Catheter ablation of VT in patients with a structural heart disease

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Specific Cardiomyopathies (1996 WHO classification)

Ischemic Cardiomyopathy

Valvular Cardiomyopathy

Hypertensive Cardiomyopathy

Inflammatory Cardiomyopathy

Metabolic Cardiomyopathy

General system disease

Muscular dystrophies

Neuromuscular disorders

Sensitivity and toxic reactions

Peripartal Cardiomyopathy

Clinical VT inducible

Ischemic Cardiomyopathy

Dilated Cardiomyopathy

Arrhythmogenic Right Ventricular Cardiomyopathy

GUIDELINES

VT ablation in patients with a structural heart disease

ACC/AHA/ESC PRACTICE GUIDELINES

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death

Class I	Ablation is indicated in patients with bundle-branch reentrant VT. *(Level of Evidence: C)*
	Ablation is indicated as adjunctive therapy in patients with an ICD who are receiving multiple shocks as a result of sustained VT that is not manageable by reprogramming or changing drug therapy or who do not wish long-term drug therapy (59,60). *(Level of Evidence: C)*
Class IIa	Ablation can be useful as adjunctive therapy in management of patients with arrhythmogenic RV cardiomyopathy with recurrent VT, despite optimal antiarrhythmic drug therapy. *(Level of Evidence: C)*

Zipes DP, Camm AJ et al., J Am Coll CArdiol 2006;48:1064-1108

GUIDELINES

ICD implantation in patients with VT & heart disease

Situation clinique	Classe	Preuve
Arrêt cardiaque par FV ou TV, sans cause aiguë ou réversible	I	A
Patients coronariens sans ou avec symptômes d'insuffisance cardiaque légère ou modérée (classe NYHA II ou III), une FEVG $\leq$ 30 % mesurée au moins 1 mois après un IDM et 3 mois après un geste de revascularisation (chirurgie ou angioplastie)	I	B
TV soutenue spontanée symptomatique sur cardiopathie	I	B
TV soutenue spontanée, mal tolérée ,en l'absence d'anomalie cardiaque , pour laquelle un traitement médical ou une ablation ne peut être réalisée ou a échoué	I	B
Syncope de cause inconnue avec TV soutenue ou FV déclenchable ,en présence d'une anomalie cardiaque sous-jacente.	I	B

Indications de l'implantation d'un défibrillateur - Recommandations françaises
Mise à jour: 21 janvier 2006
Aliot E et al. Arch Mal Cœur 2006;99:141-54

Patients coronariens, aux antécédents d'infarctus, avec dysfonction ventriculaire gauche (fraction d'éjection 31 à 35 %)	IIb	C
Patients atteints de cardiopathie dilatée en apparence primitive, avec dysfonction ventriculaire gauche (fraction d'éjection de 31 à 35 %) et une classe NYHA II ou III	IIb	C
TV soutenue mal tolérée chez un patient en attente de transplantation cardiaque	IIb	C
Syncope (s) de cause inconnue sans trouble du rythme déclenchable	III	C
TV ou FV incessantes malgré le traitement	III	C
TV ou FV curables par chirurgie ou ablation, ne mettant pas en jeu le pronostic vital (ex : TV fasciculaires, TV infundibulaires…)	III	C
TV ou FV dues à des causes aiguës ou réversibles (ex : ischémie, hypokaliémie…)	III	C
TV ou FV et maladie mentale susceptible d'être aggravée par l'implantation ou d'empêcher le suivi	III	C
Arrêt circulatoire par TV ou FV avec séquelles neurologiques graves	III	C
TV ou FV et maladie terminale avec espérance de vie de moins d'un an	III	C
TV ou FV et insuffisance cardiaque terminale chez un patient non candidat à la transplantation	III	C

Indications de l'implantation d'un défibrillateur - Recommandations françaises
Mise à jour: 21 janvier 2006
Aliot E et al. Arch Mal Cœur 2006;99:141-54

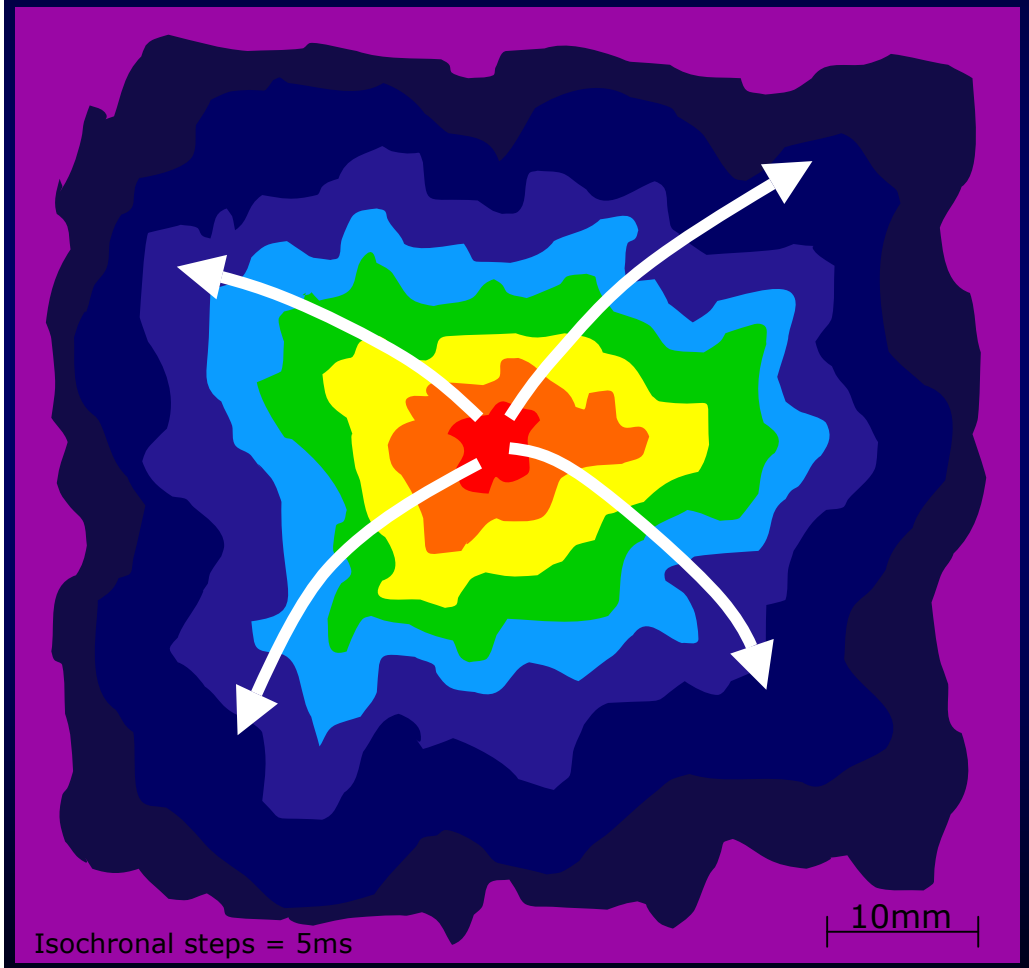
CONVENTIONAL MAPPING

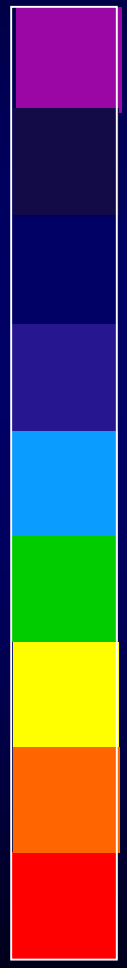
Activation Mapping

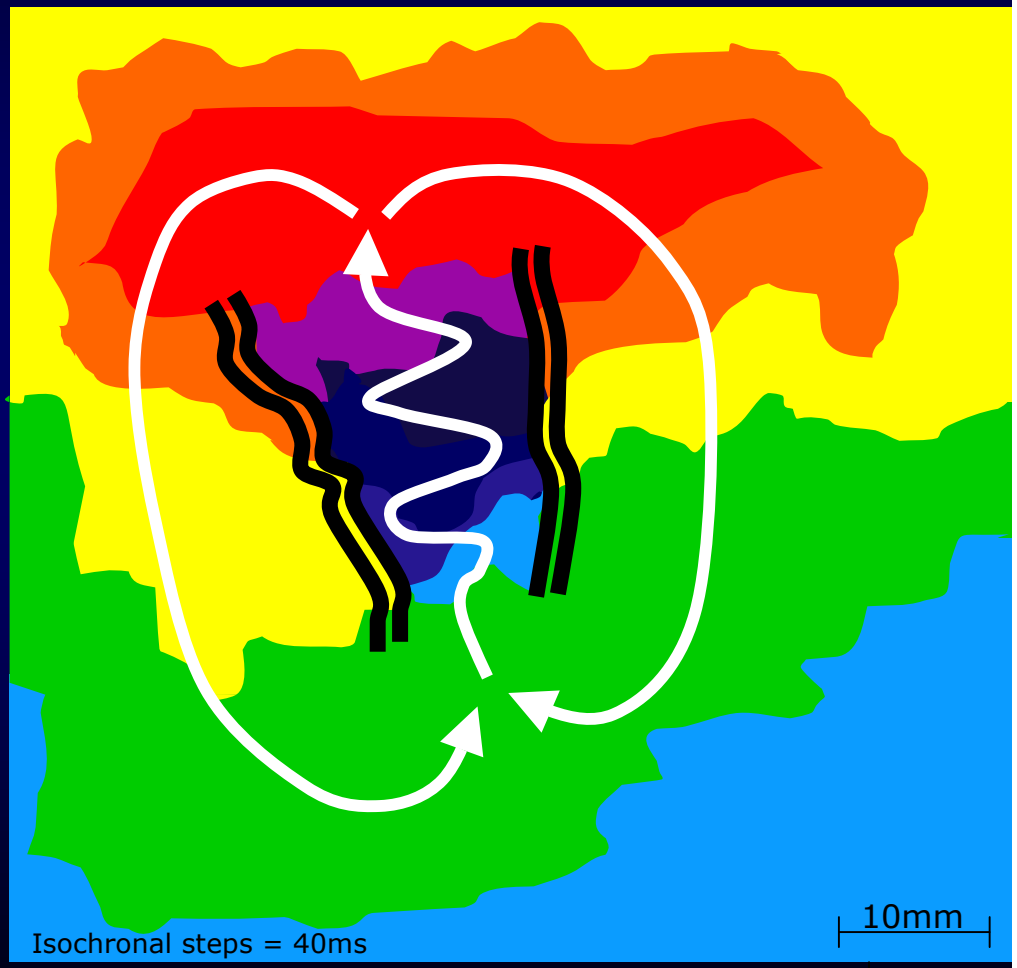
Entrainment & PPI

Pace Mapping

Endocardial mapping during VT
➔ color-coded isochronal maps



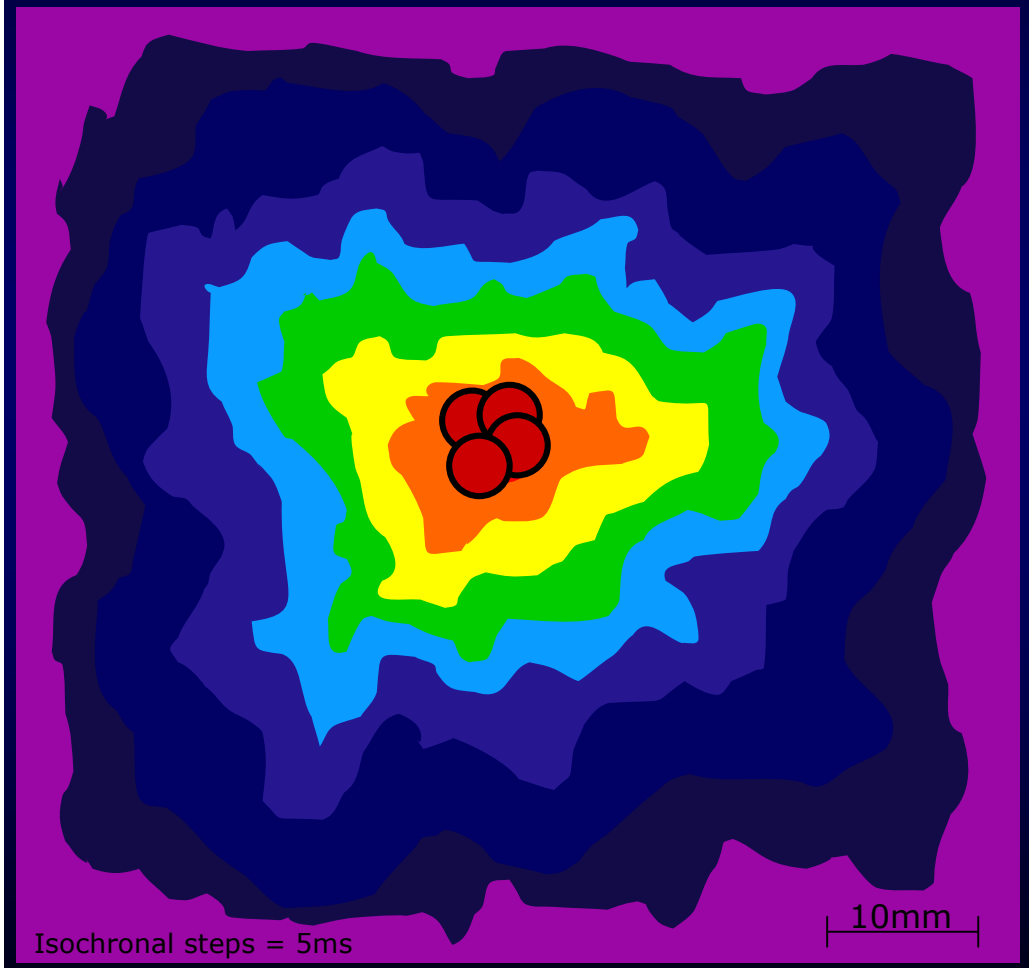


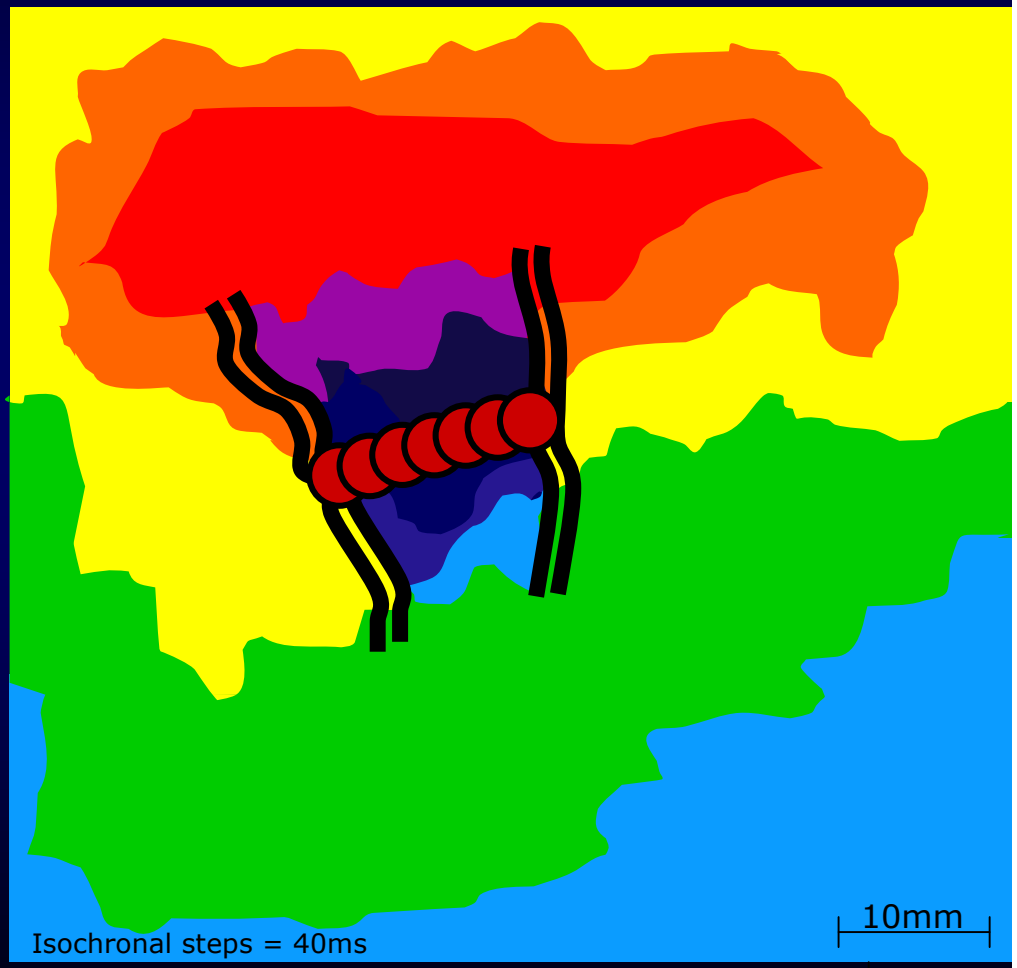


Focal VT pattern

Reentrant VT pattern

What is the ablation target ?

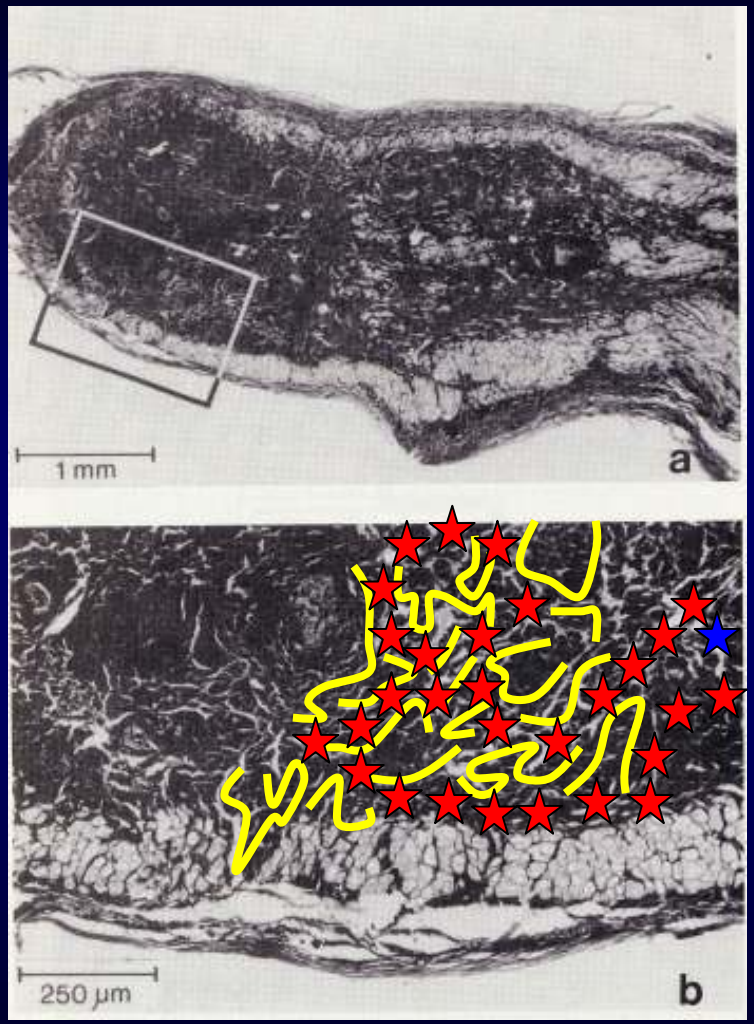


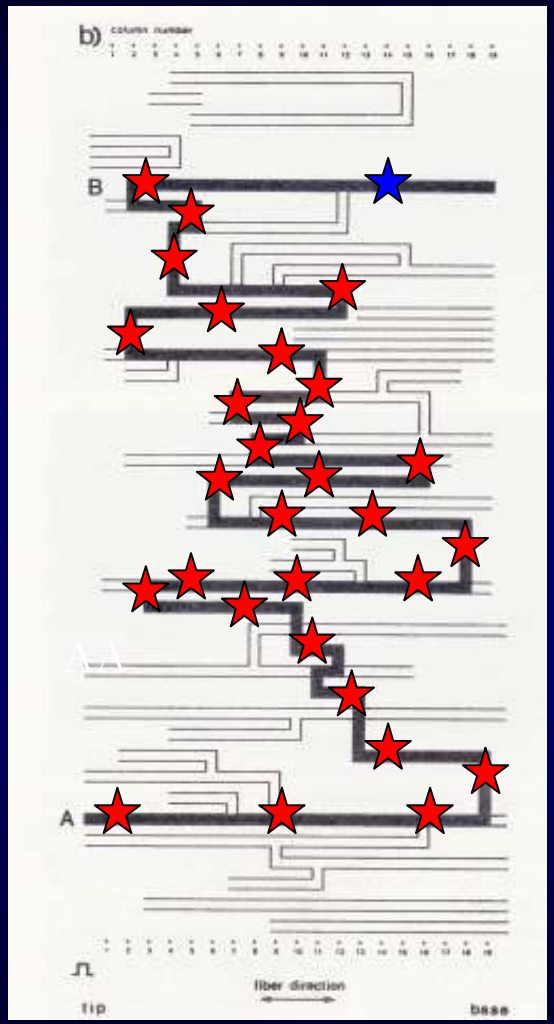


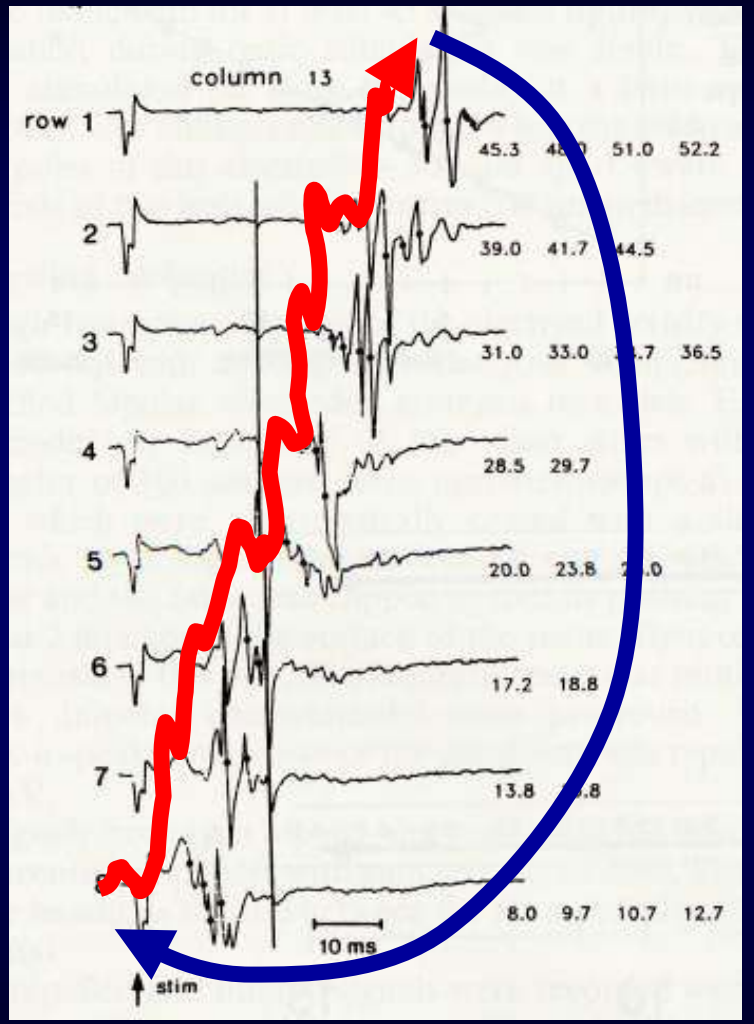
Focal VT pattern

Reentrant VT pattern

Fibrosis & Slow Conduction







Slow conduction perpendicular to the fiber direction in infarcted myocardial tissue is caused by a ''zigzag'' course of activation at high speed. Activation proceeds along pathways lengthened by branching and merging bundles of surviving myocytes unsheathed by collagenous septa.

