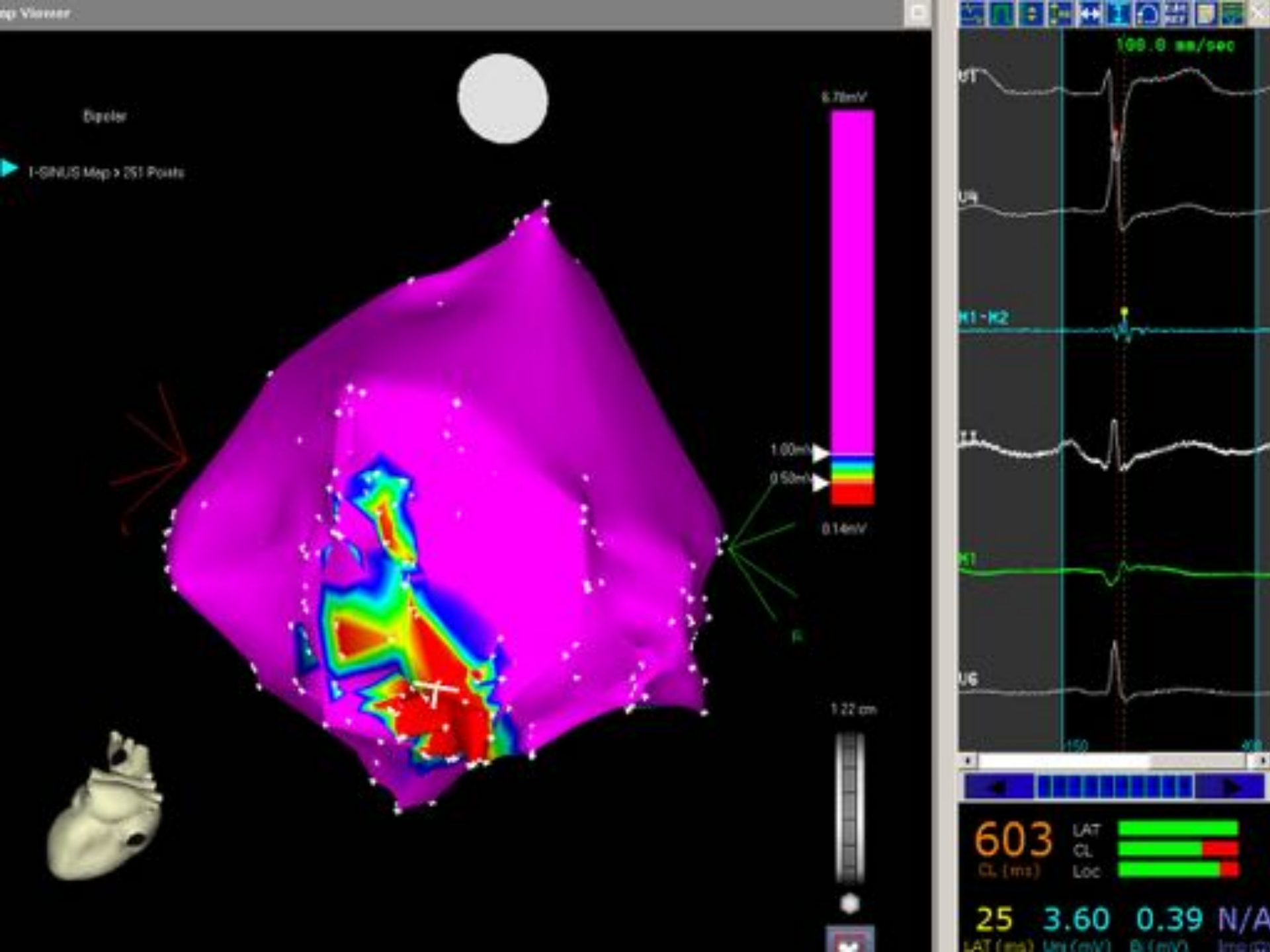
History

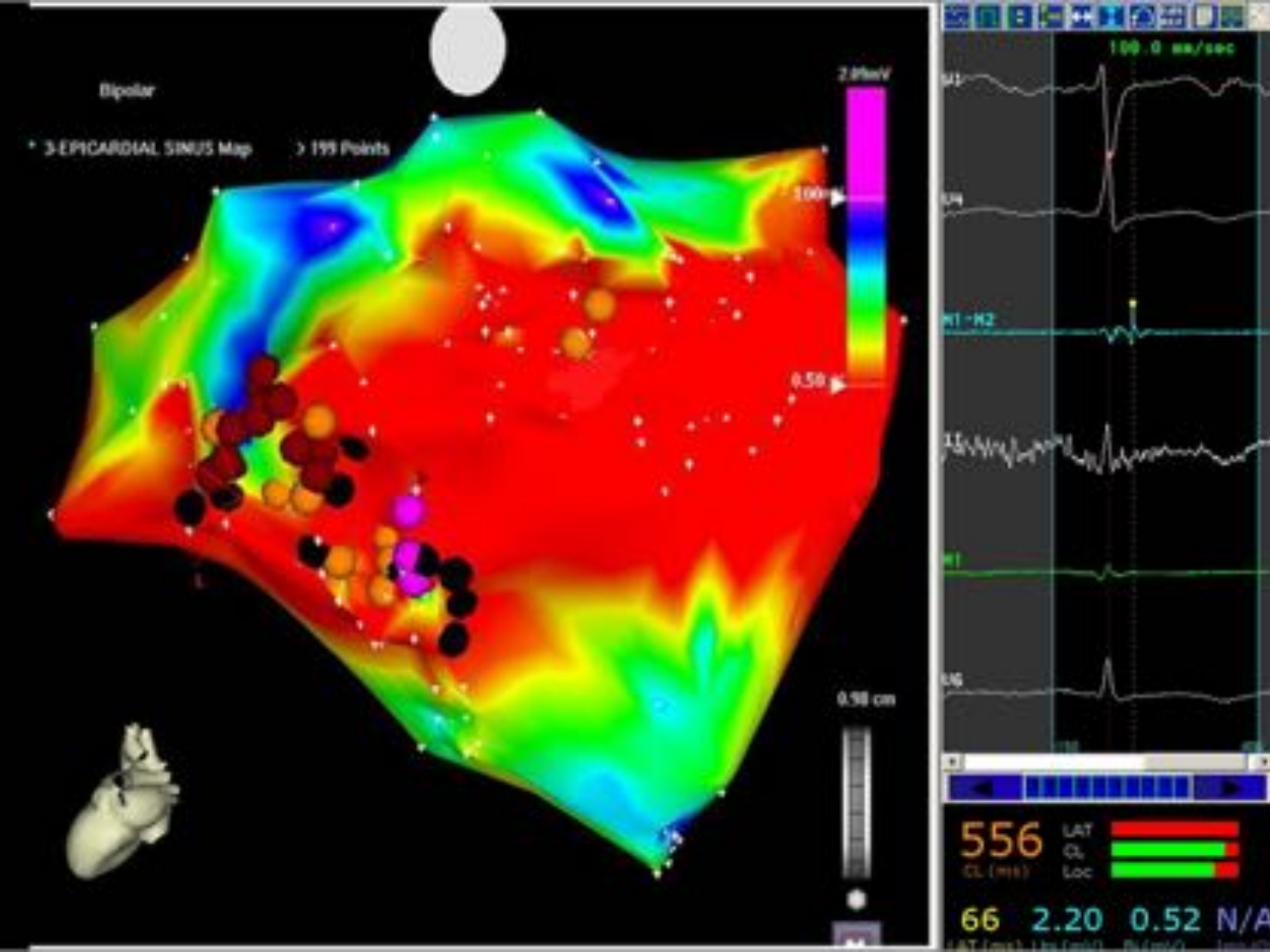
- 47 yr old male
- Diagnosed patient of cardiac sarcoidosis
- Recurrent episodes of VT, underwent ICD implantation.
- severe LV systolic dysfunction with normal coronaries
- VT storm- ICD shocks (n=50) on amiodarone, sotalol and beta blockers
- Treated with steroids and immunosuppresants

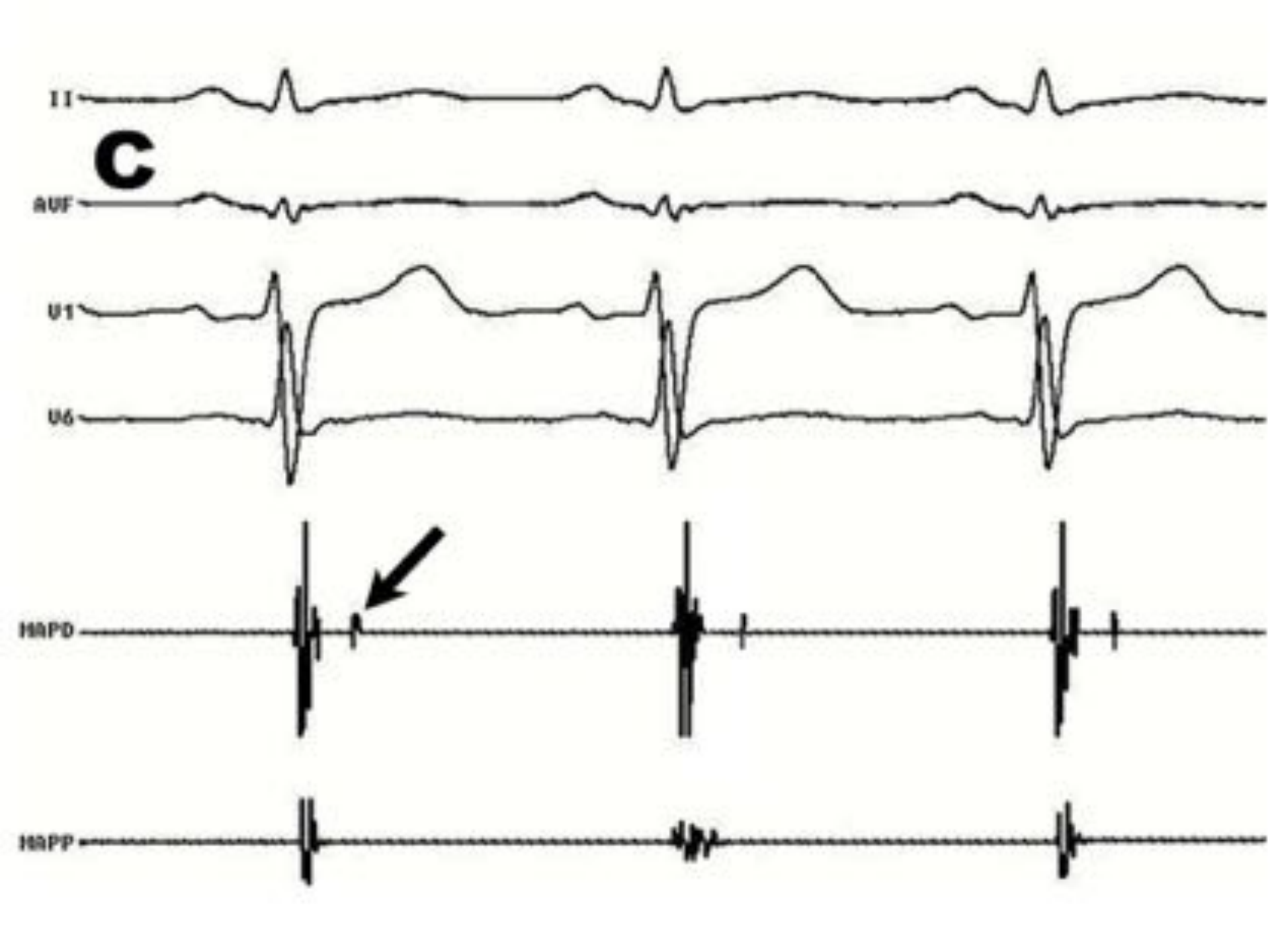
VT of posterobasal LV morphology

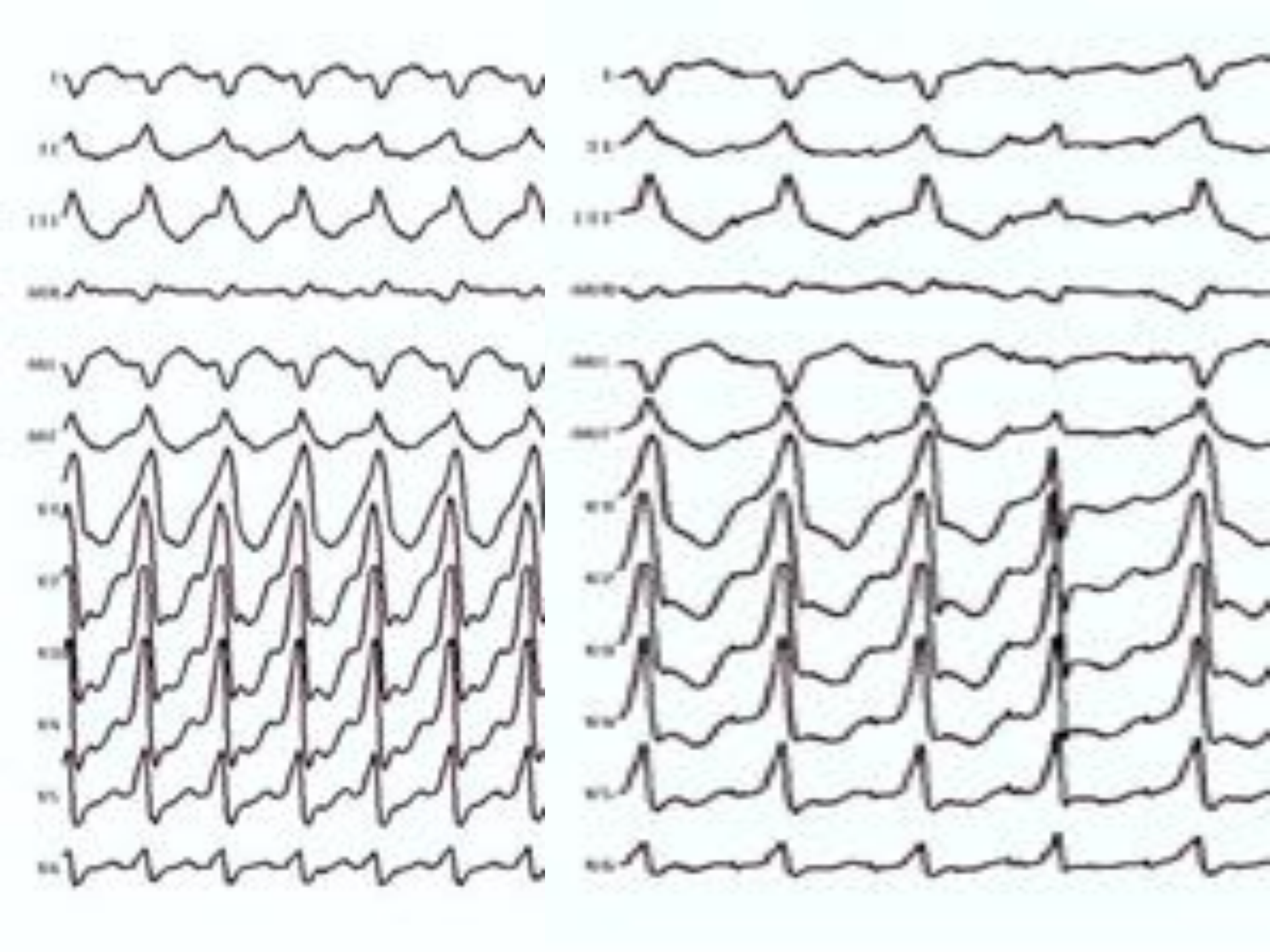