de Bakker JMT. et al. Circulation 1993;88:915-26

VT mechanism in relation with the underlying heart disease

	No Structural Heart Disease	Structural Heart Disease
Fibrosis ➔ VT substrate	No	Yes
Endocardial Mapping		
Focal VT	>90%	<10%
Reentrant VT	<10% (ILVT)	>90%

Actual significance of the VT mechanism showed by mapping

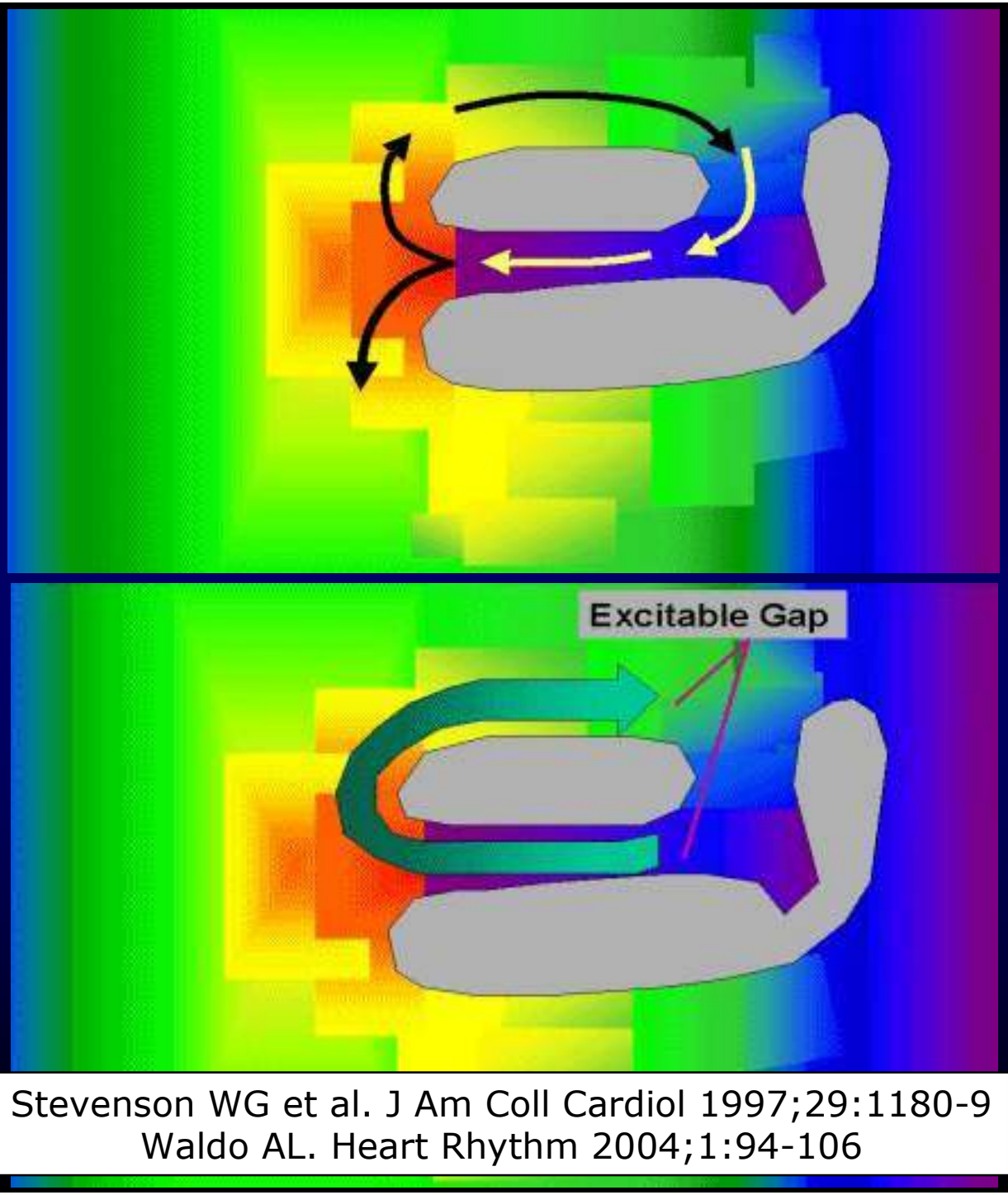
No Structural Heart Disease : focal is really focal !

Structural Heart Disease : a ‘true’ focal mechanism is actually rare (10-15%) !

Activation Mapping

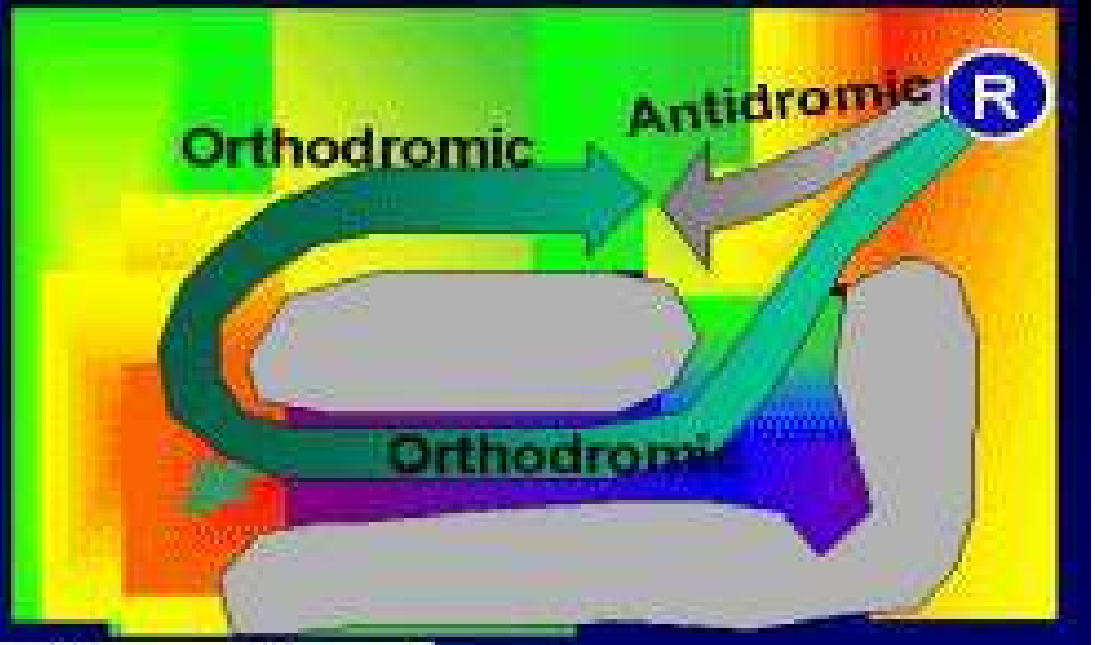
Entrainment & PPI

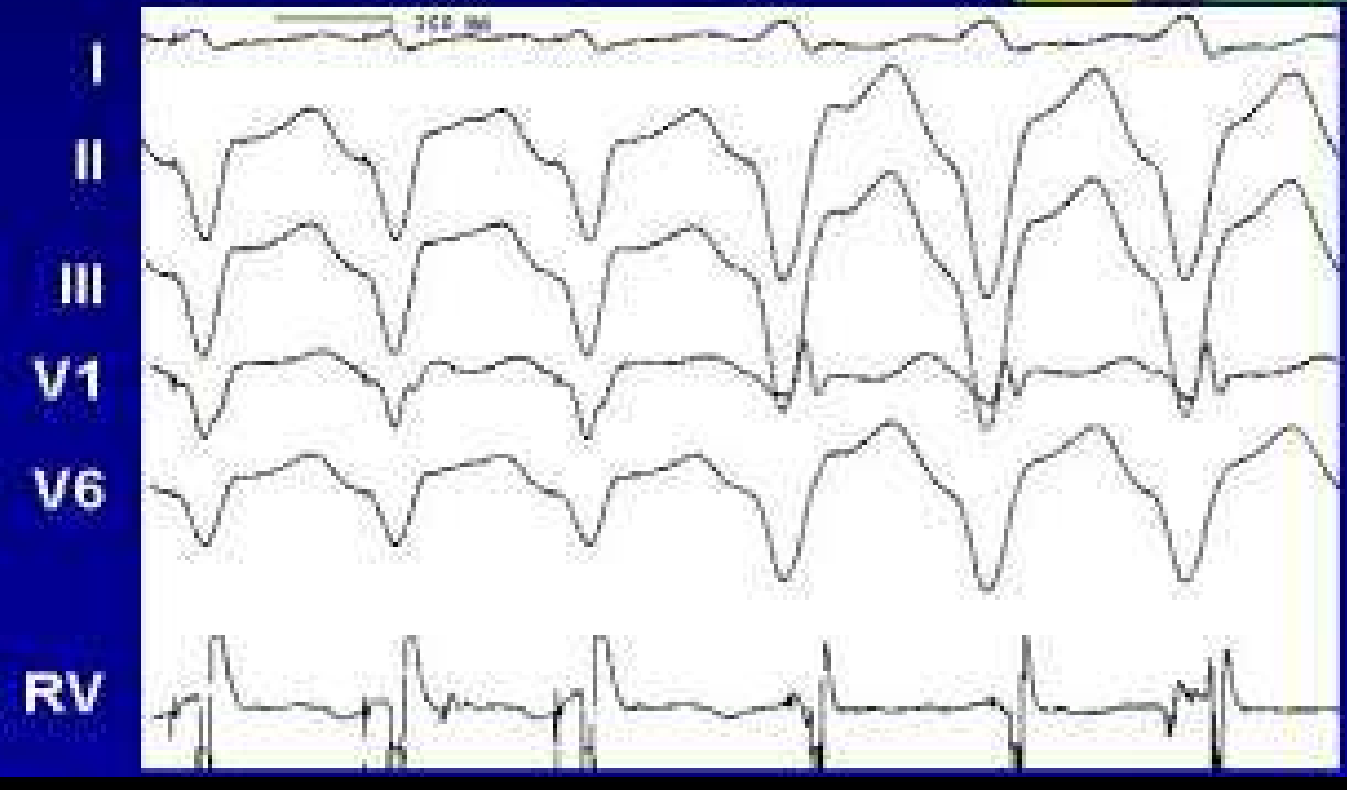
Pace Mapping



Stevenson WG et al. J Am Coll Cardiol 1997;29:1180-9
Waldo AL. Heart Rhythm 2004;1:94-106

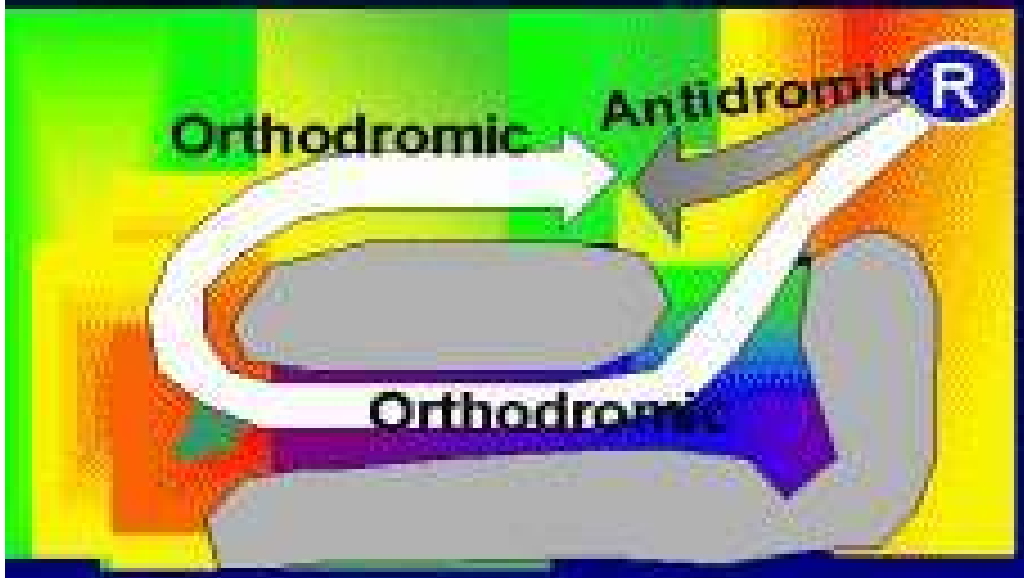
Entrainment

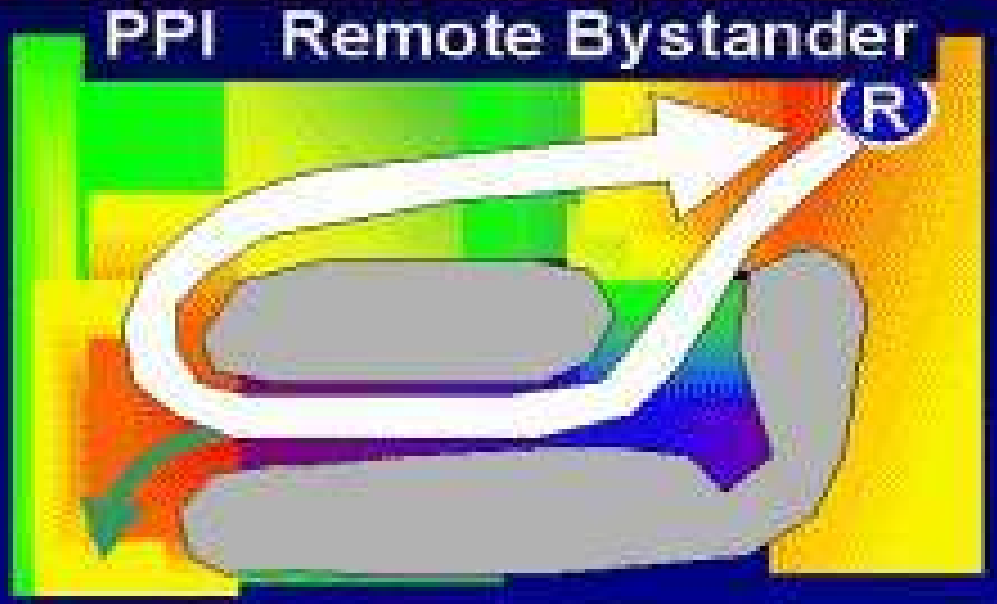


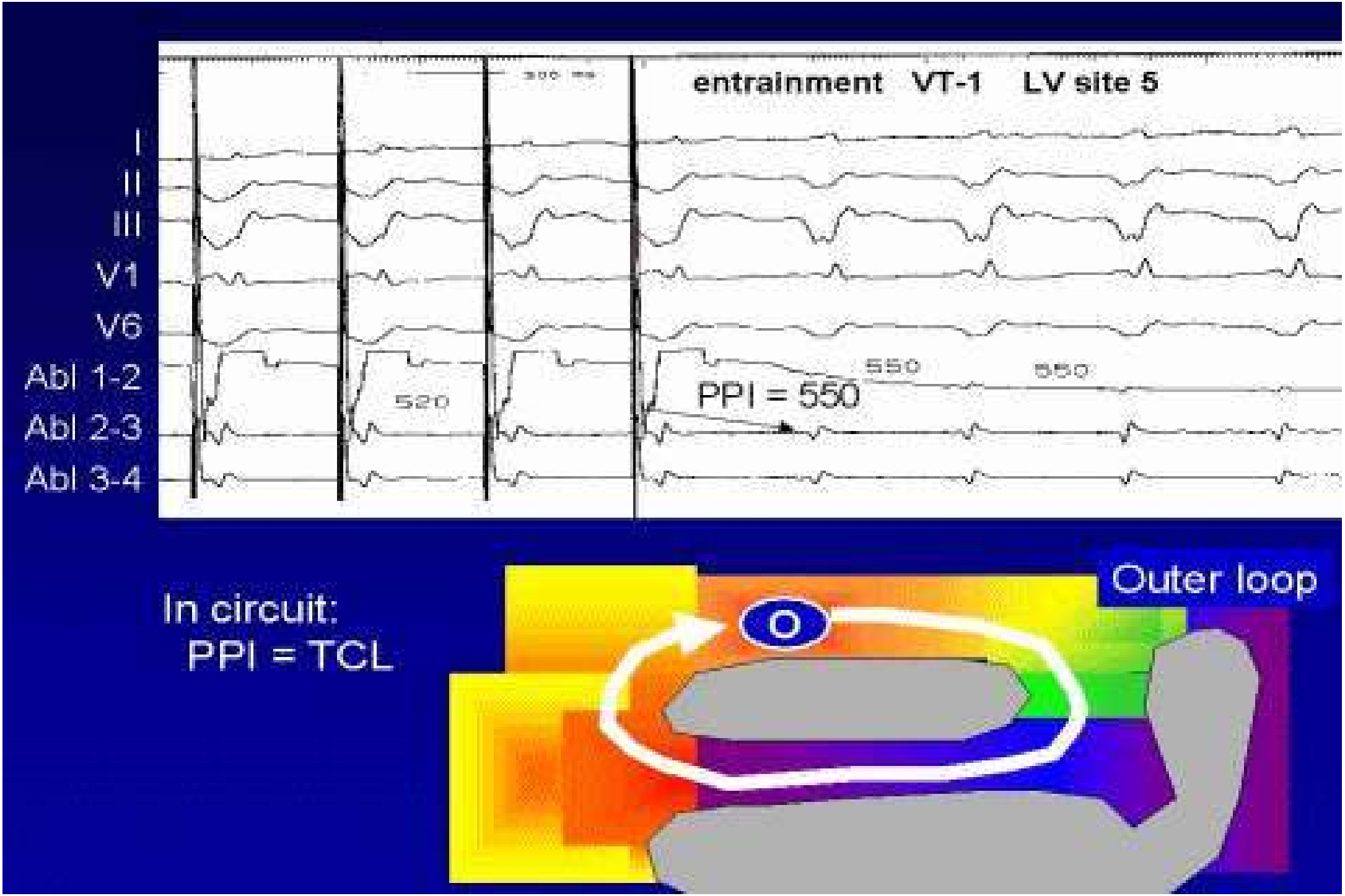


Is the site in the circuit? post pacing interval

PPI = conduction time from pacing site to circuit
revolution time through circuit
conduction time from circuit back to pacing site

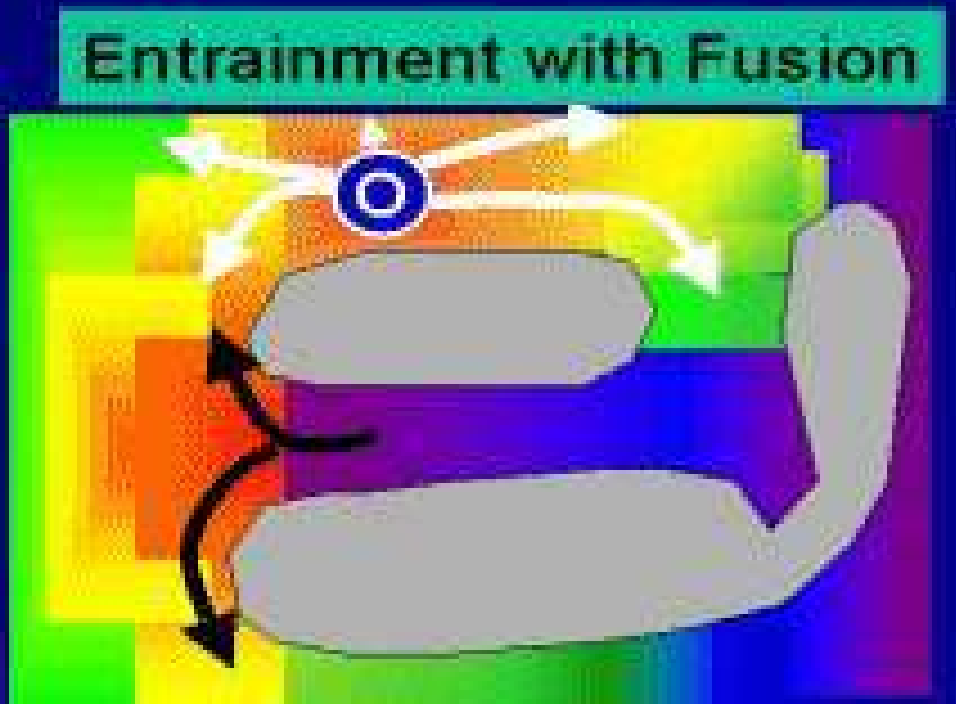


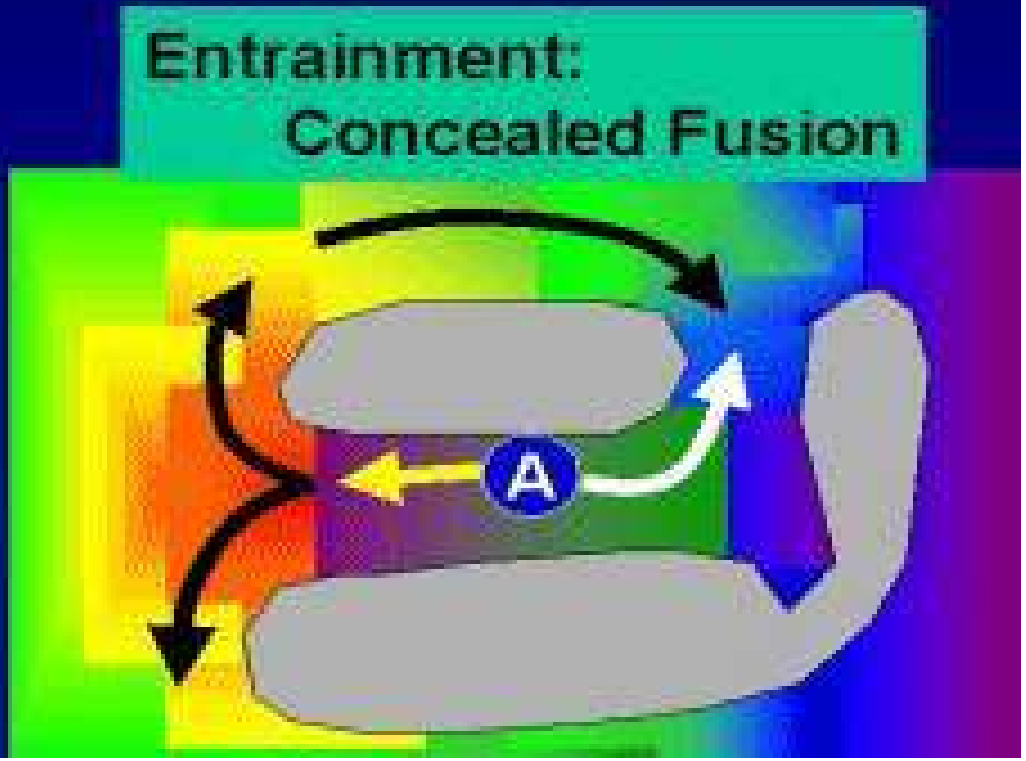



entrainment VT-1 LV site 5
I
II
III
V1
V6
Abl 1-2
Abl 2-3
Abl 3-4
520
550
550
PPI = 550
In circuit:
PPI = TCL
Outer loop
O