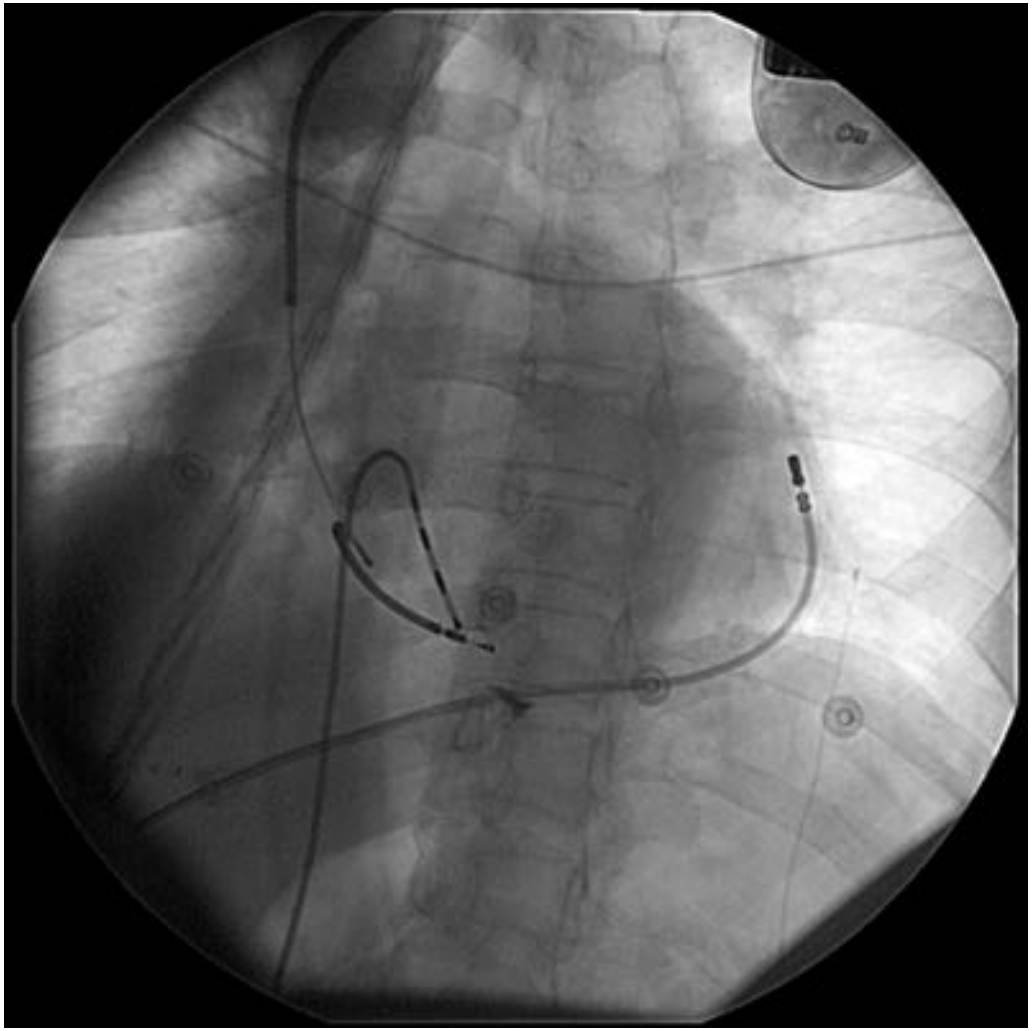

Map Viewer
LAT
2-VT Map • 159 Points
LAD
342
CL (ms)
LAT
CL
Loc
-87 14.22 5.44 N/A