Reentry Circuit Site:
Broad Loop versus
narrow isthmus (channel)

Fusion during entrainment
- overt
- concealed





Concealed entrainment

Activation Mapping

Entrainment & PPI

Pace Mapping

‘Conventional Electrophysiology’ Tools

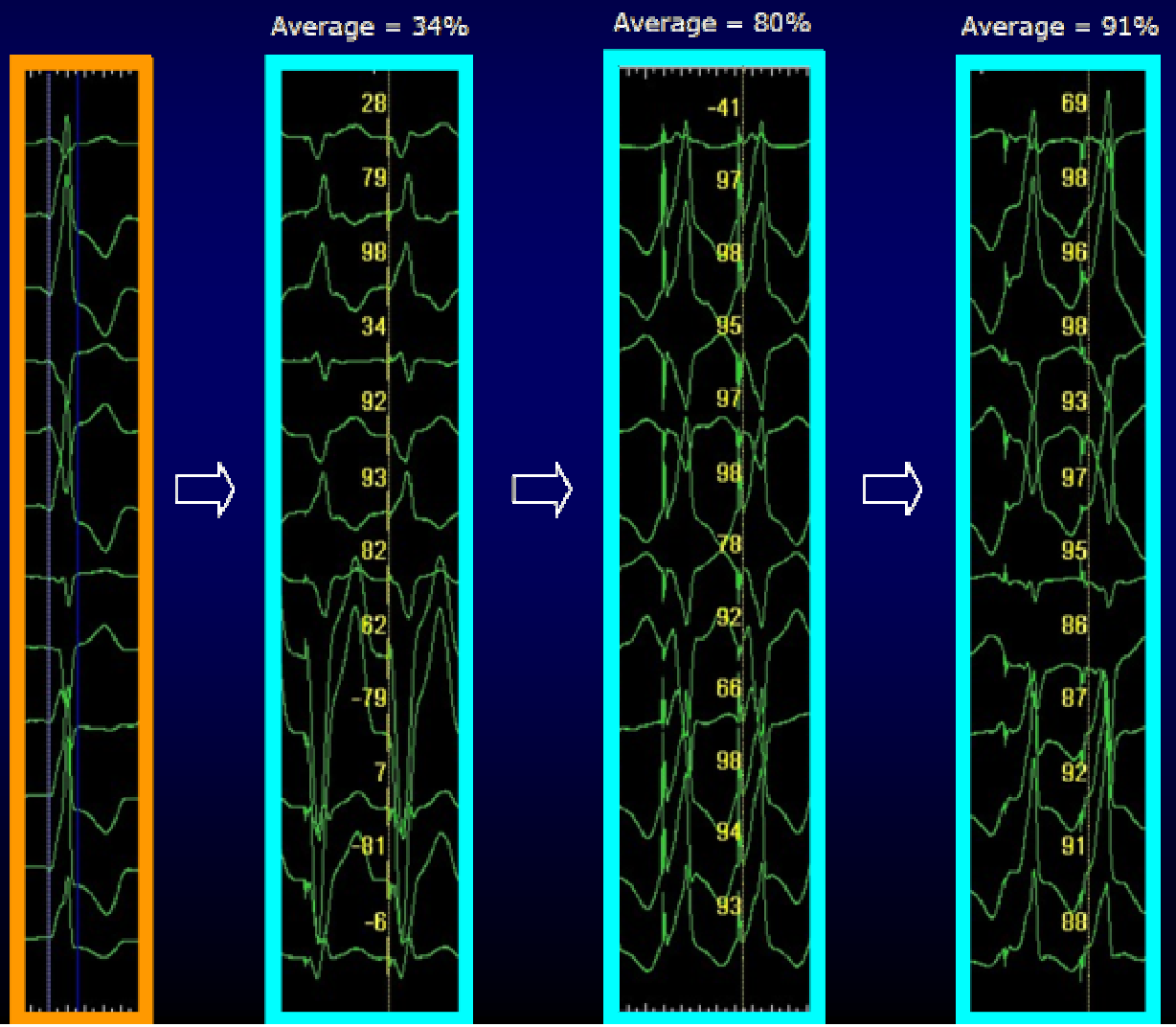
Pace mapping during sinus rhythm

Online comparison analysis with the BARD template matching algorithm

Reference QRS complex on the 12-lead ECG

$$\mathrm{CORR} = \frac{\sum_{\text{Lead 1}}^{12}\left[\sum_{i=1}^{n}(X_i - \overline{X}) \times (Y_i - \overline{Y})\right]}{\sum_{\text{Lead 1}}^{12}\left[\sqrt{\sum_{i=1}^{n}(X_i - \overline{X})^2 \times \sum_{i=1}^{n}(Y_i - \overline{Y})^2}\right]}$$

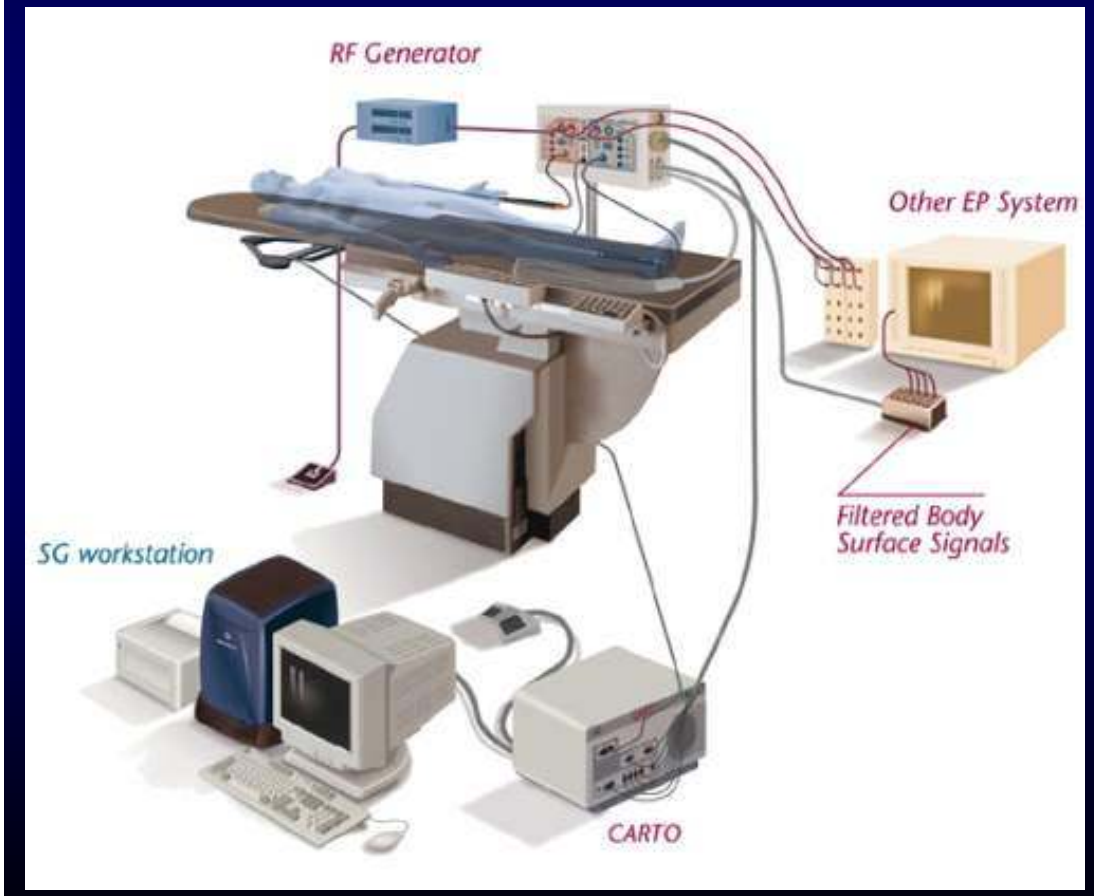
Gerstenfeld EP et al. J Am Coll Cardiol 2003;41:2046-53

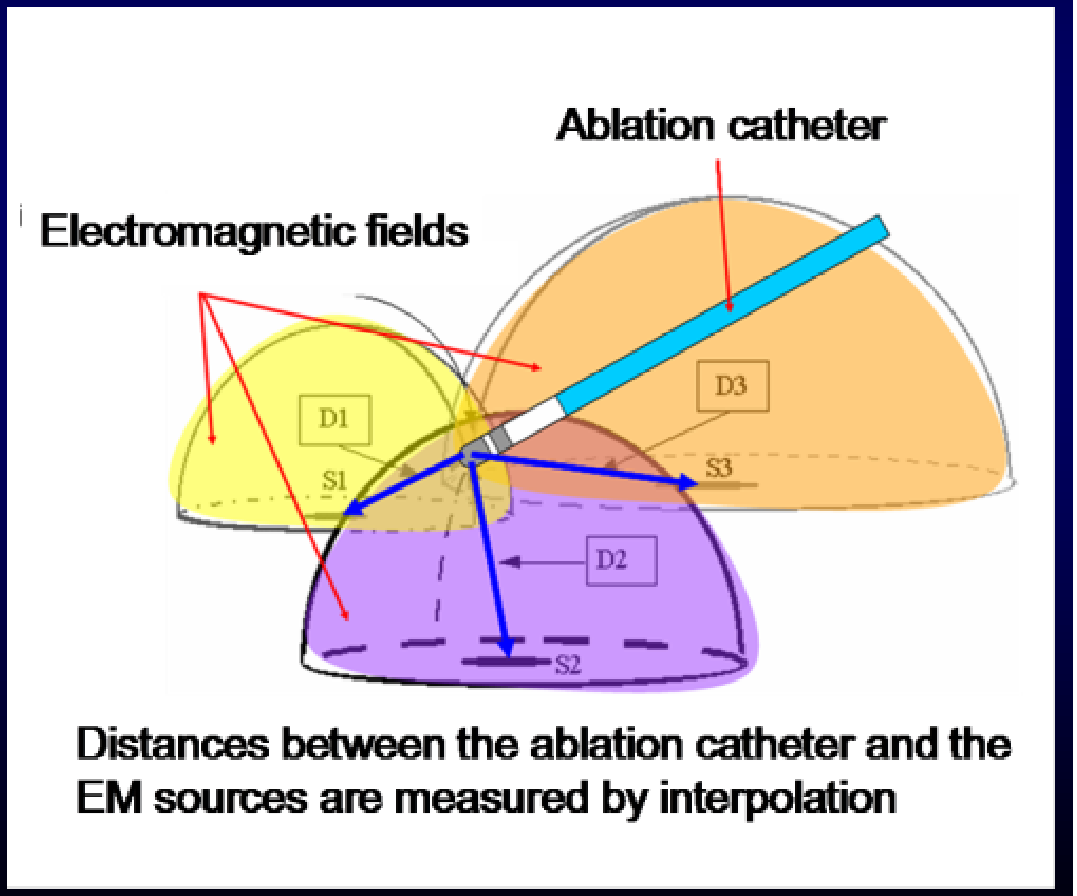


'HIGH-TECH' MAPPING

‘High-Tech’ Tools

3D mapping systems



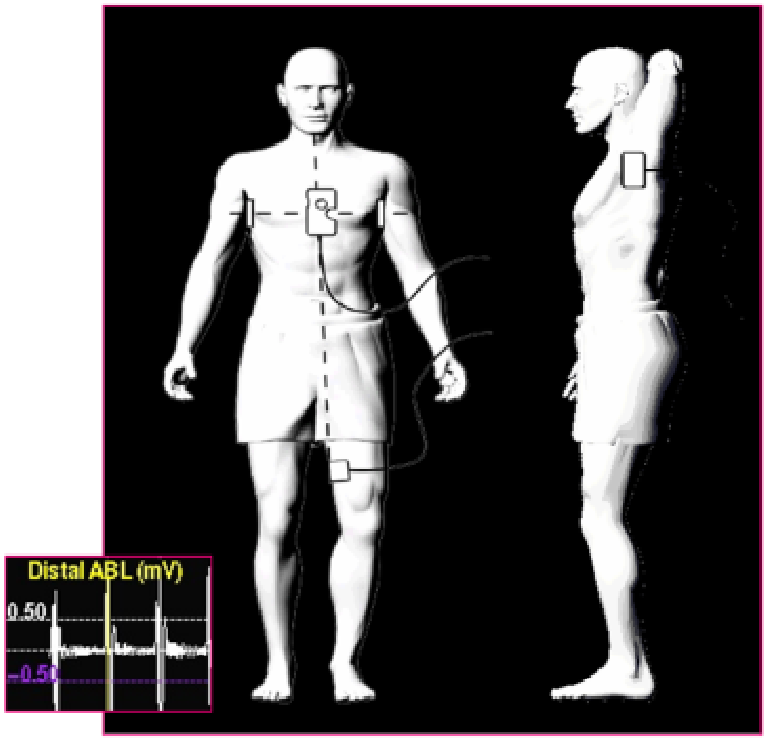


Distances between the ablation catheter and the EM sources are measured by interpolation

‘High-Tech’ Tools

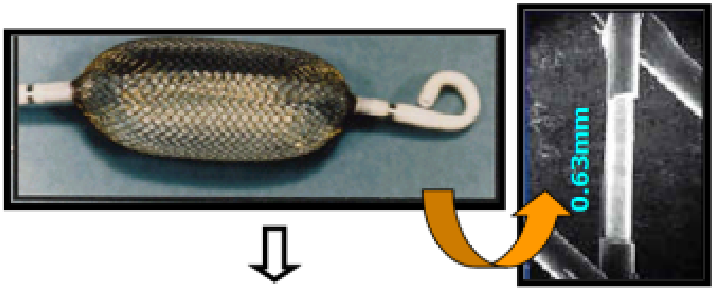
3D mapping systems

NavX® Methodology

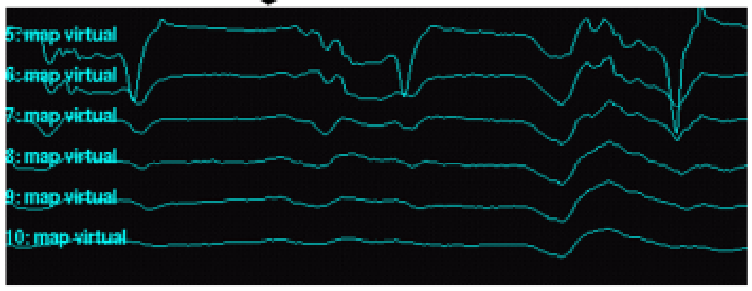


Three orthogonal electrical fields (X, Y and Z axis) → 3D location of electrodes and catheters

Ensite® Methodology



$$\int\int_{\partial D}\left[v\frac{\partial w}{\partial n}-w\frac{\partial v}{\partial n}\right]dA=\int\int\int_{D}[v\nabla^2 w-w\nabla^2 v]dD$$



Virtual EGM

	Without a 3D mapping system	With a 3D mapping system
Navigation		
Real-time positioning		
Mapping & Ablation		

CONTRIBUTION OF THE 12-LEAD ECG TO LOCALIZE VT ORIGIN

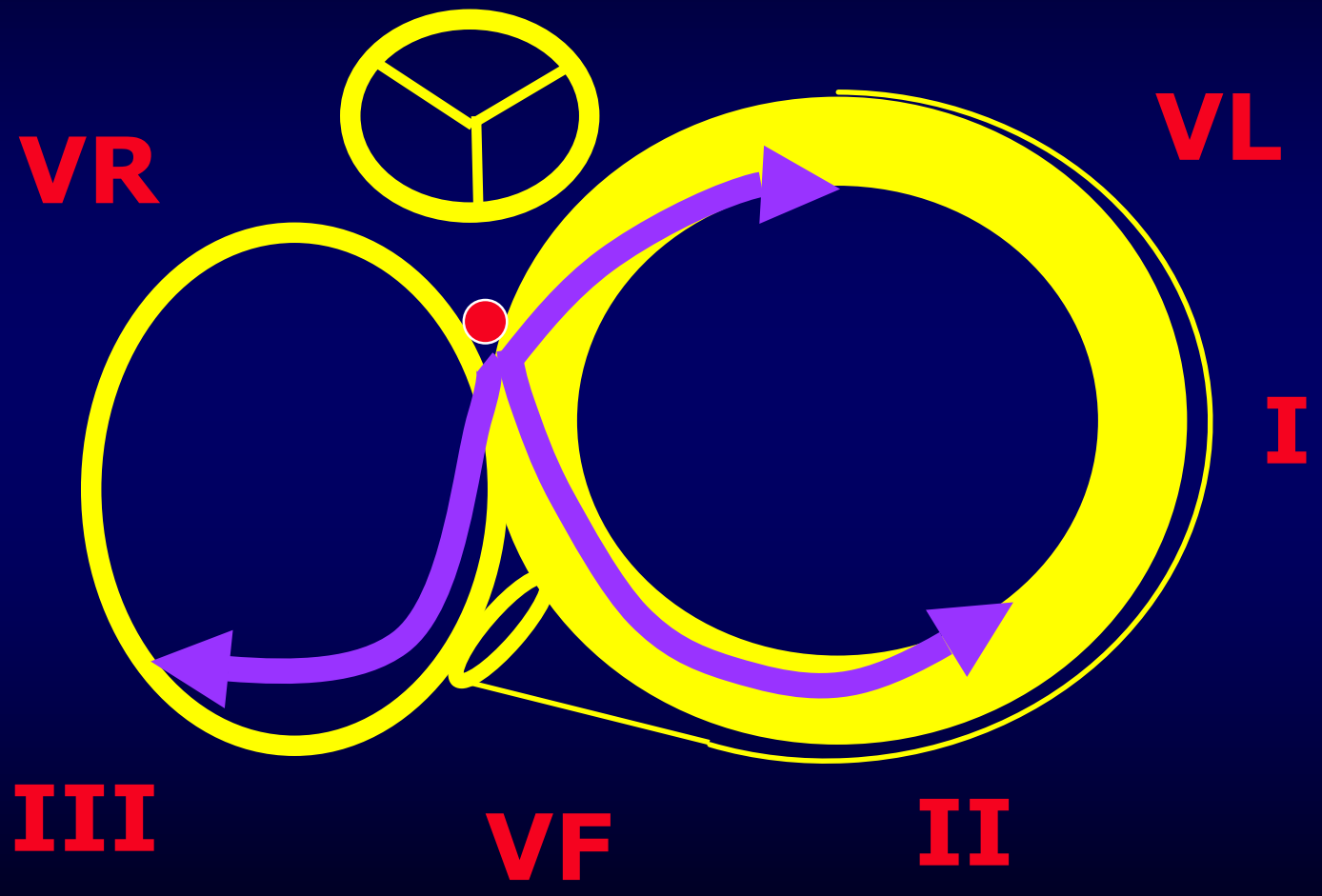
Pour quoi faire ?

Site d'origine de la TV # zone arythmogène
Zone arythmogène = zone saine ?
Corrélation avec cardiopathie sous-jacente

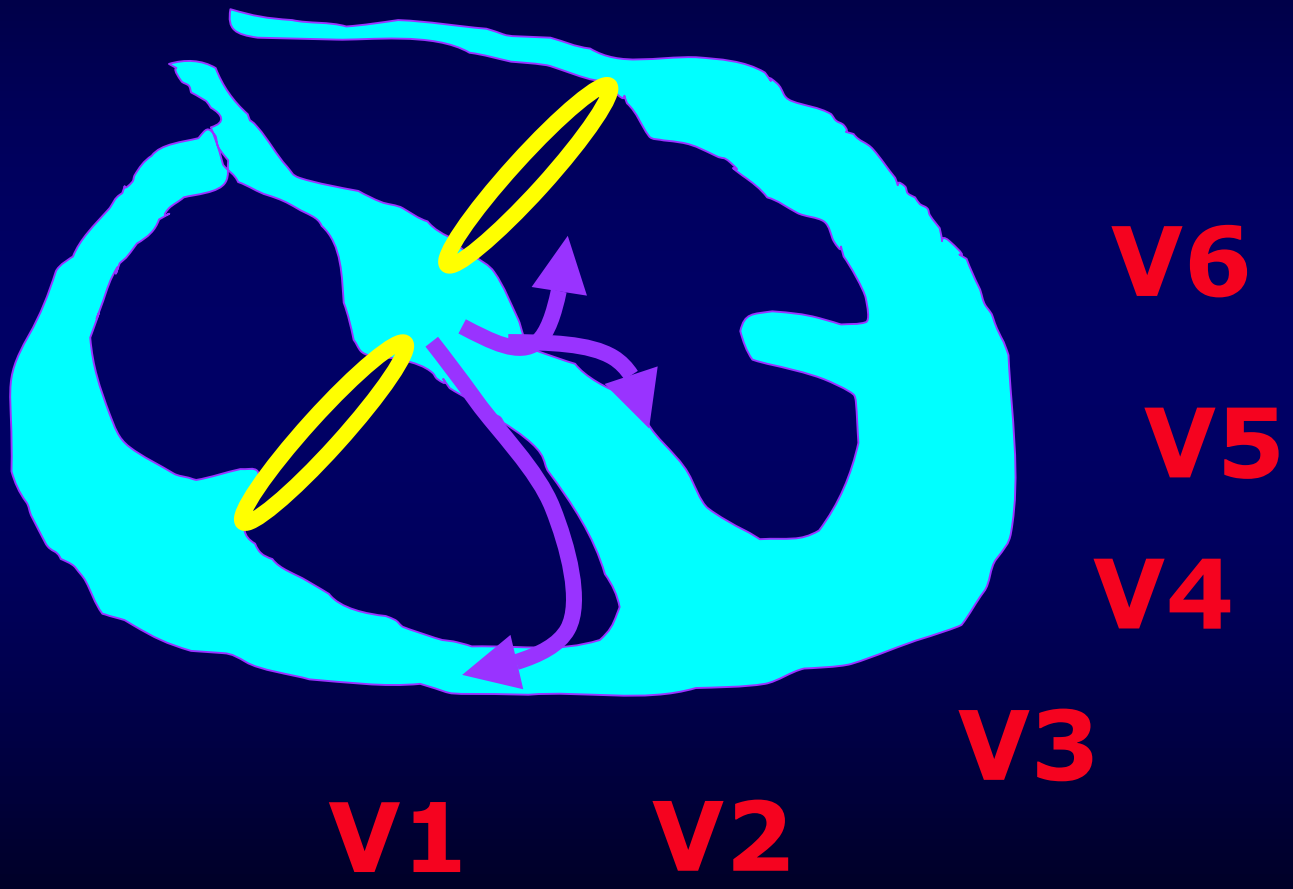
Comment faire ?

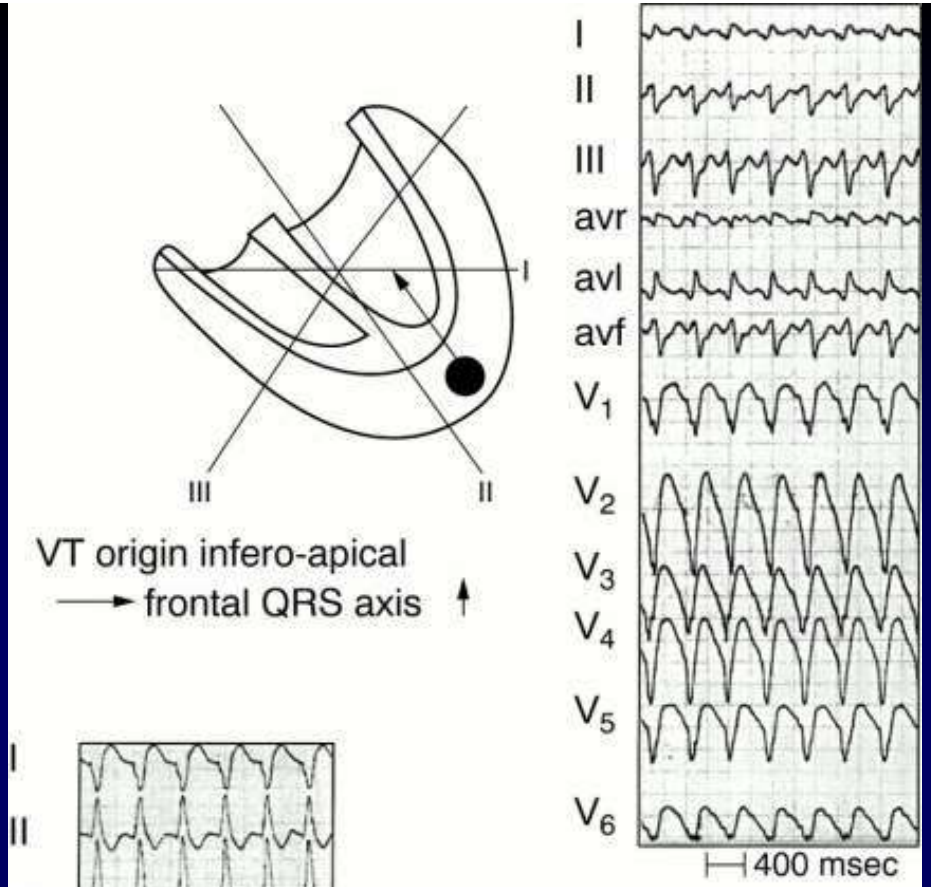
Kuchar DL et al. J Am Coll Cardiol 1989;13:893-900
de Chillou C et al. Arch Mal Coeur 2004;97(IV):13-24

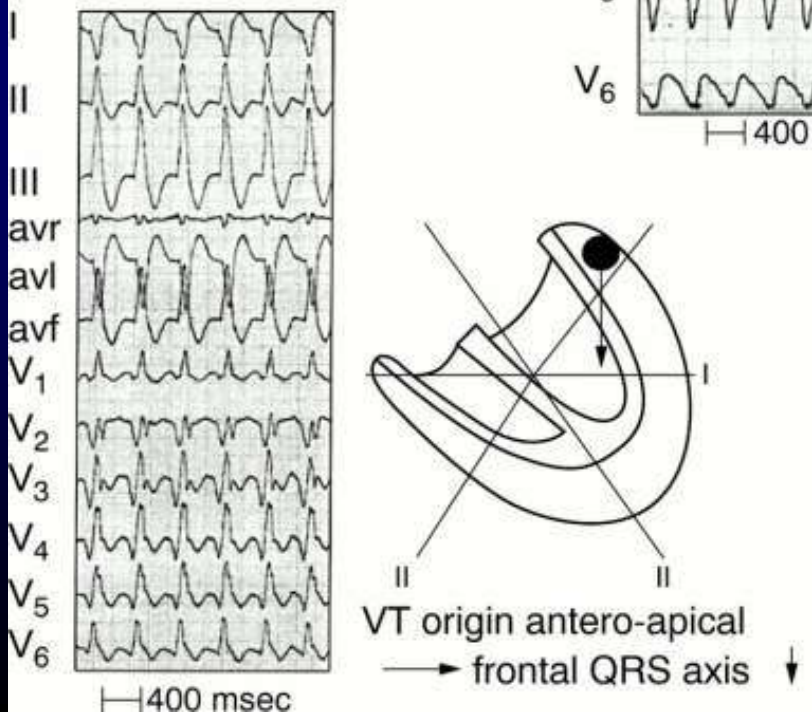
Vue en OAG

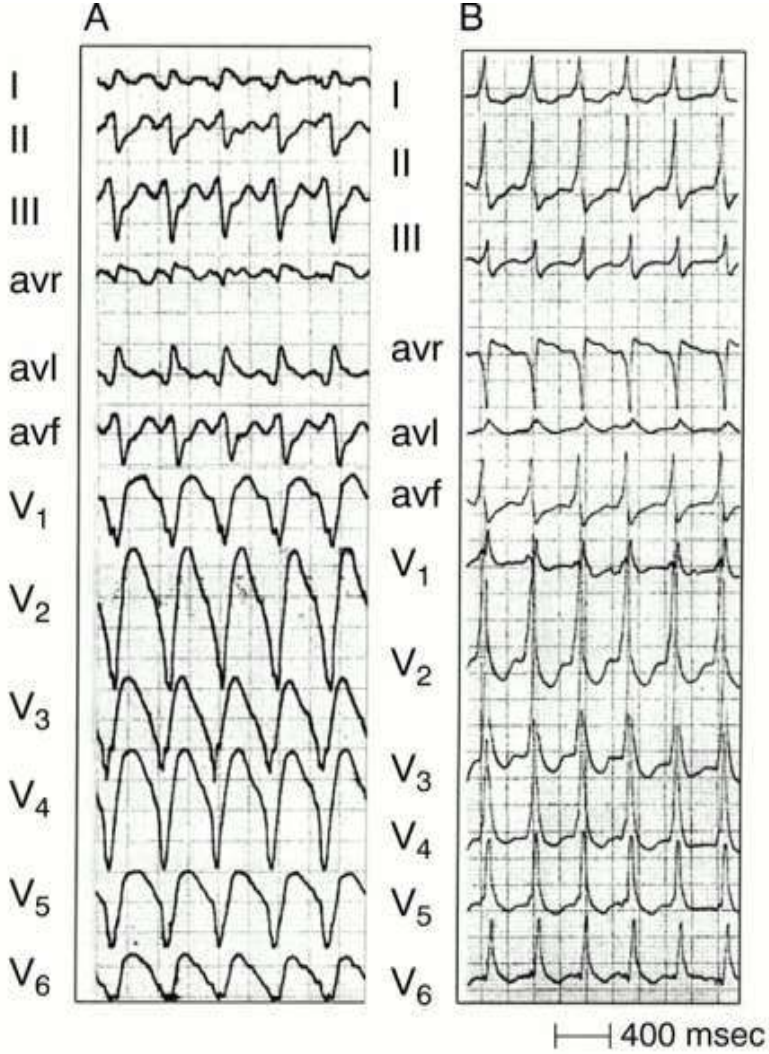


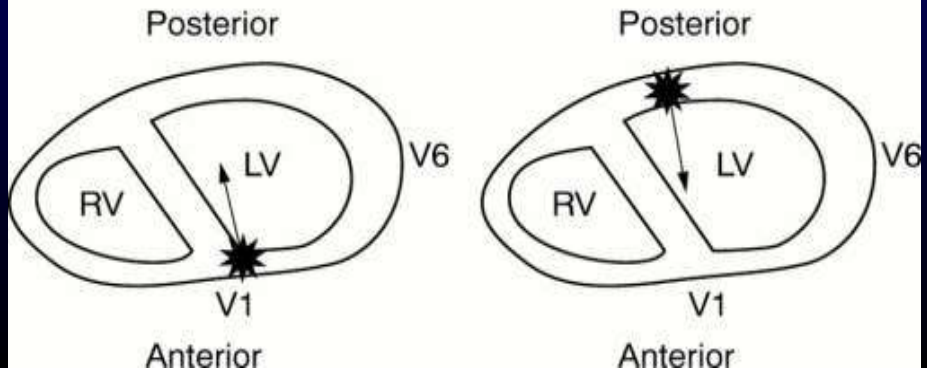
Coupe horizontale



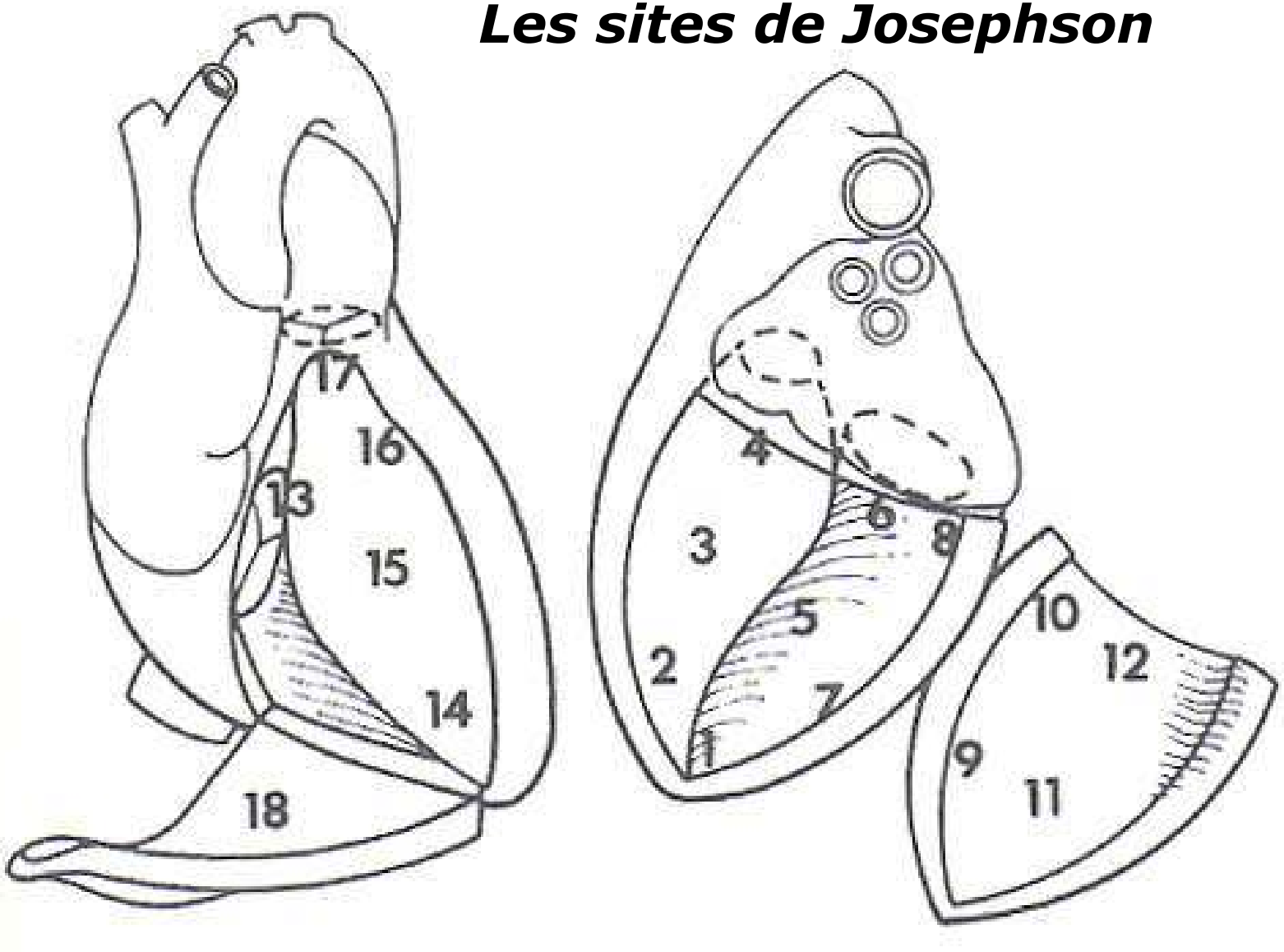

I
II
III
VT origin infero-apical
frontal QRS axis
I
II
III
avr
avl
avf
V1
V2
V3
V4
V5
V6
400 msec

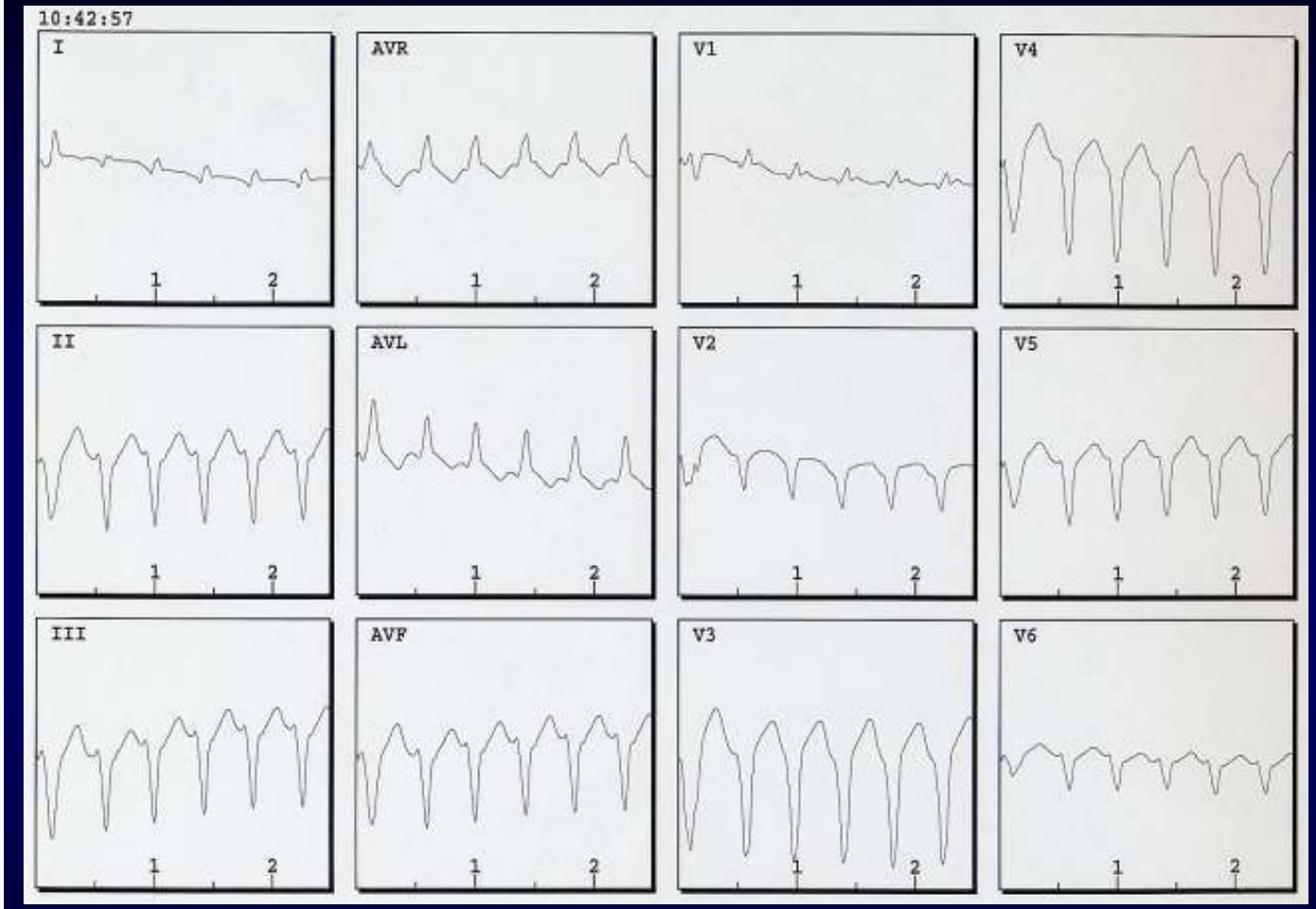

I
II
III
avr
avl
avf
V1
V2
V3
V4
V5
V6
400 msec
I
II
II
VT origin antero-apical
frontal QRS axis