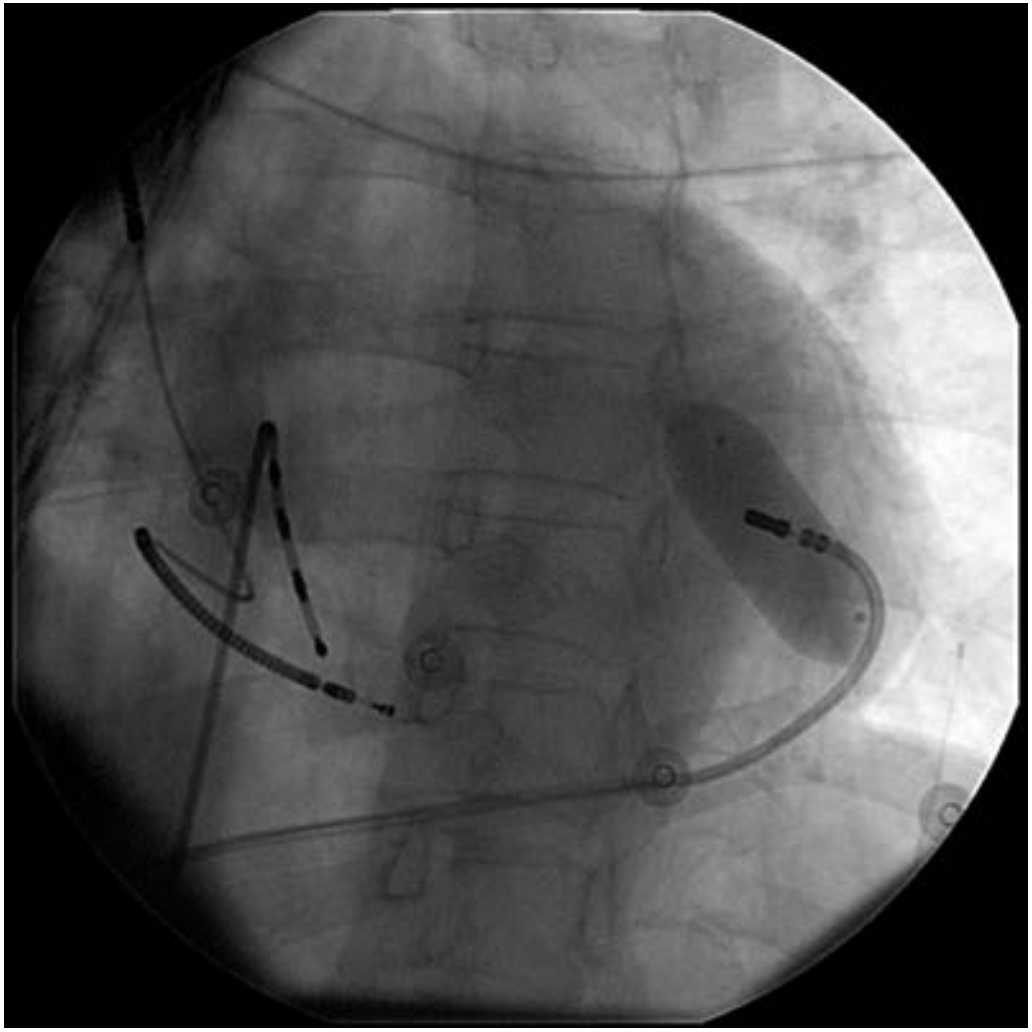

Bipolar
I-SINUS Map > 251 Points
100.0 mm/sec
603
CL (ms)
LAT
CL
Loc
25
3.60
0.39
N/A

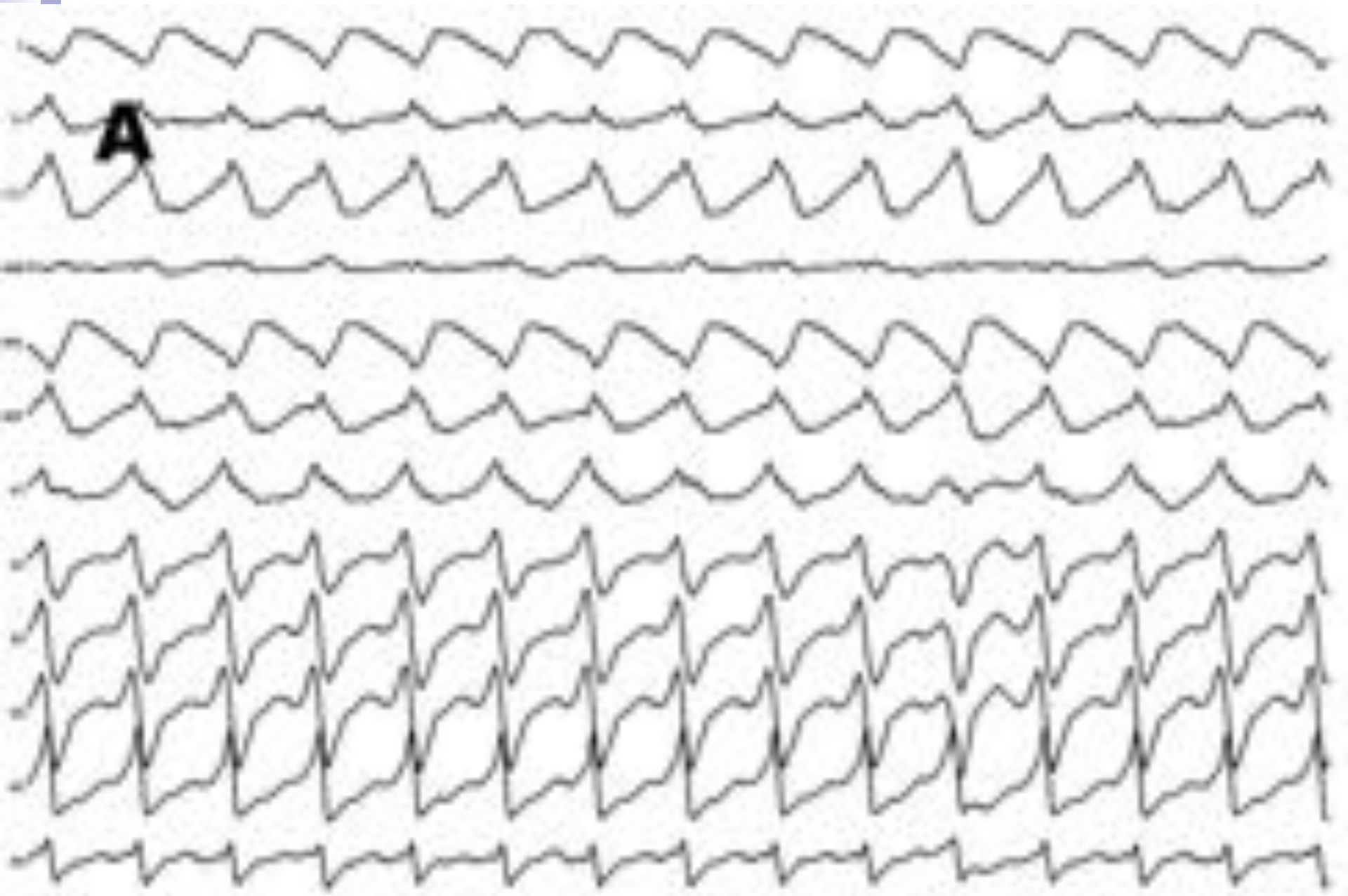

Bipolar
3-EPICARDIAL SINUS Map
199 Points
100
0.50
0.98 cm
100.0 mm/sec
556
LAT
CL
Loc
66
2.20
0.52
N/A