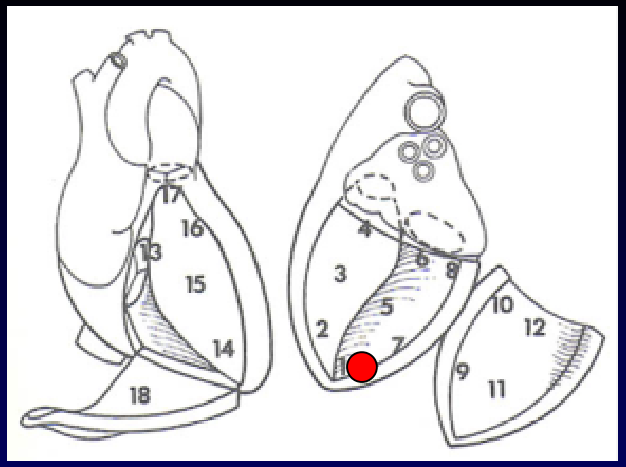

A
B
I
II
III
avr
avl
avf
V1
V2
V3
V4
V5
V6
400 msec

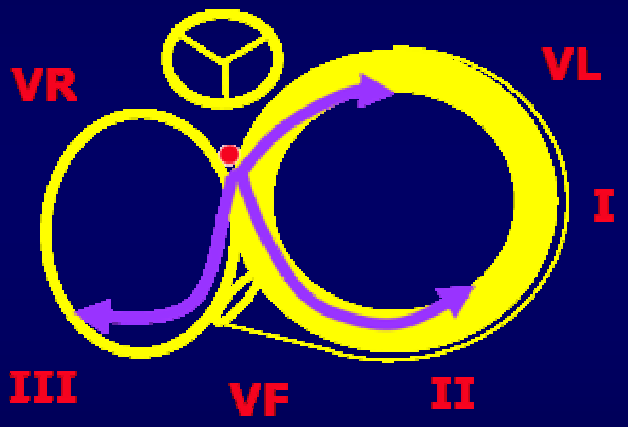

Posterior
RV
LV
V6
V1
Anterior
Posterior
RV
LV
V6
V1
Anterior

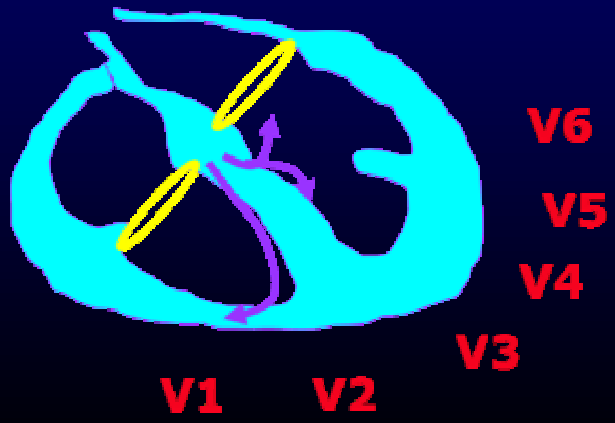
Les sites de Josephson



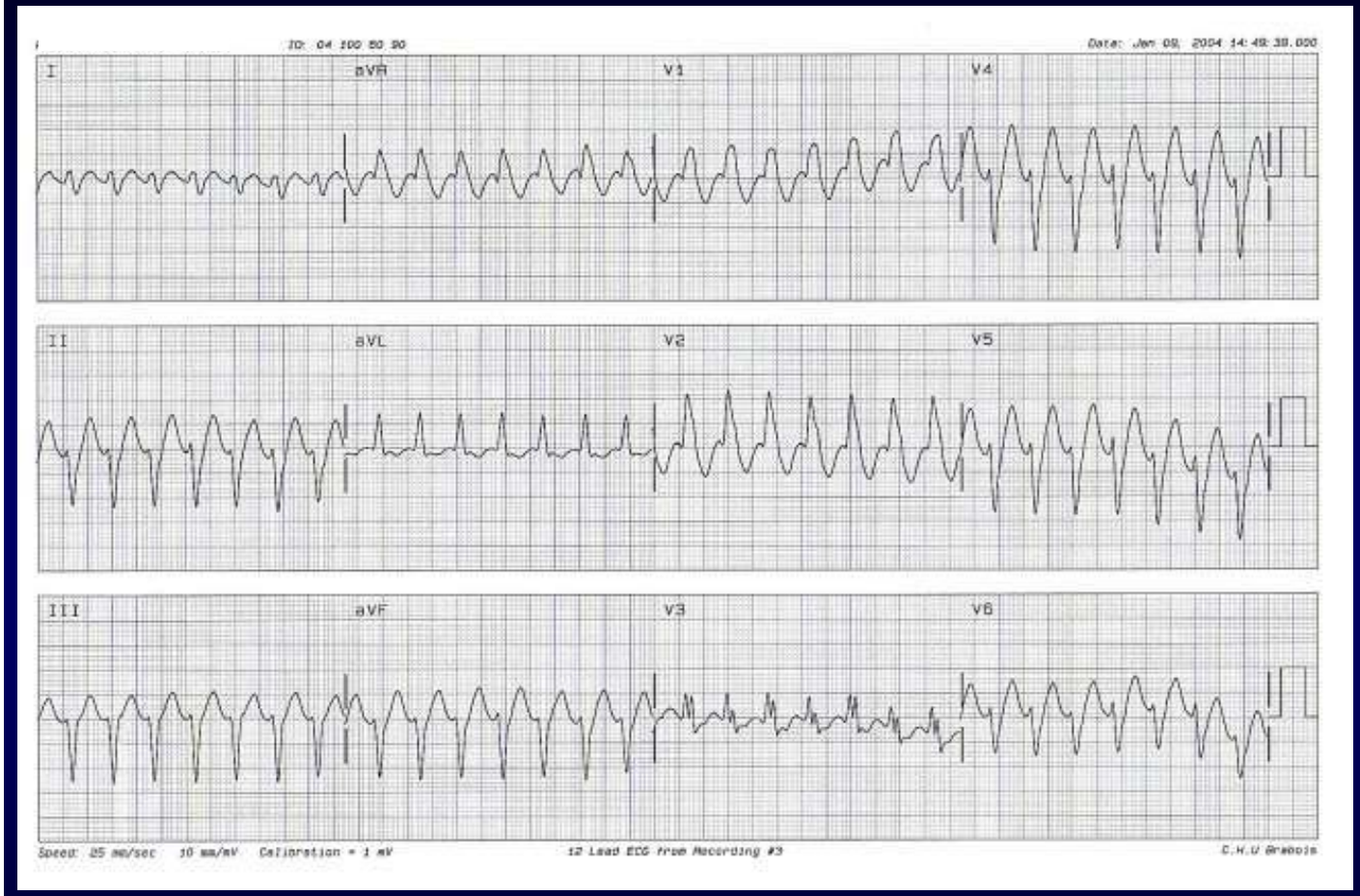


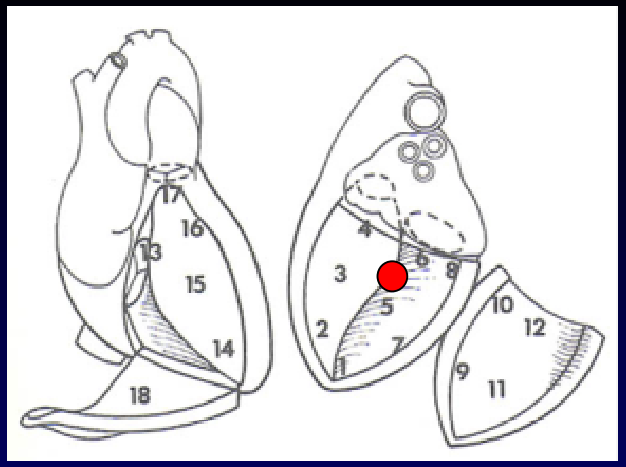


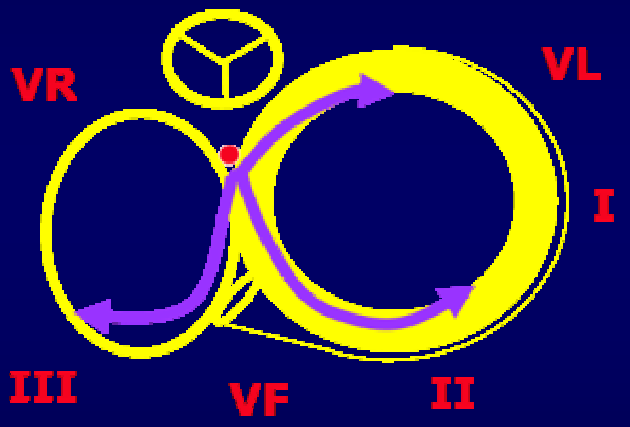


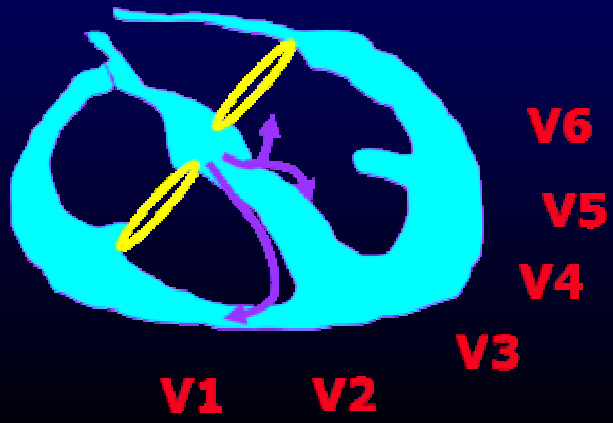


TV inféro-apicale du VG post IDM



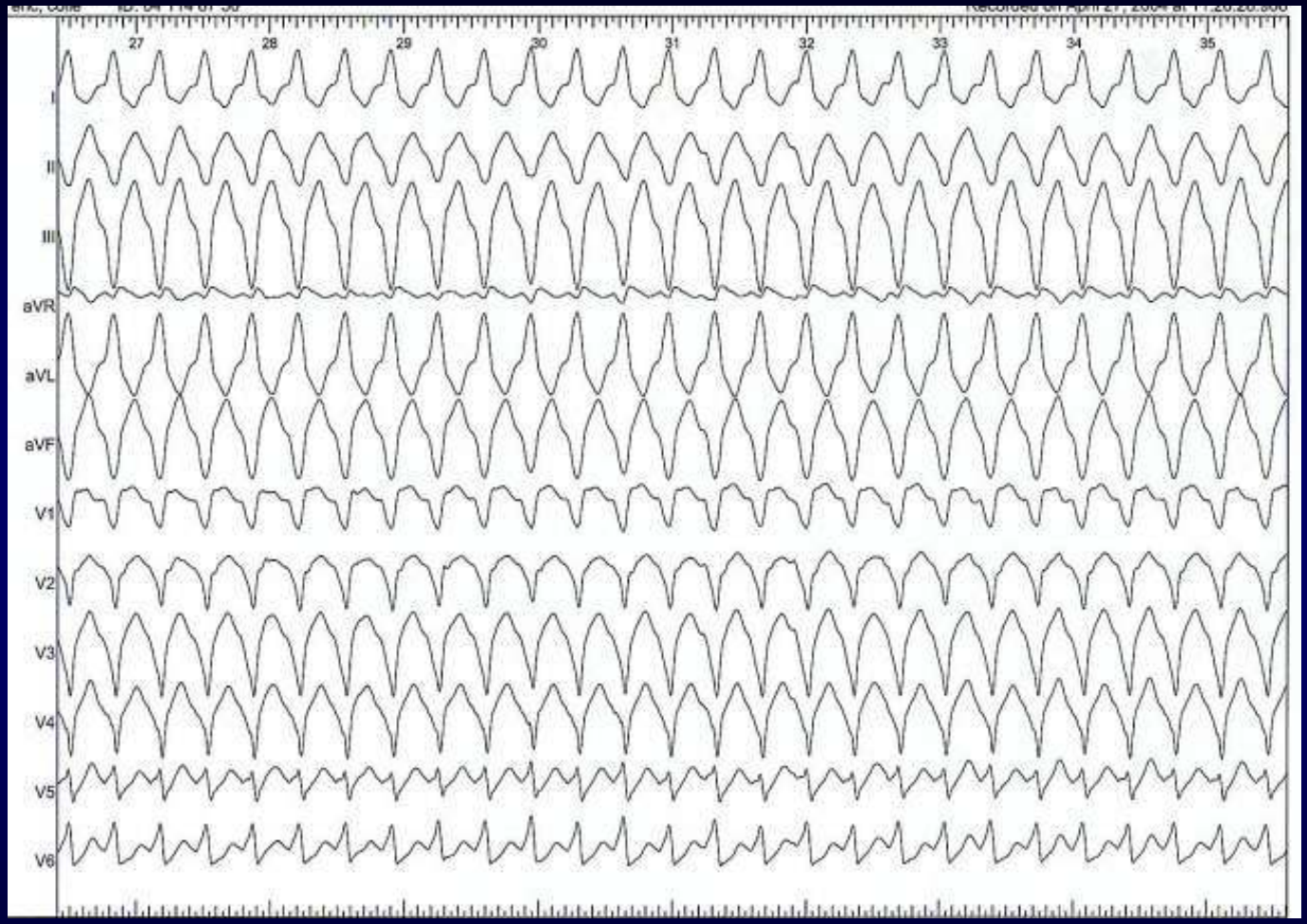


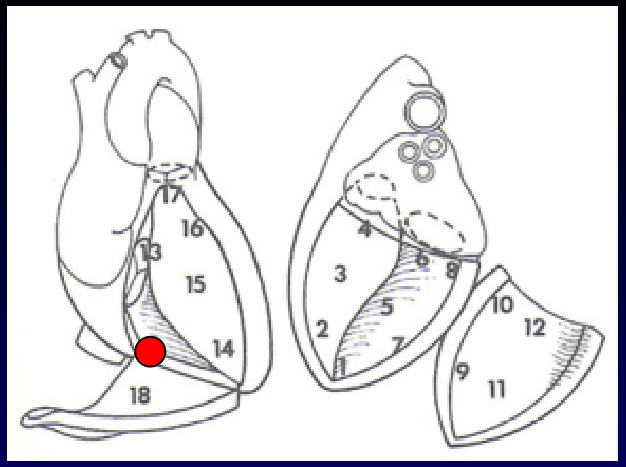


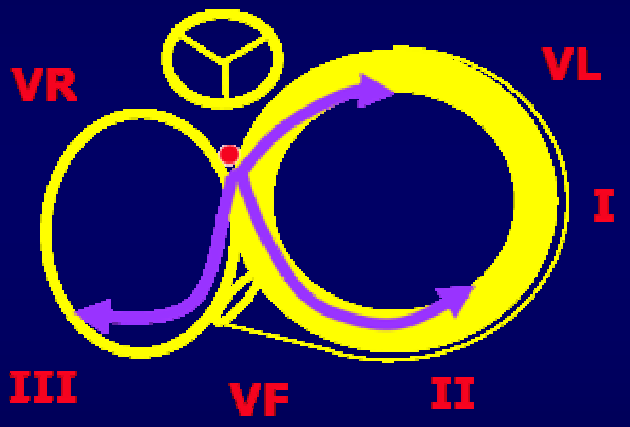


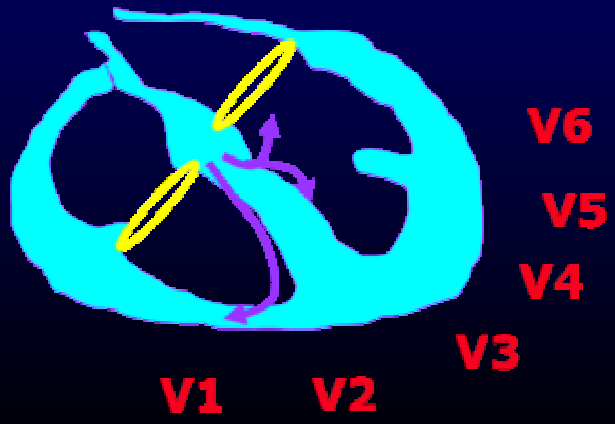
TV fasciculaire HBPG sur cœur sain

TV morphologie BBG axe gauche ➔ méfiance !

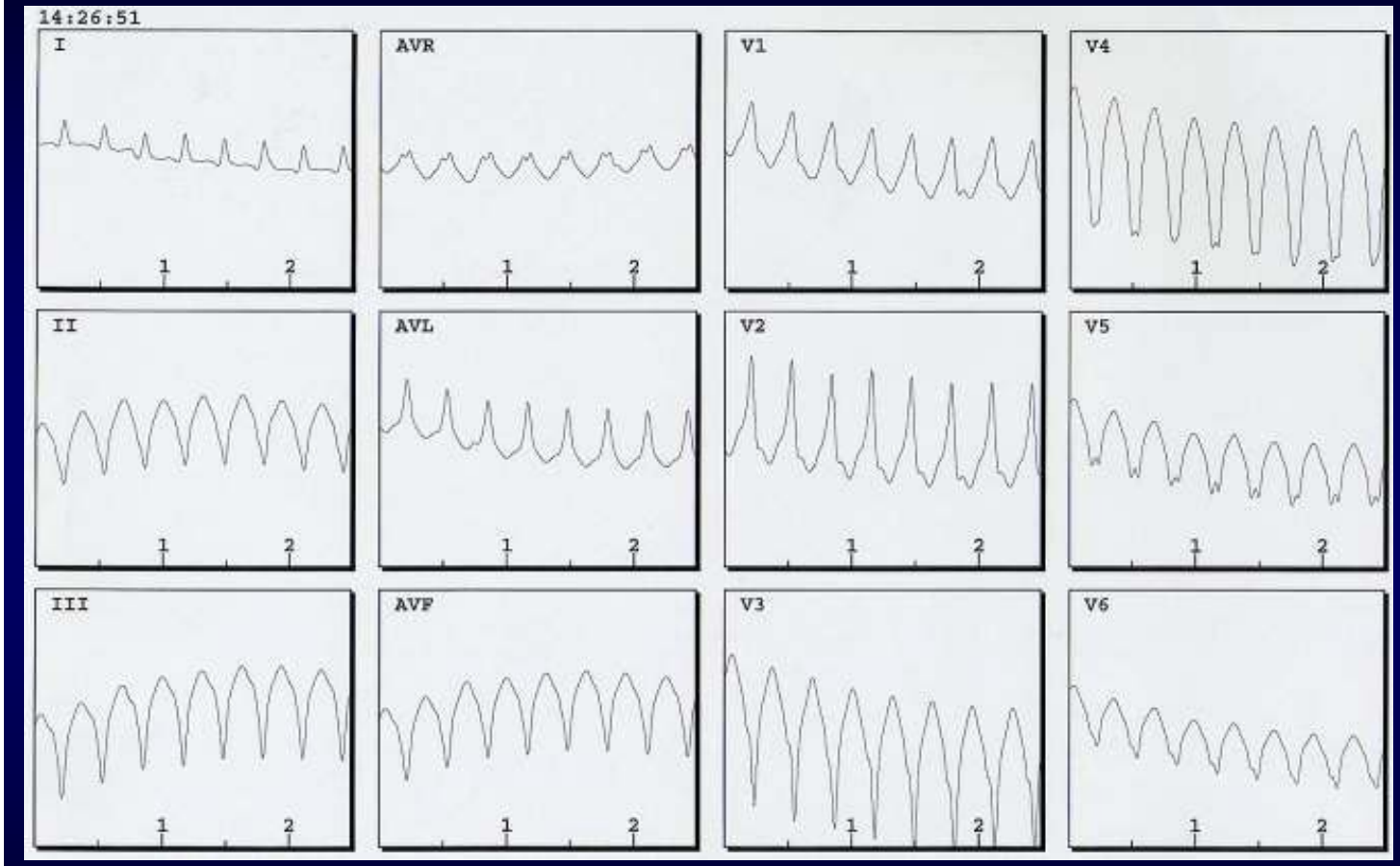


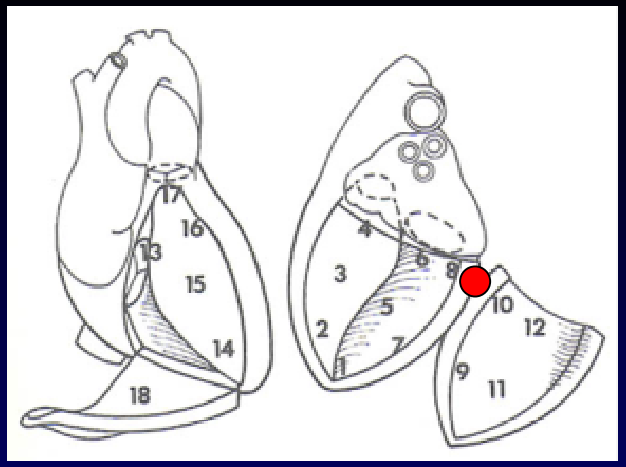


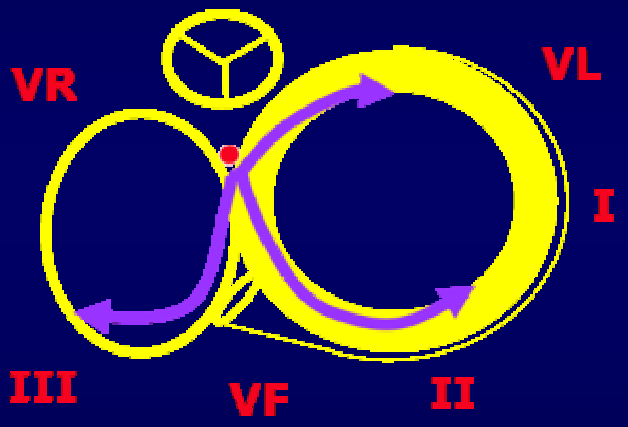


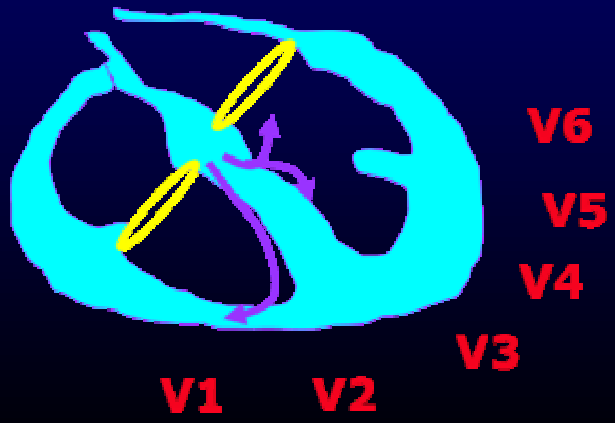


TV inféro-basale du VD sur DAVD









TV postéro-basale du VG post IDM

6

TECHNICAL ASPECTS OF VT ABLATION IN PATIENTS WITH A STRUCTURAL HEART DISEASE

Bundle Branch Reentrant Ventricular Tachycardia

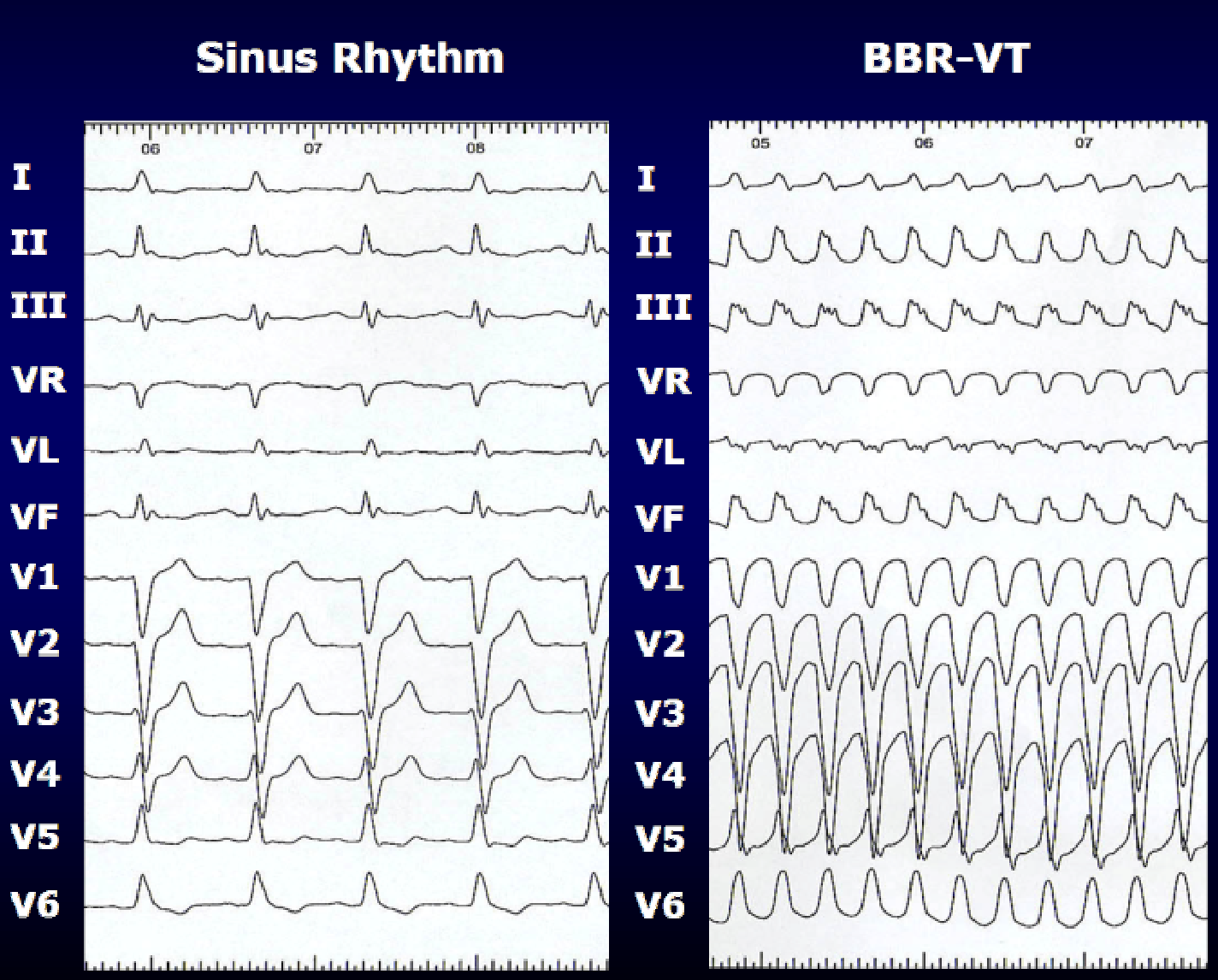
Bundle Branch Reentry (BBR) as the Mechanism of VT

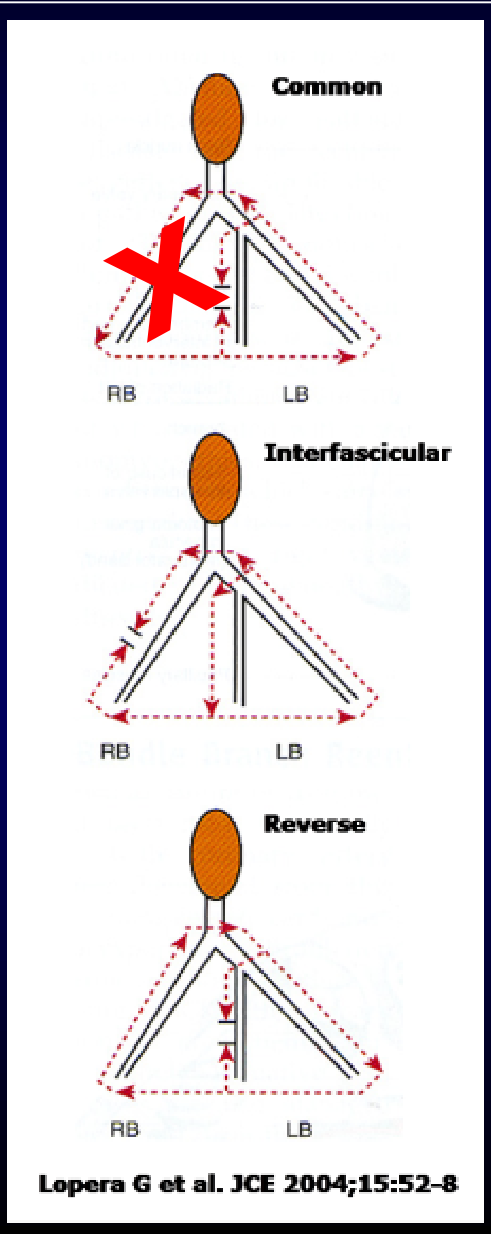
- Accounting for 5-6% of VT cases in CAD
- And up to 40% in non ischemic DCM*
- Diagnoses** associated with BBR-VT (CAD excluded)
  - Non ischemic DCM (50-70% of all BBR-VTs)
  - Valvular heart disease : aortic valve replacement, mitral valve disease
  - Muscular dystrophies, hypertrophic CM, Ebstein's anomaly
  - No structural heart disease but His-Purkinje conduction delays
- Heart is dilated in the vast majority of cases
- Enlarged QRS, prolonged H to V interval