II
C
AVF
V1
V6
HRPD
HRPP



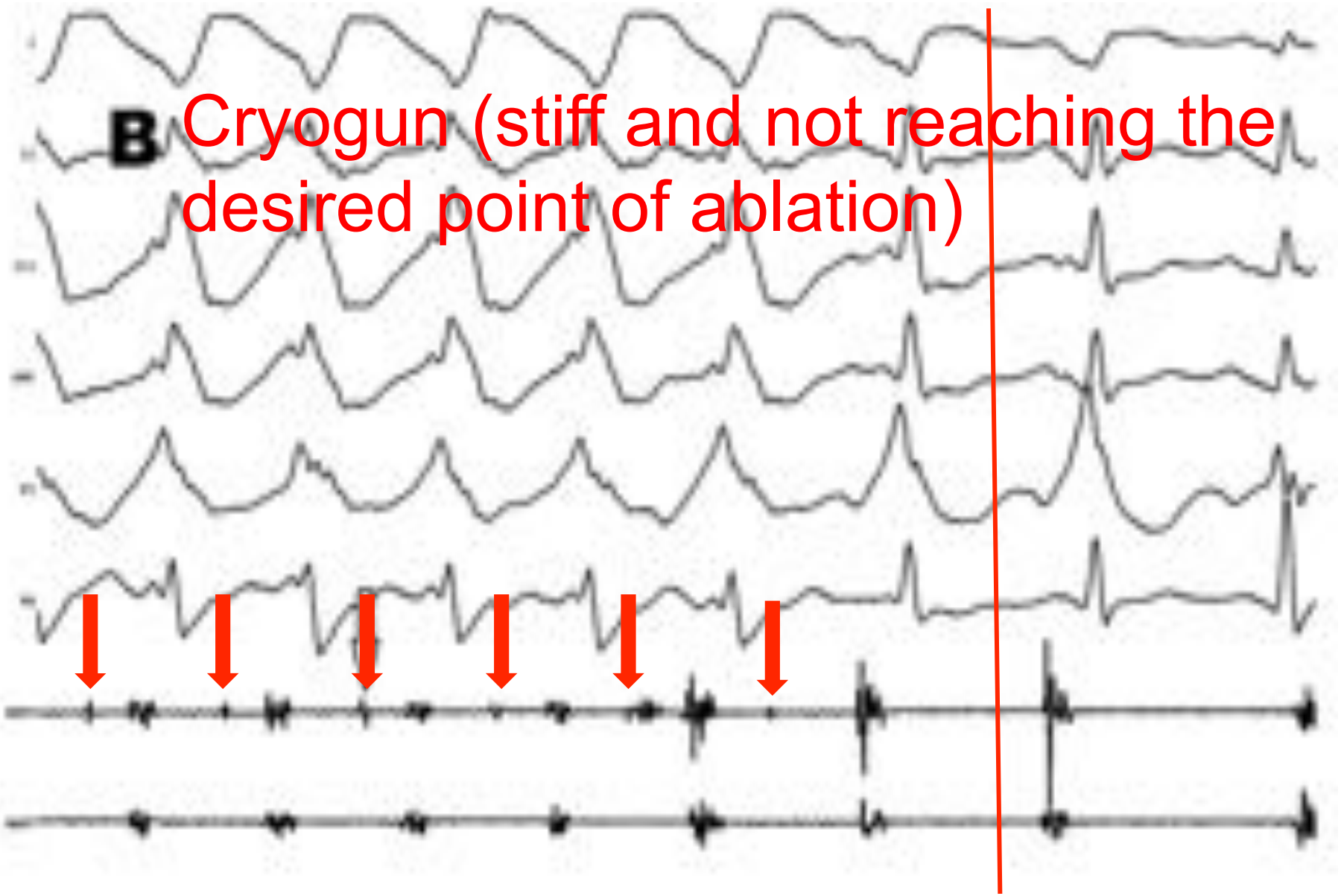




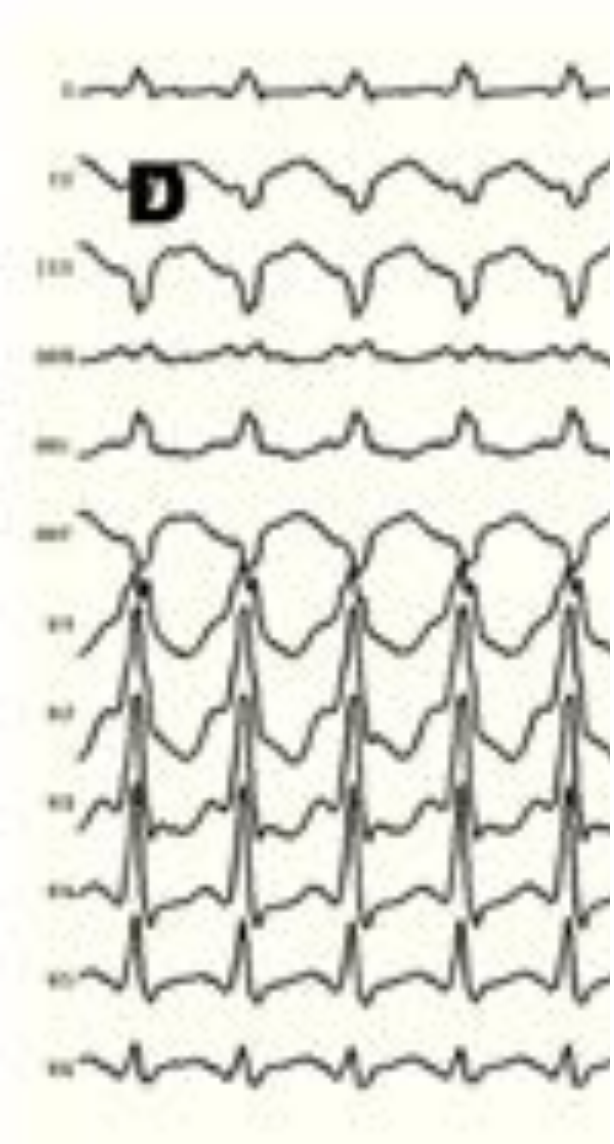

A

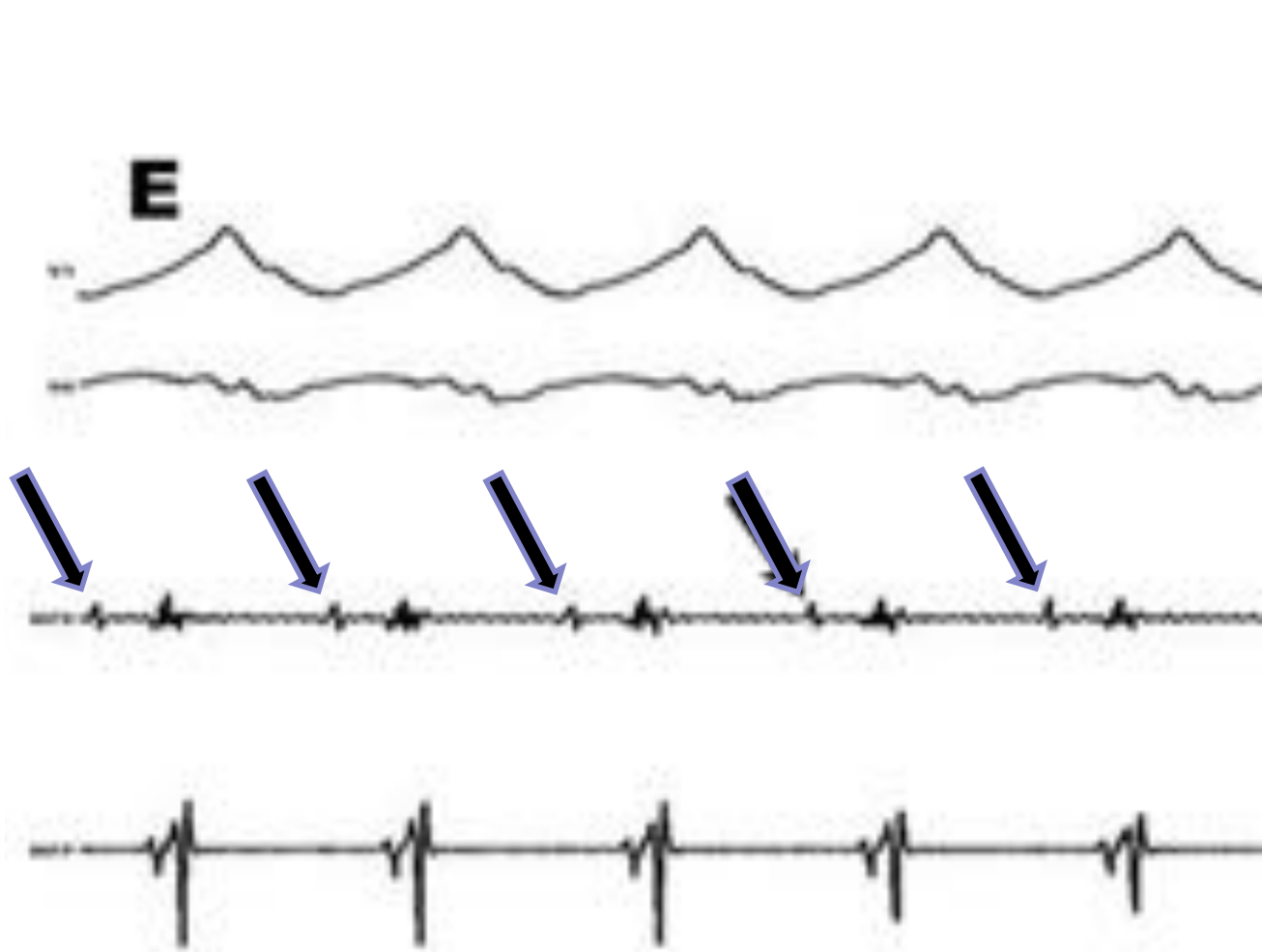
- VT of mid lateral LV morphology
- Catheter manuveribility poor due to dense adhesions in lateral wall
- Bleeding in pericardial space while breaking adhesions
- Recurrent VT (ICD shocks) after percutaneous ablation
- Left lateral thoracotomy

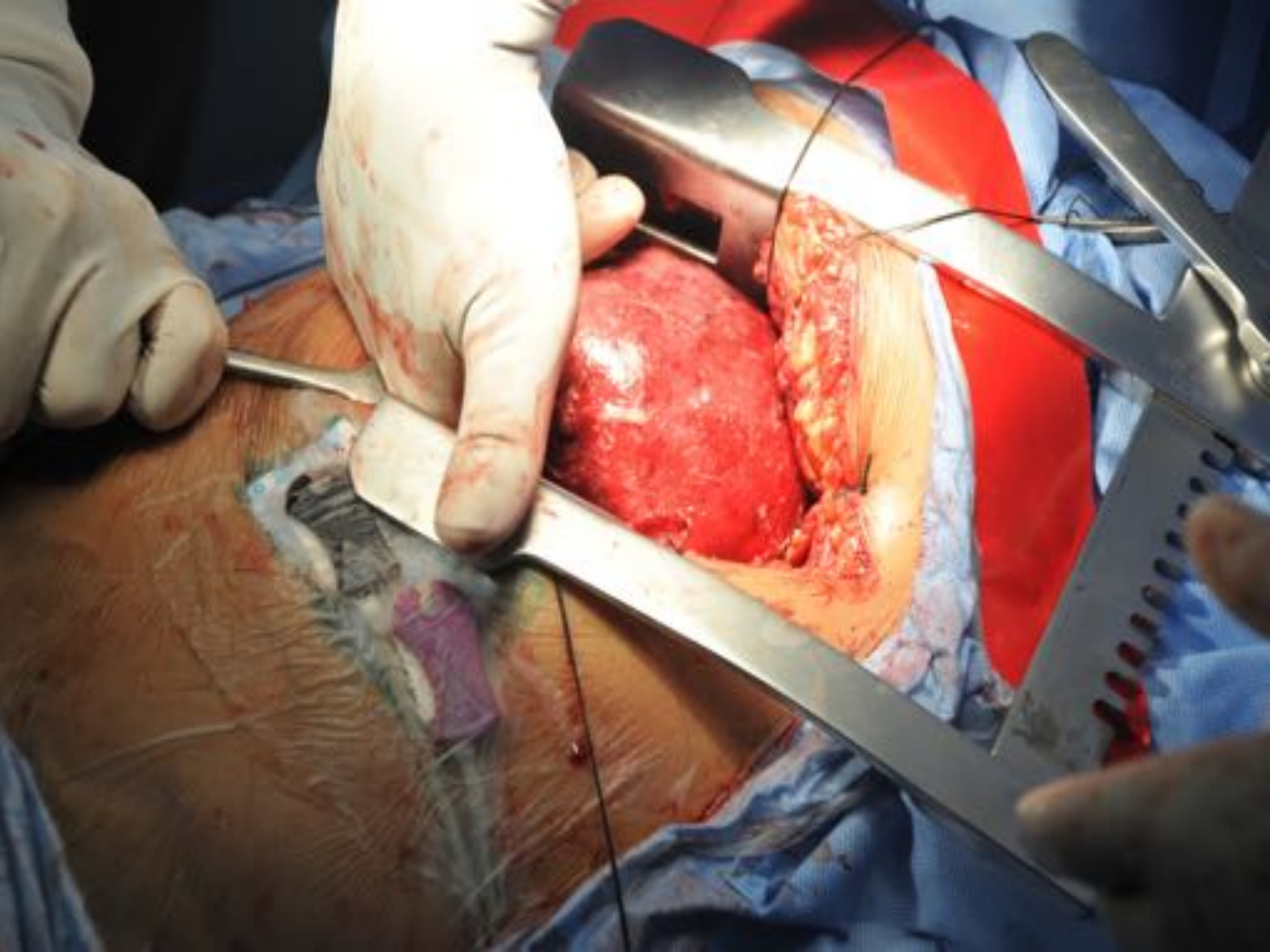
VT of mid lateral mitral annulus exit with mid diastolic potential (arrow) and termination during ablation