* Blanck Z et al. In Zipes DP, Jalife J Eds. Cardiac Electrophysiology: From cell to bedside, 2nd ed. Philadelphia Saunders, 1995, pp 878-85

** Narasimhan C et al. Circulation 1997;96:4307-13
Merino JL et al. Circulation 1998;98:541-6
Blanck Z et al. J Cardiovasc Electrophysiol 1993;4:253-62
Blanck Z et al. J Am Coll Cardiol 1993;22:1718-22
Li YG et al. J Cardiovasc Electrophysiol 2002;13:1233-9

Ablation of Bundle Branch Reentrant VT

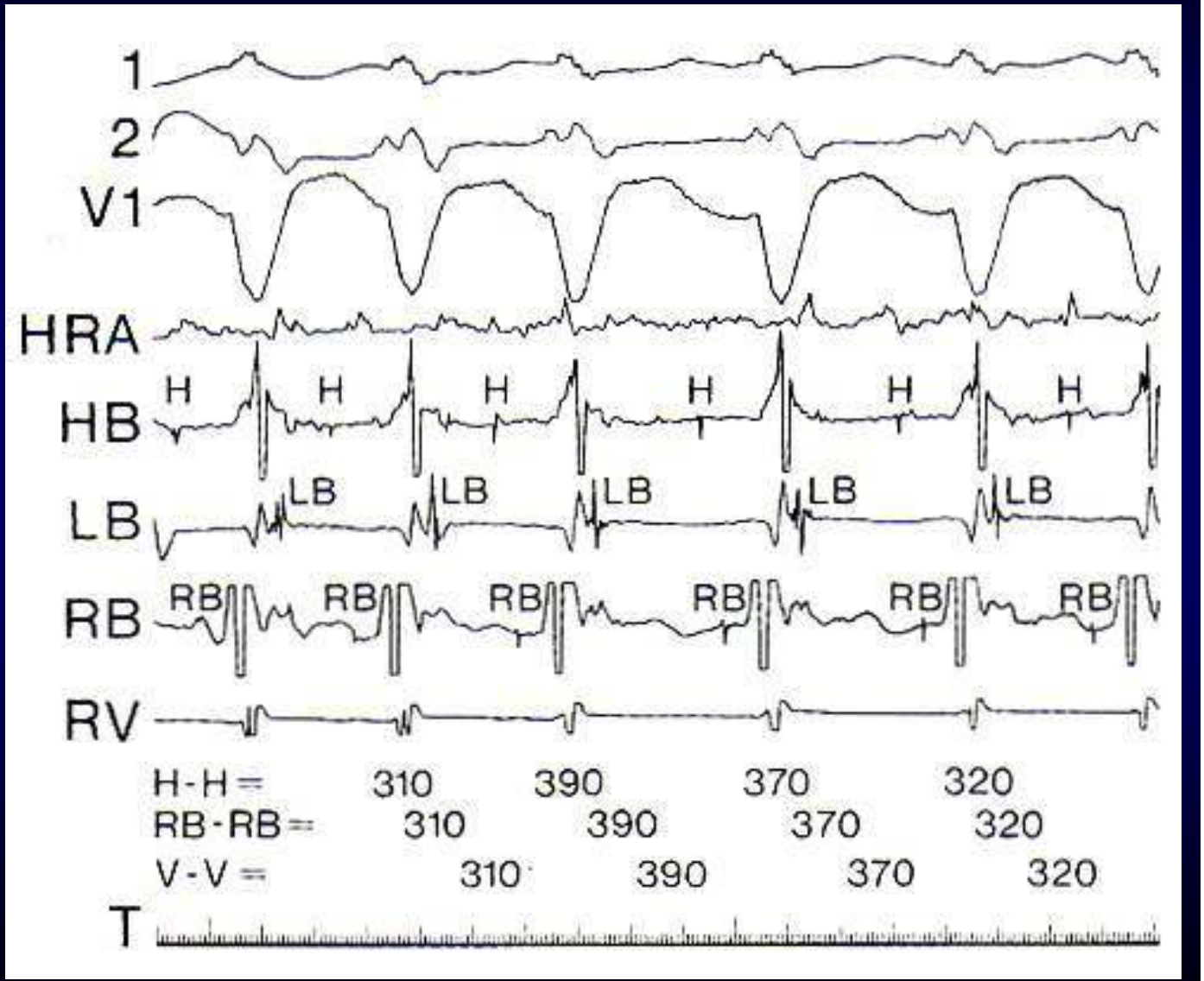




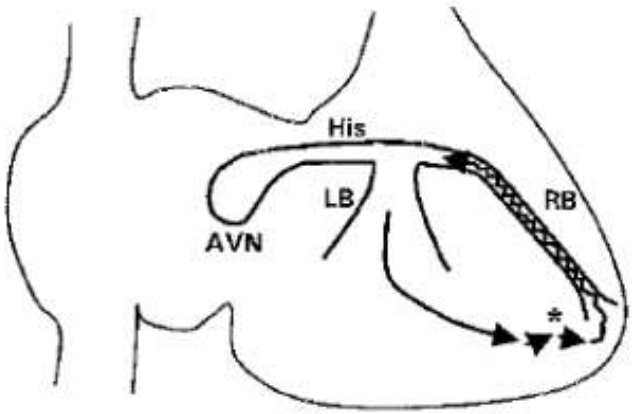
Lopera G et al. JCE 2004;15:52-8

EP characteristics of BBR-VT

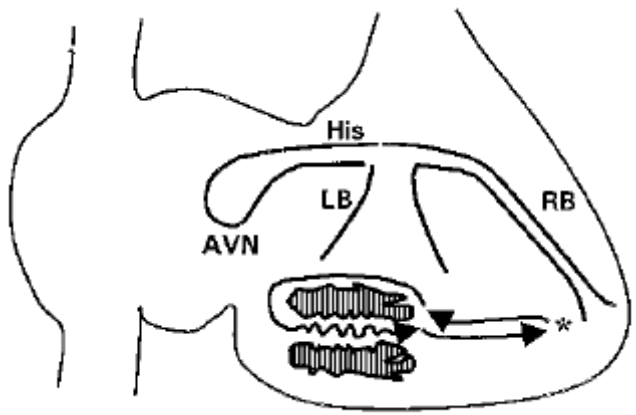
- H and RB potentials preceding V with appropriate sequence according BBR-VT type (A,B or C)
- HV interval identical or 10-30 ms longer than during sinus rhythm (type B excepted)
- H-H variations preceding V-V variations
- Induction depending upon a critical delay in the HPS
- Termination by block in the HPS
- Short-long-short sequences frequently required to induce
- Non inducibility after RBB ablation



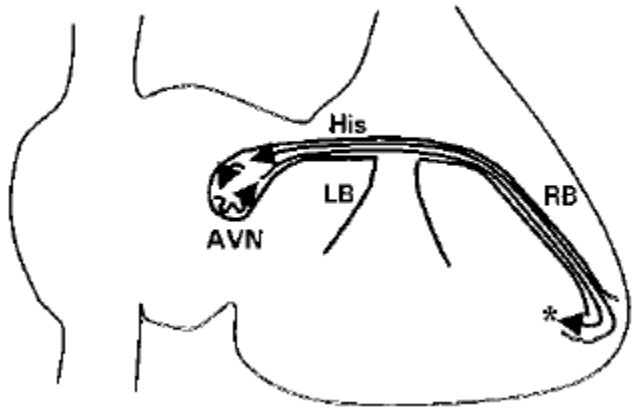
RV apical stimulation to measure PPI in suspected BBR-VT



BBR-VT
PPI ≈ TCL



MR-VT
PPI > TCL



AVNR-T
PPI > TCL

Merino JL et al. Circulation 2001;103:1102-8

Ventricular Tachycardia not Related to a Bundle Branch Reentry Mechanism

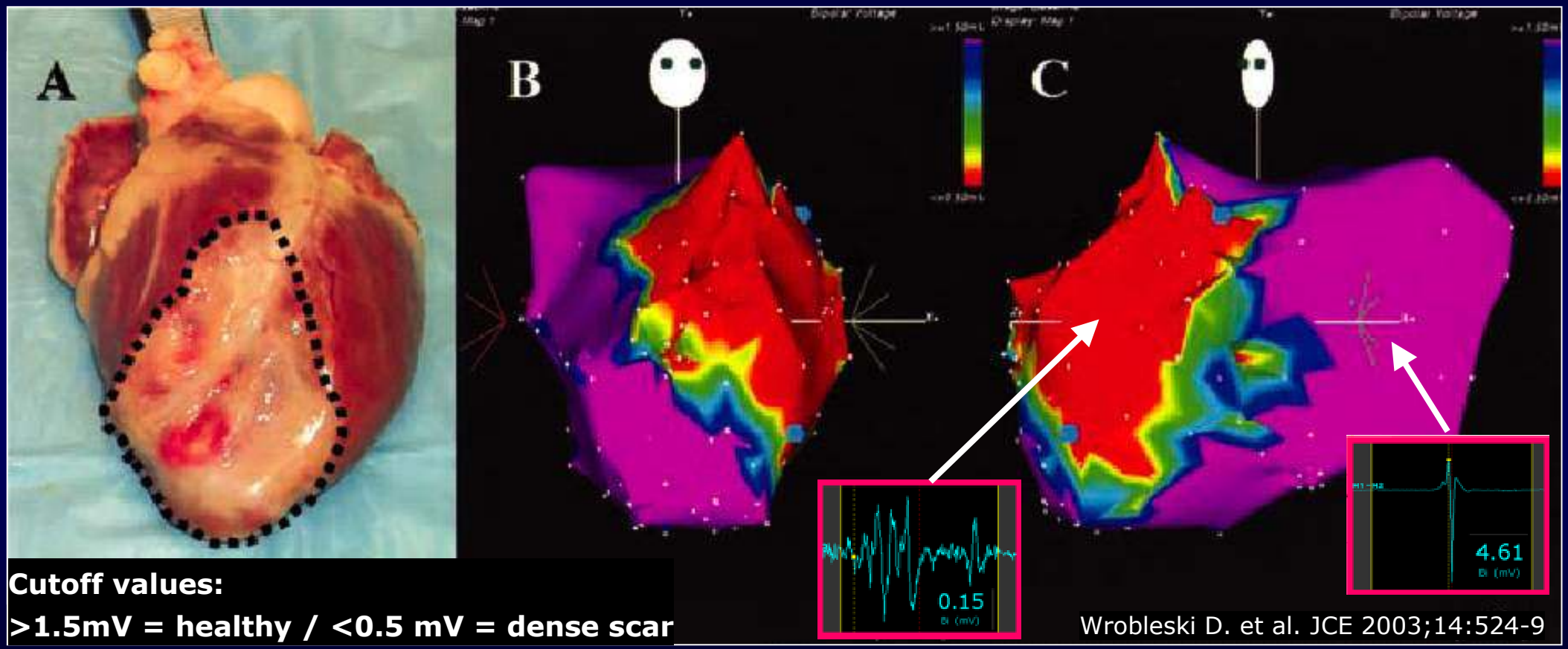
Substrate mapping

VT mapping

Ablation results

1

Correlation between voltage mapping and histology in myocardial infarction



Cutoff values:
>1.5mV = healthy / <0.5 mV = dense scar

Wrobleski D. et al. JCE 2003;14:524-9

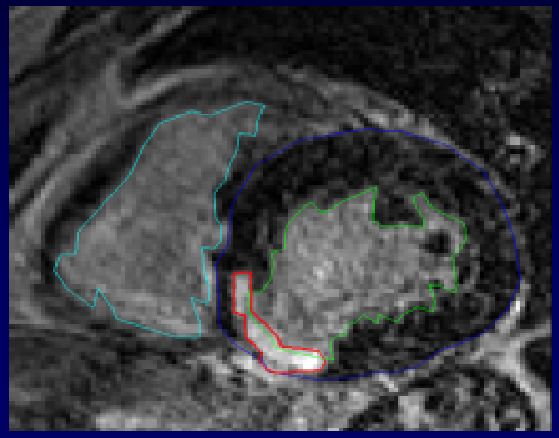
Animal studies – ***Callans DJ. et al. Circulation 1999;100:1744-50***

Human studies - ***Marchlinski FE. et al. Circulation 2000;101:1288-96***

1

University Hospital, Nancy

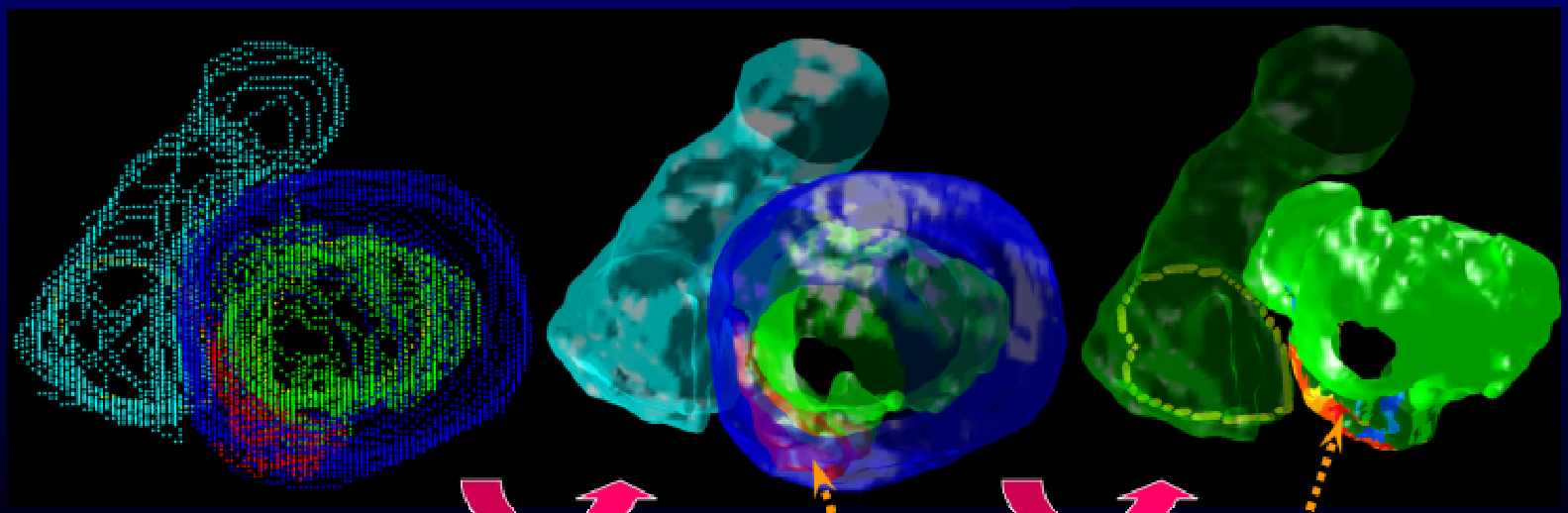
Departments of Cardiology & Radiology



2D manual definition of:
- endocardium
- epicardium
- scar contours

Topographic landmarks:
- RV and outflow tract
- mitral and tricuspid annulus

2D contours stack | 3D surface of the cavities | 3D LV endocardial surface

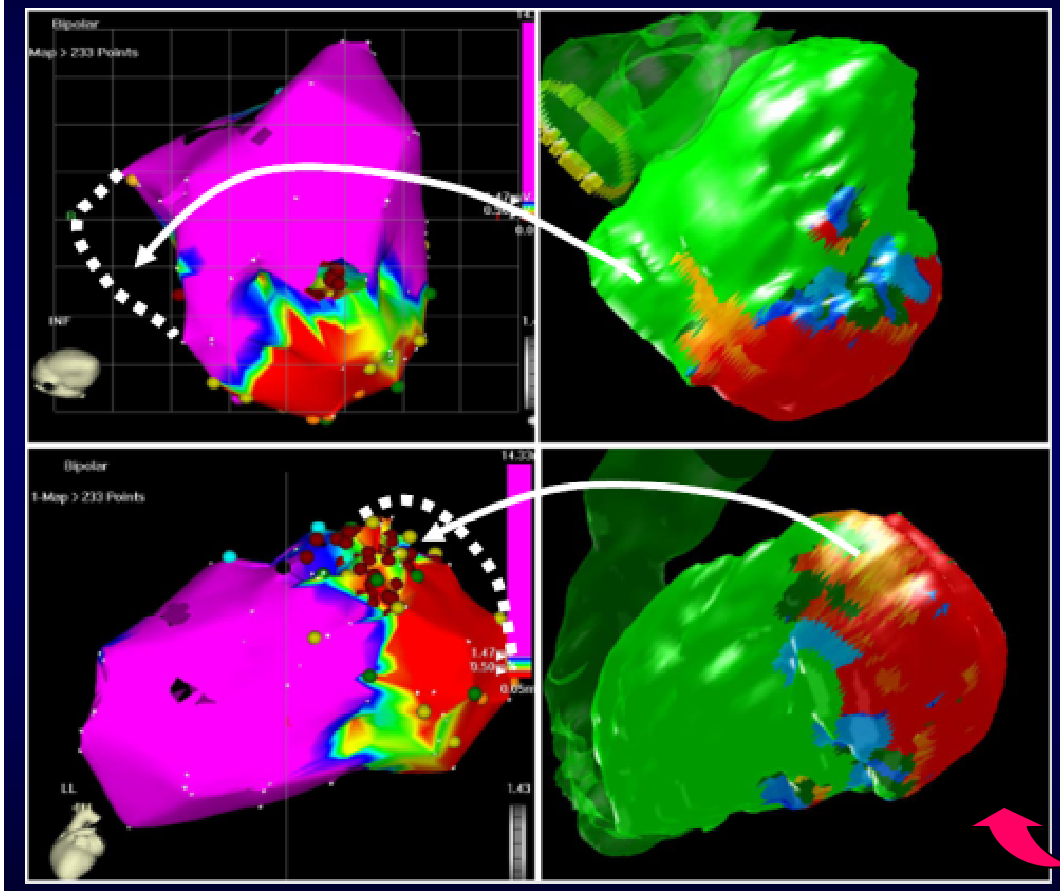


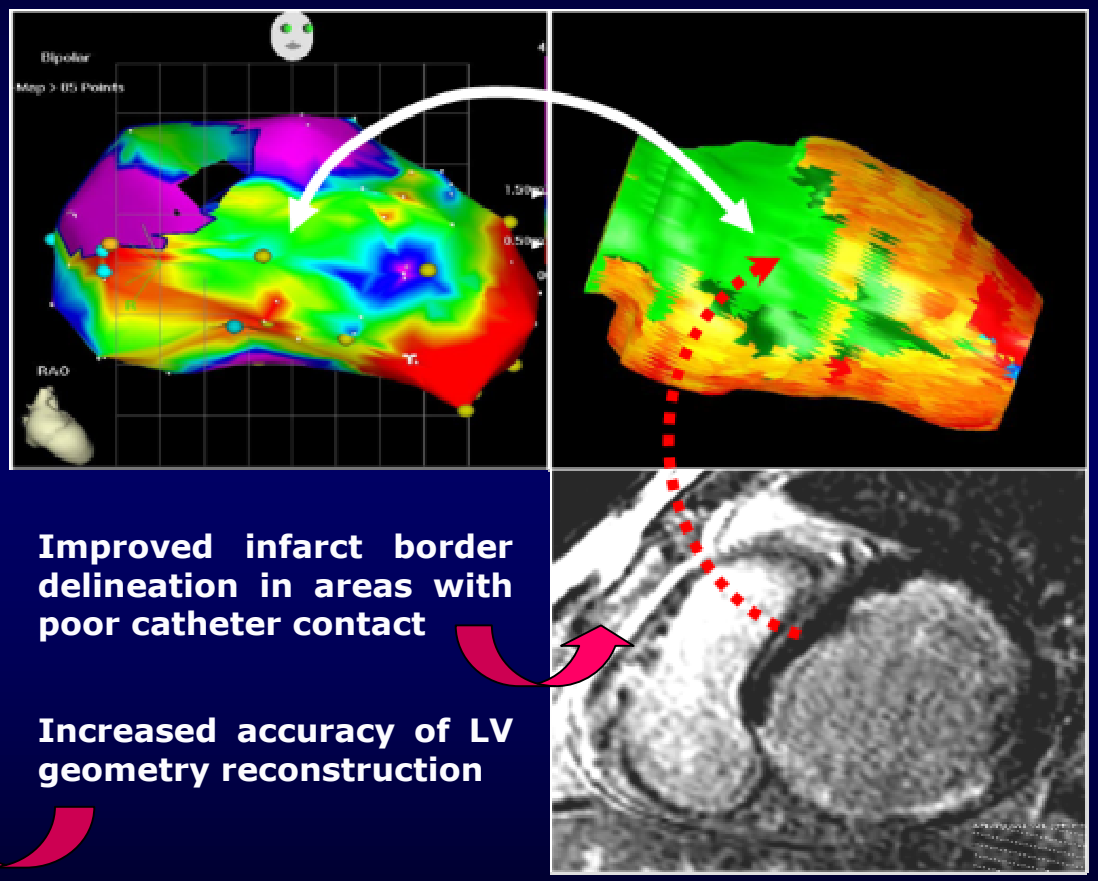
Smoothing of 2D contours + Isosurface extraction algorithm

Location and transmural extent of fibrous scars

1

Comparison between 3D MRI infarct reconstruction and 3D CARTO™ mapping



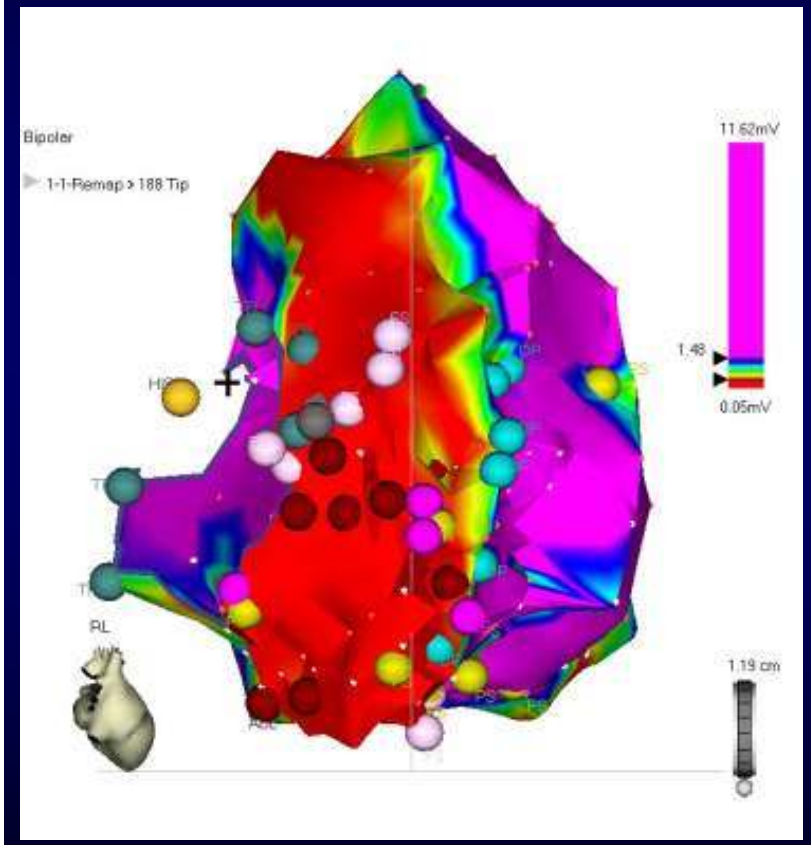


Improved infarct border delineation in areas with poor catheter contact

Increased accuracy of LV geometry reconstruction

Normal **Subendocardial to transmural** **Subepicardial** **Intramural**

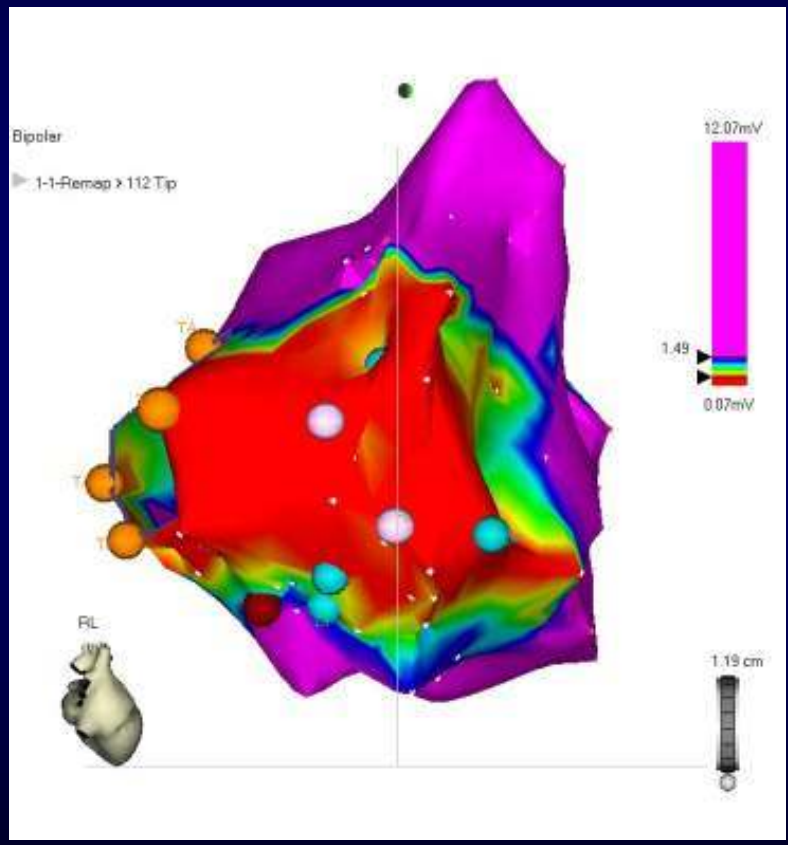
ARVD/C & Voltage mapping



11/11 patients (100%) with ARVD/C & VT showed RV areas with low bipolar voltage .

➔Latero-basal RV = 10/11 (±infero-basal)
➔ RVOT = 6/11
➔ RV apex = 0/11

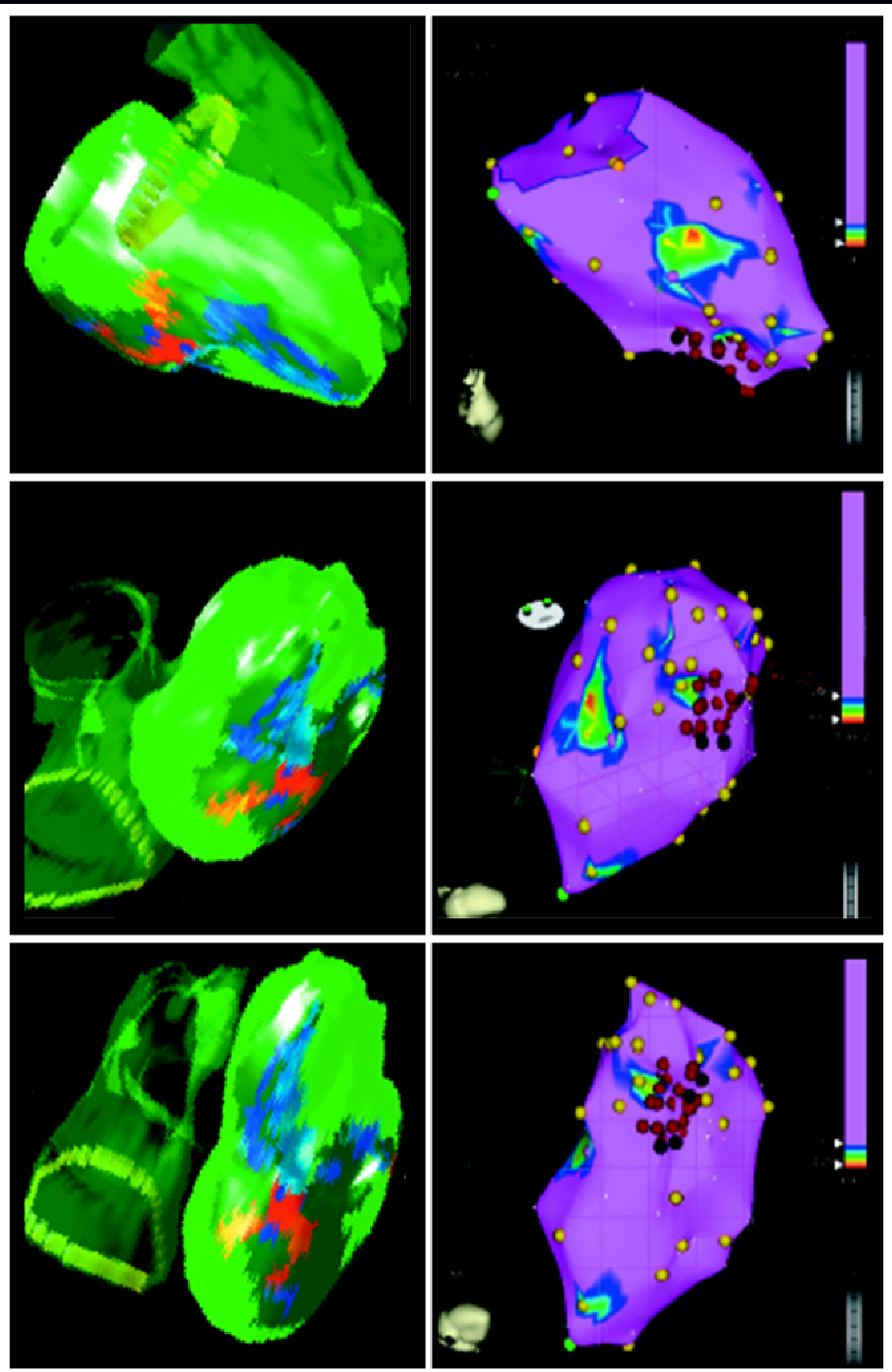
Miljoen H, de Chillou C et al. Europace 2005;7:516-24



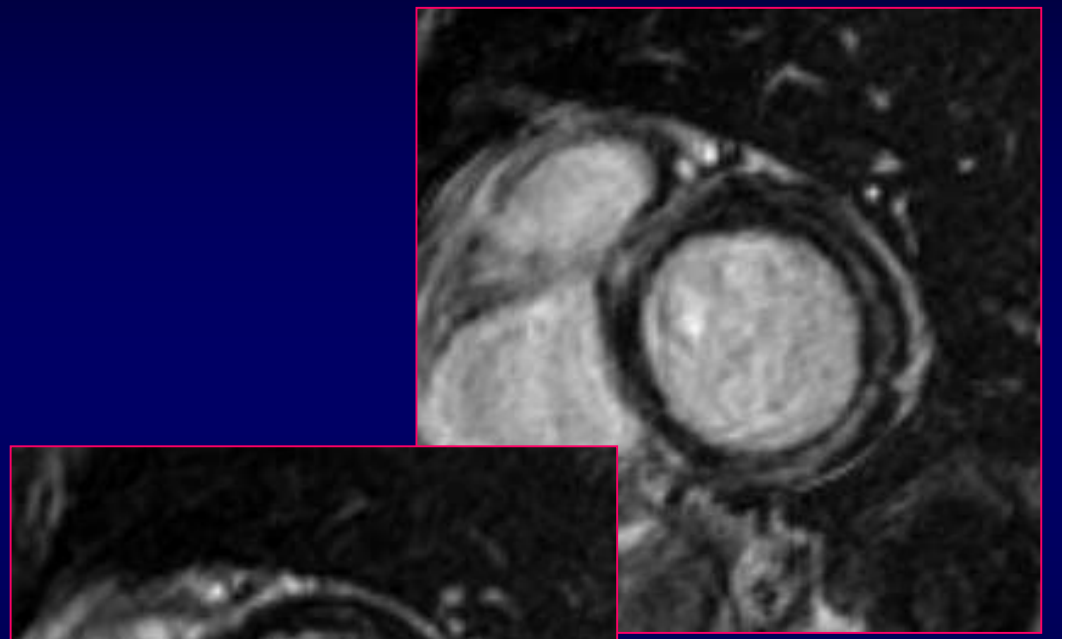
Similar results:

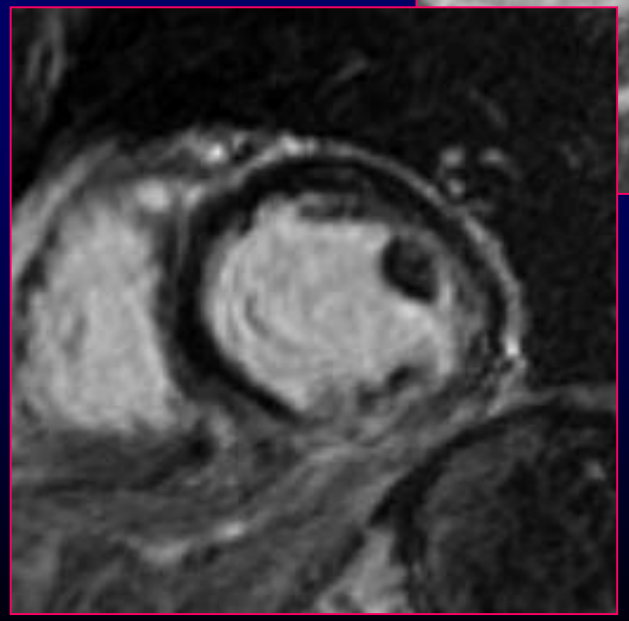
Boulos M et al. J Am Coll Cardiol 2001;38:2020-7 ➔ RV apex involved in 2/7 patients
Marchlinski FE et al. Circulation 2004;110:2293-8
Satomi K et al. J Cardiovasc Electrophysiol 2006;17:469-76 } Apex always spared

3

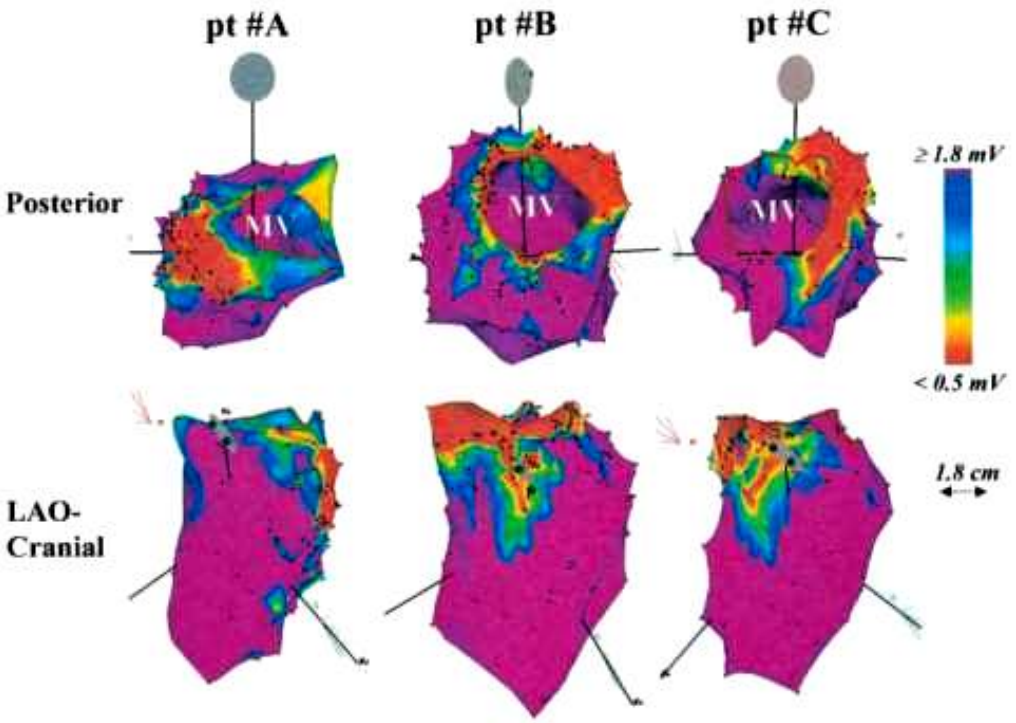


LV 3D map reconstruction with tissue characterization in idiopathic DCM





Idiopathic DCM & Voltage mapping

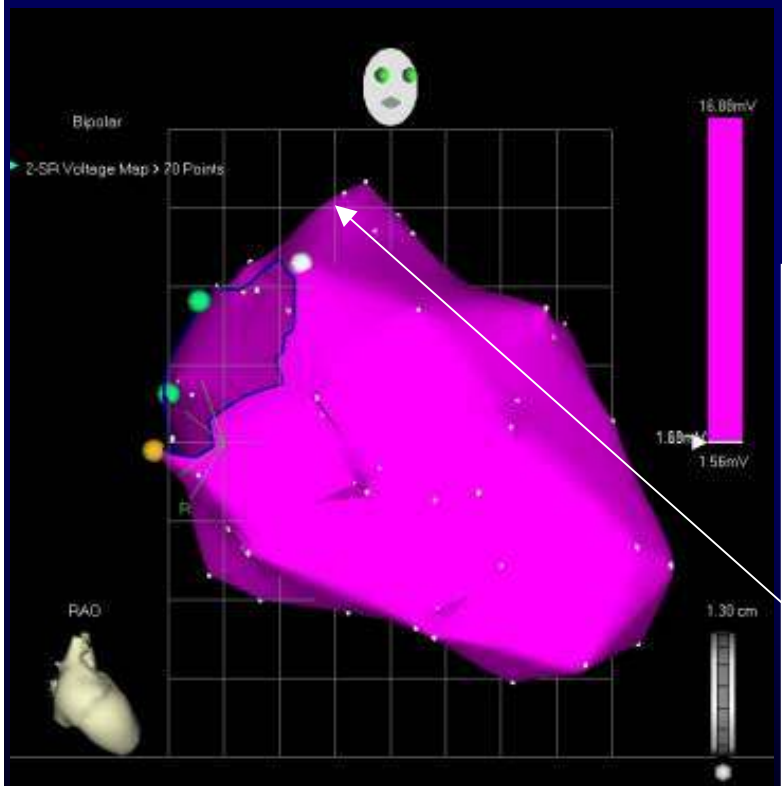


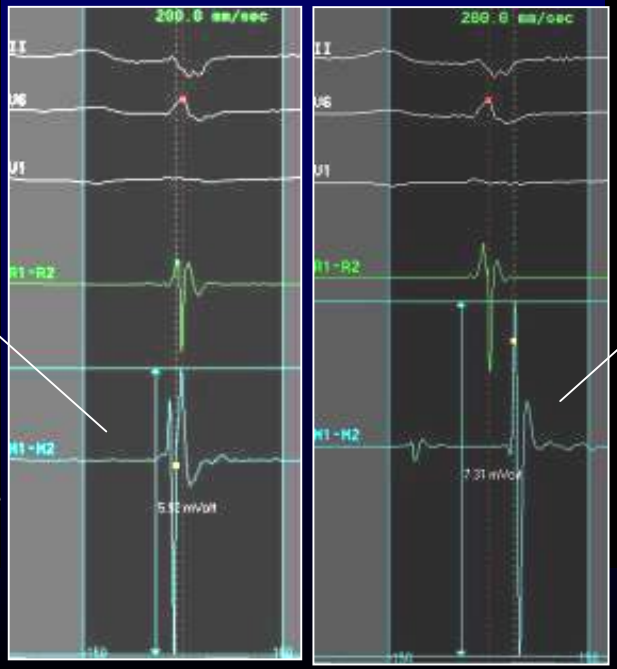
Region	Abnormal Endocardial Electrogram Location (19 patients)	VT Site-of-Origin (57 VTs)
Basal	19/19 (100%)	50/57 (88%)
Nonbasal	1/19 (5%)	7/57 (12%)

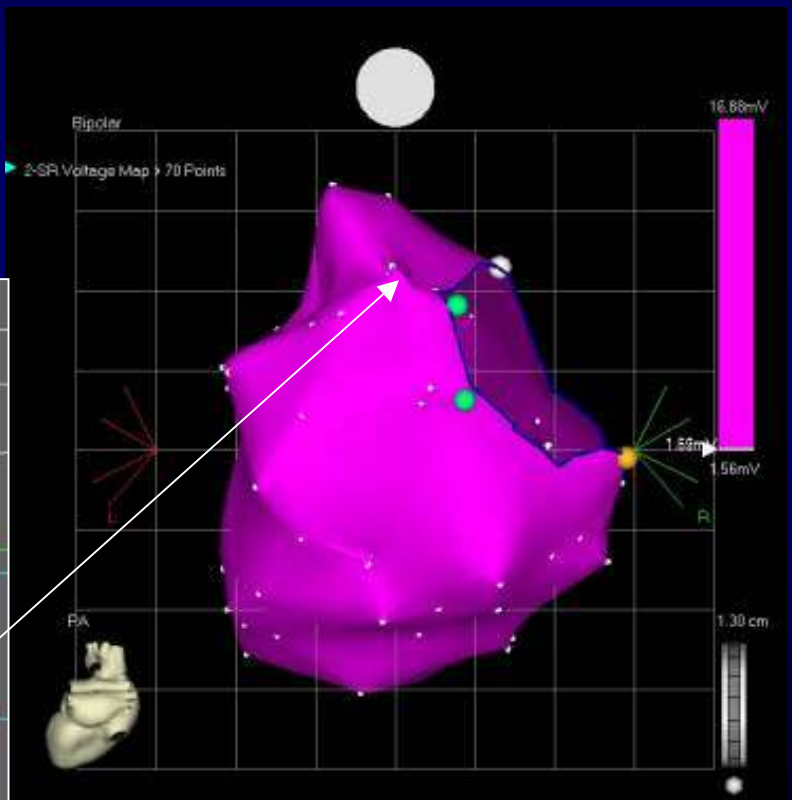
Hsia HH et al. Circulation 2003;108:704-10

Idiopathic DCM & Voltage mapping

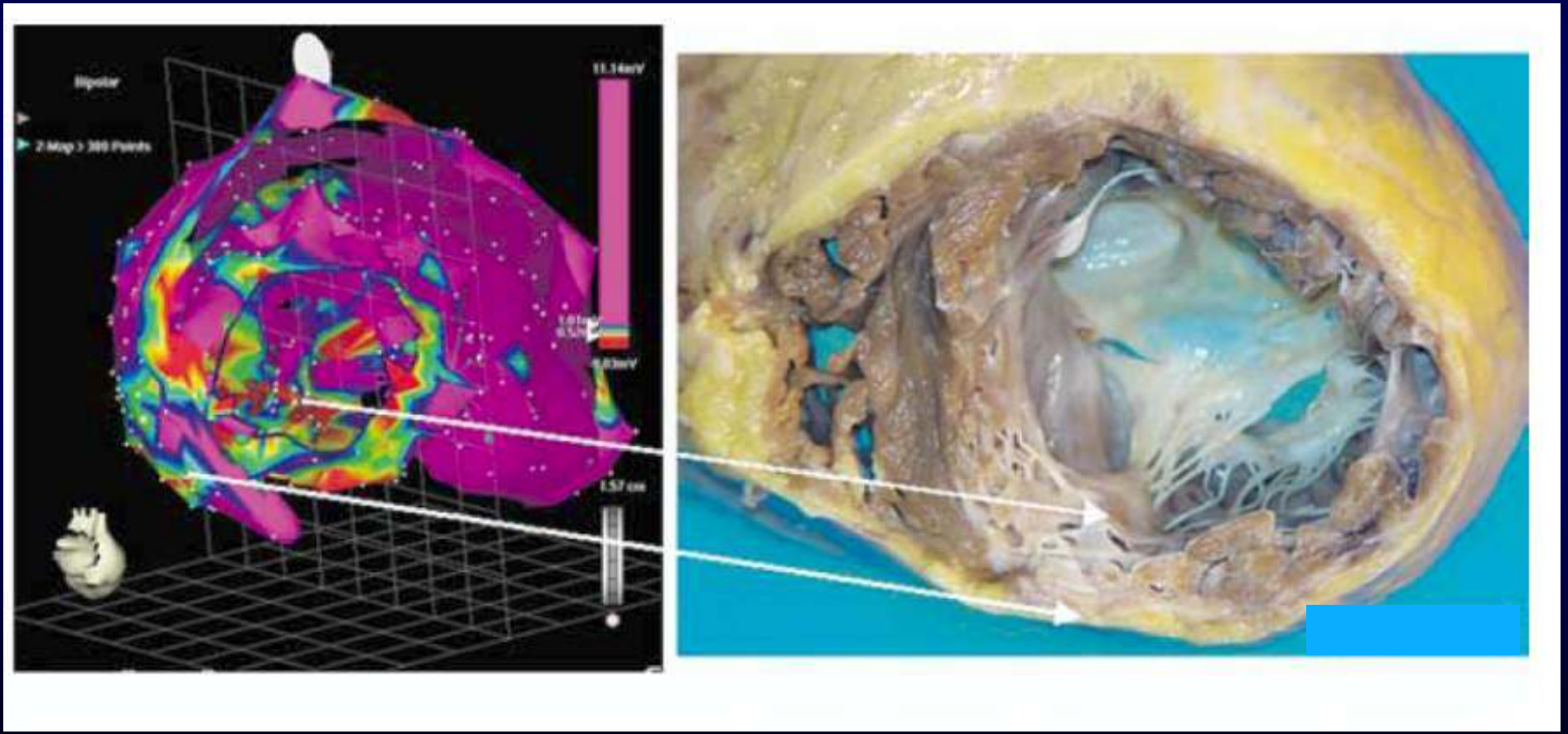
6/6 patients (100%) with idiopathic DCM showed no low bipolar voltage areas (personnal data)