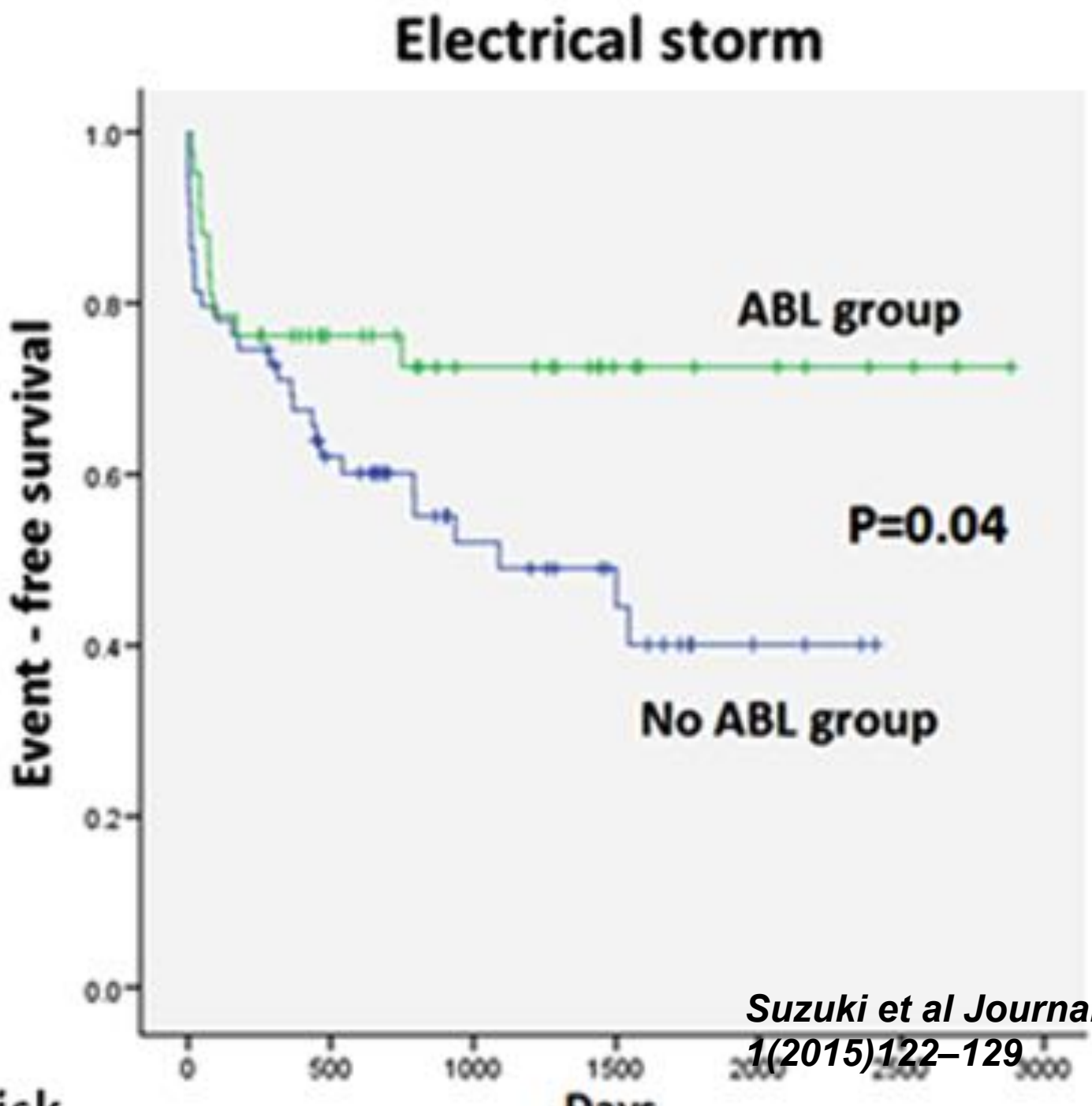


Third VT mid posterior LV morphology inducible









Suzuki et al Journal ofArrhythmia3 1(2015)122–129

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- Dr. Johann Christopher

Summary

- Characterize the substrate
- Try to induce the clinical Vt and obtain a template…
- Detailed endo / epicardial map
- Consider focal / HPS related vt

Summary

- SMVTs in CMP are usually due to reentry (from low voltage areas-ie . Scar)
- Scars are often perivalvualar , or midseptal

- Combined endocardial and epicardial mapping approaches are likely to improve the success of ablation.