Idiopathic DCM & Voltage mapping



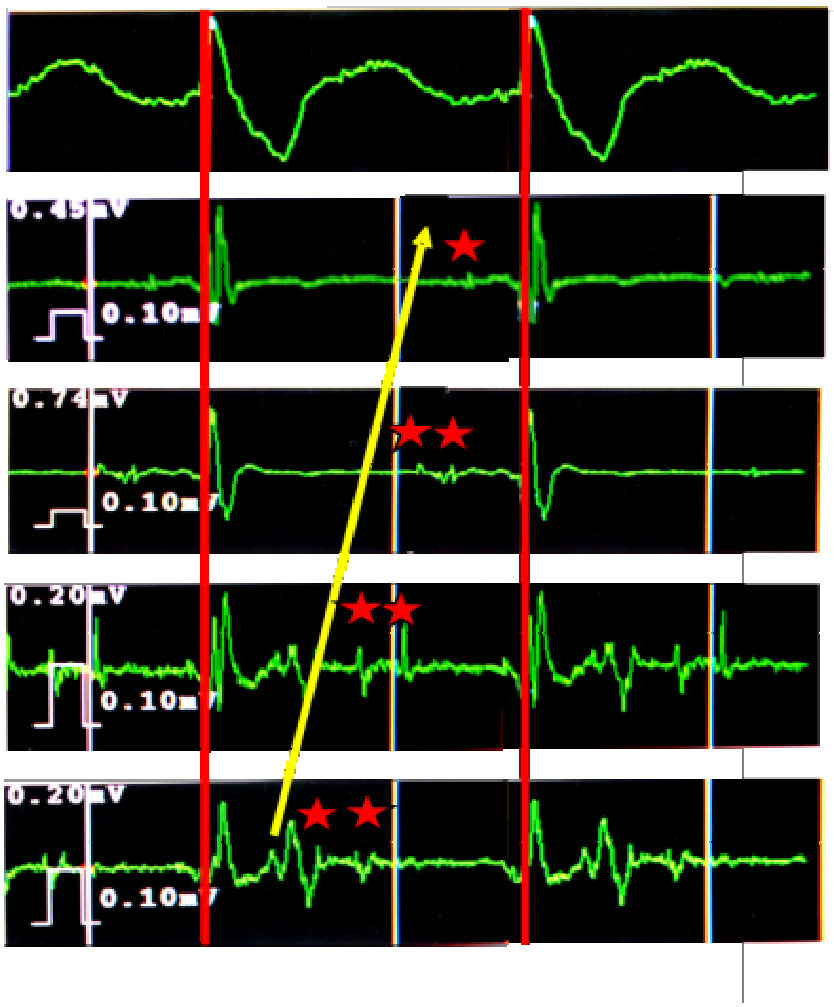
Cesario DA et al. Heart Rhythm 2006;3:1-10

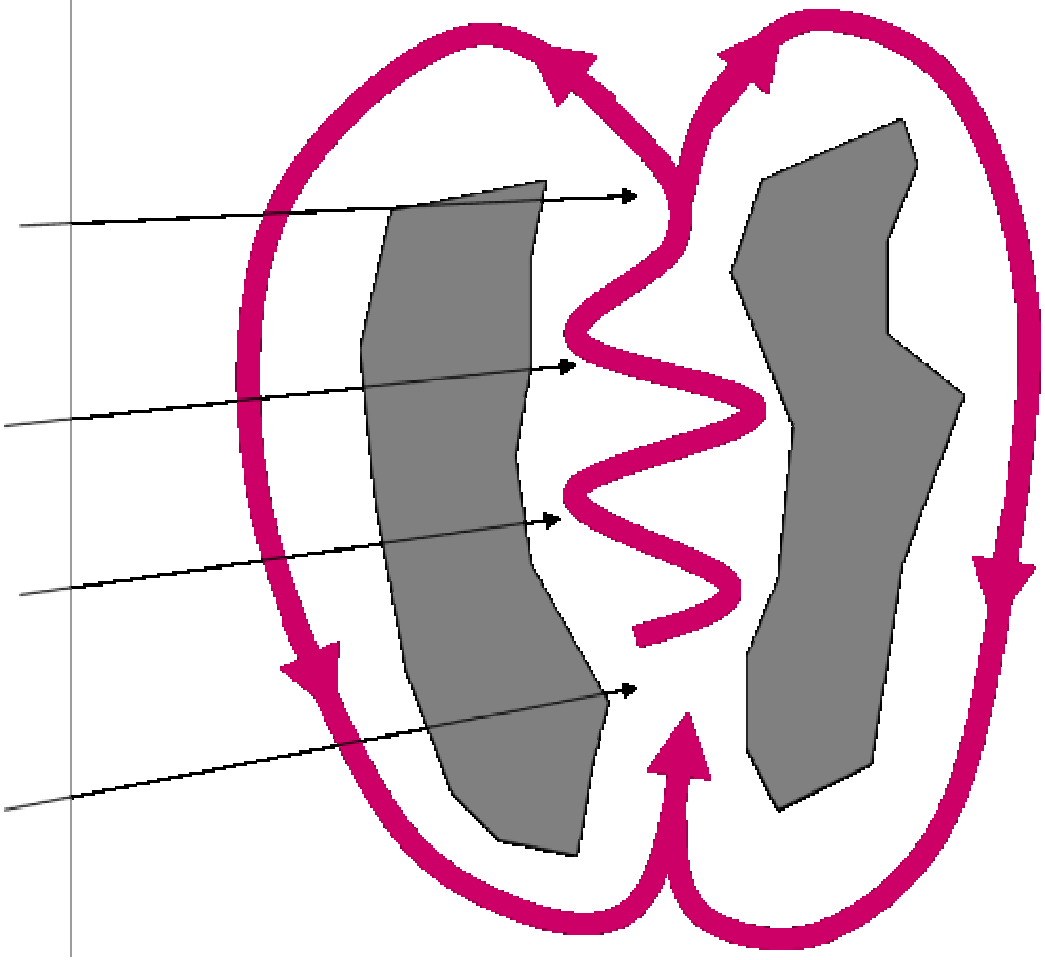
Substrate mapping

VT mapping

Ablation results

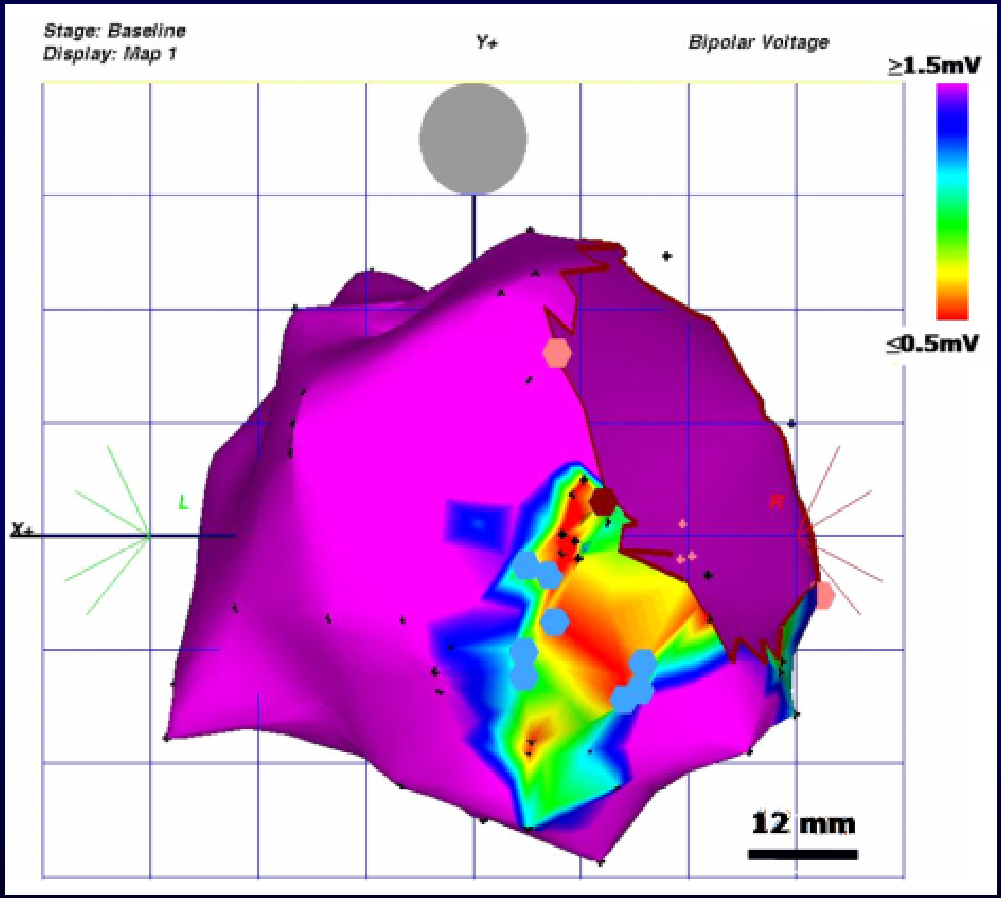
Endocardial reentrant circuit with a protected isthmus in >90% of post-MI related mappable VTs

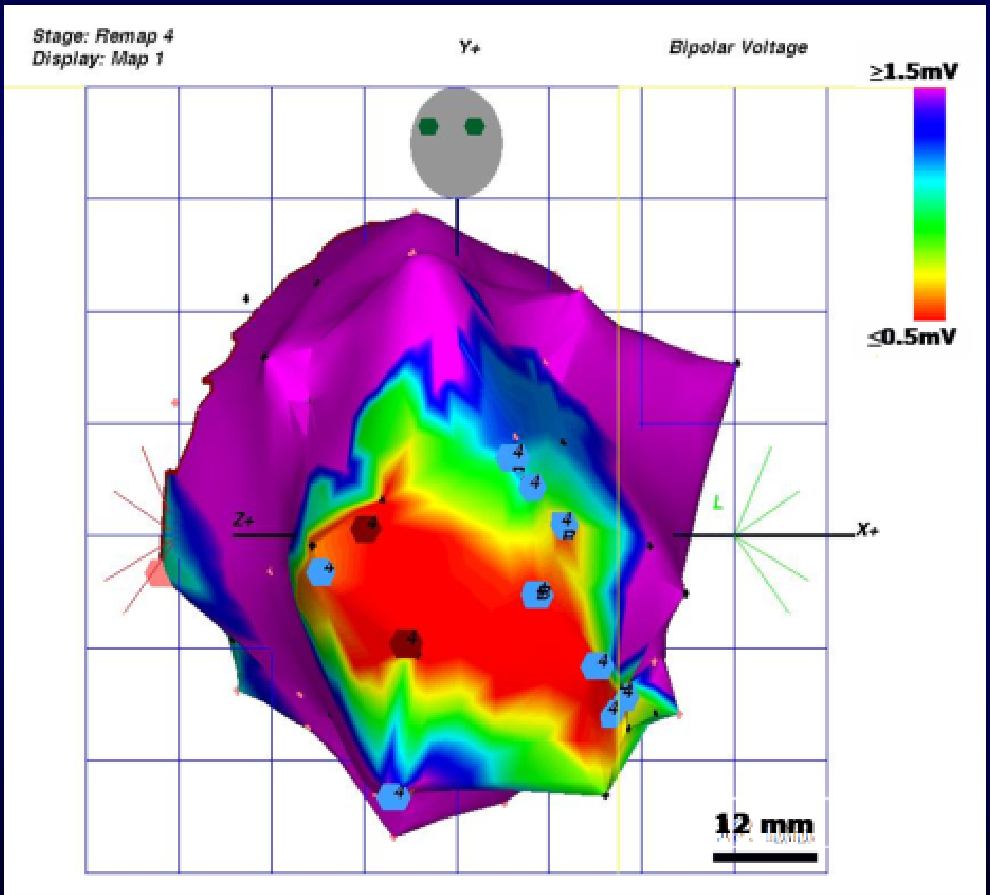




Post-infarct mappable VT ablation

Step # 1 = substrate mapping





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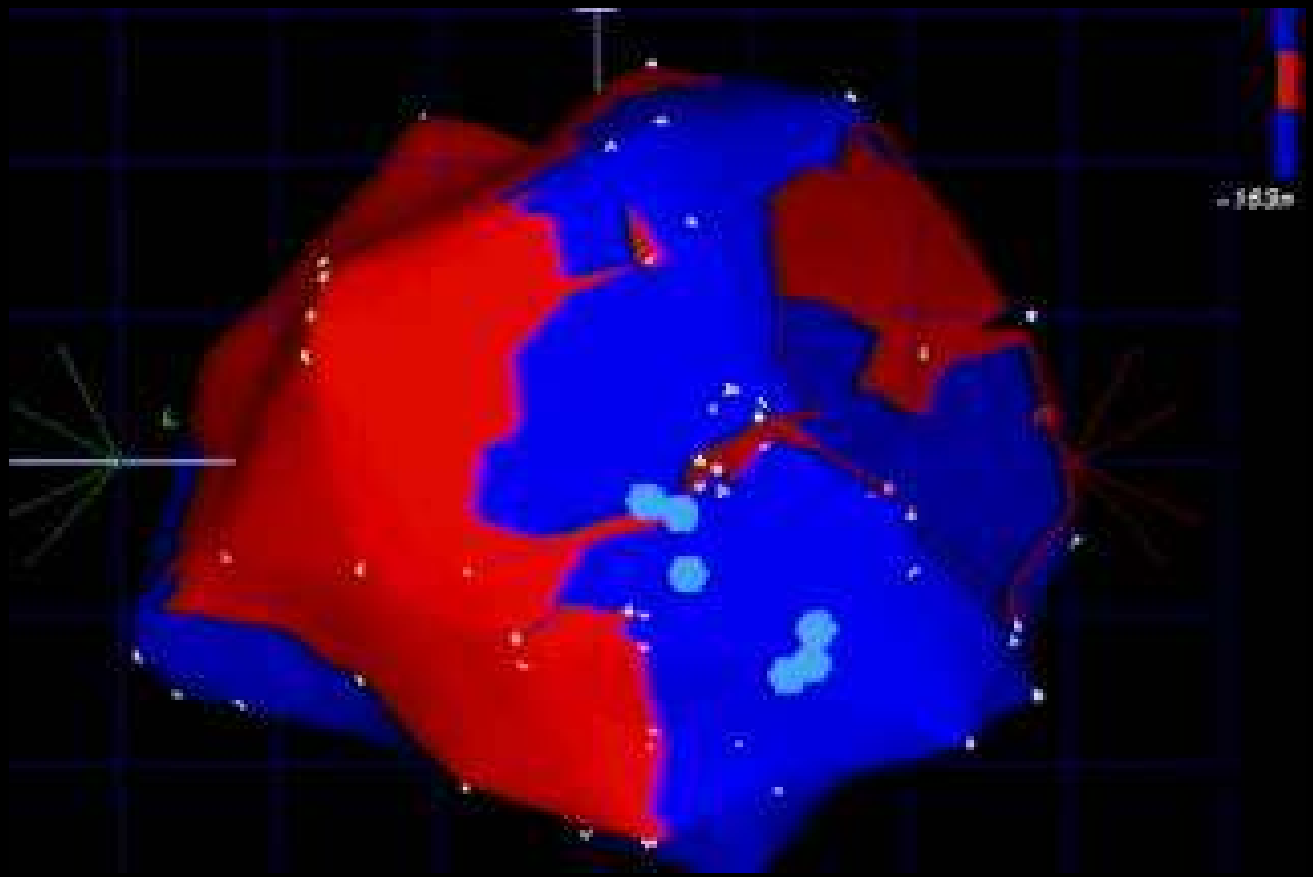
Post-infarct mappable VT ablation

Step # 2 = VT induction & VT mapping



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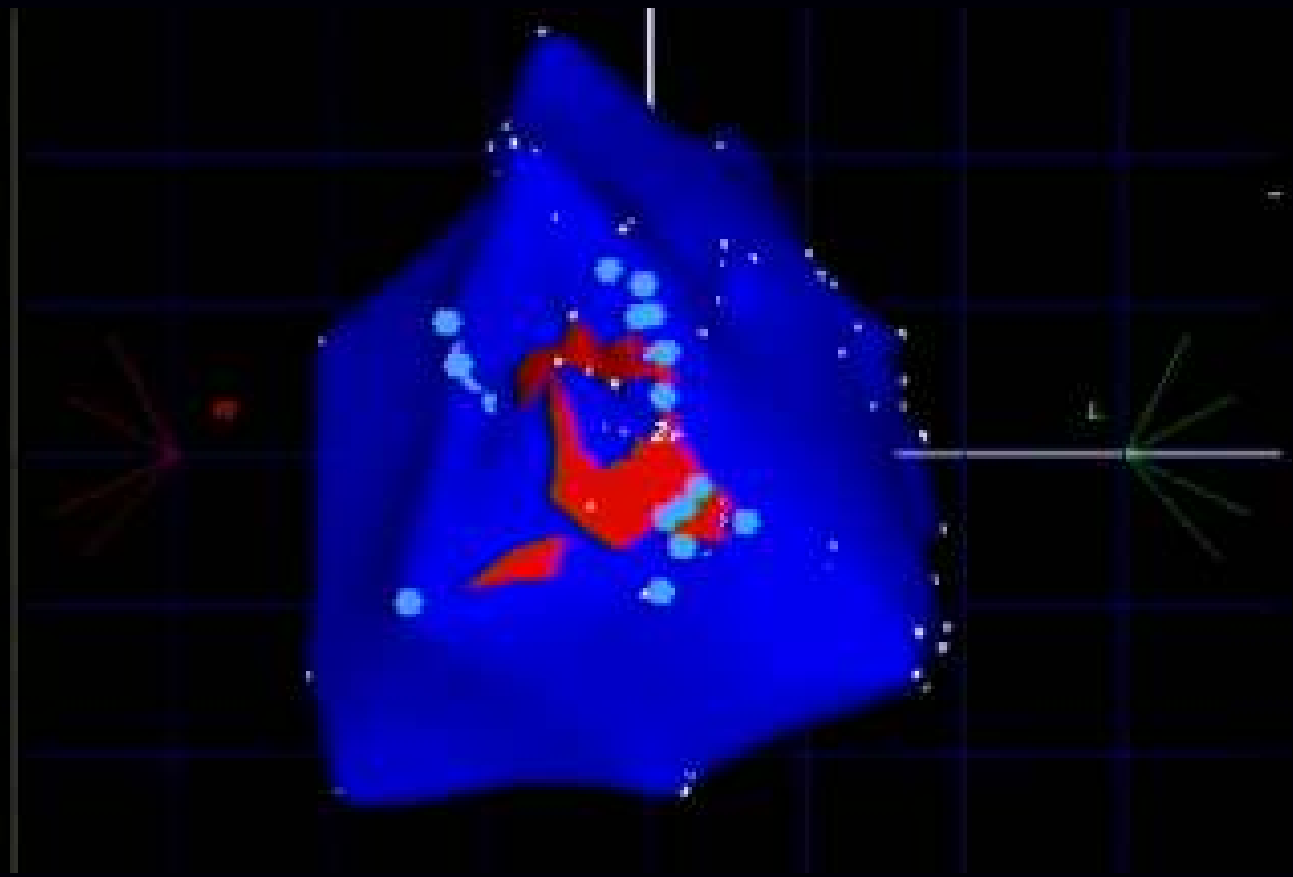
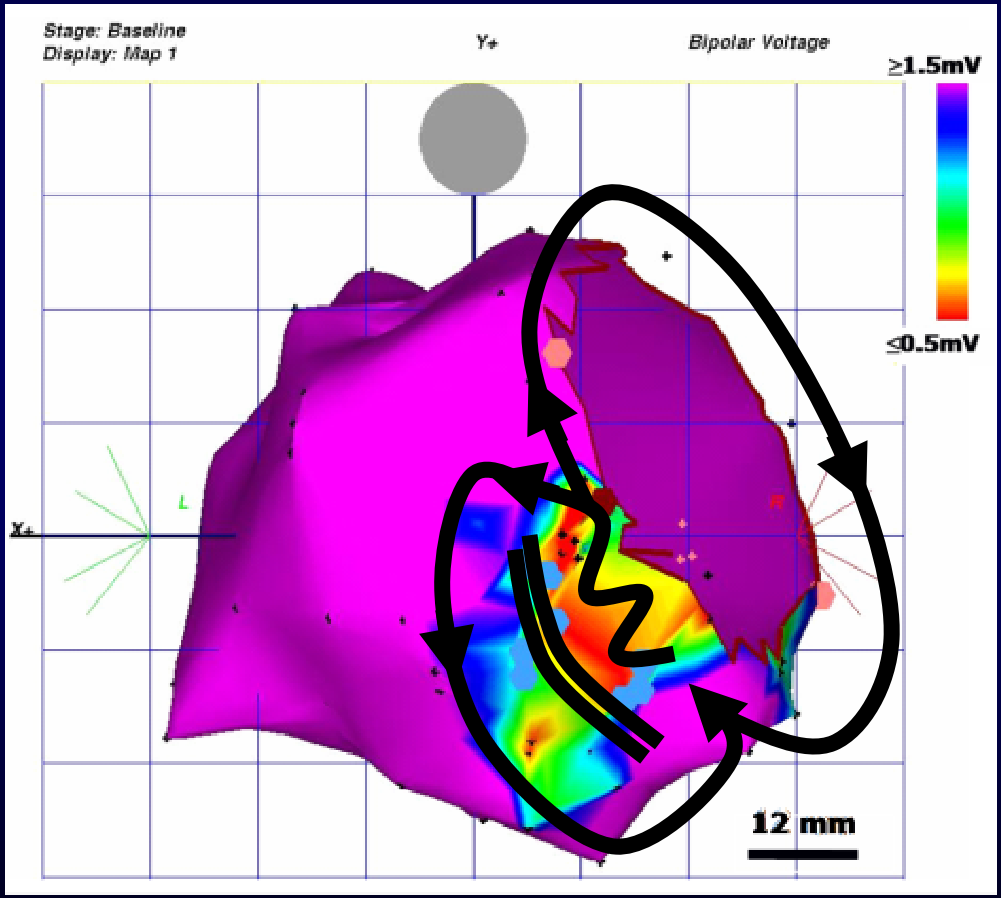
Post-infarct mappable VT ablation

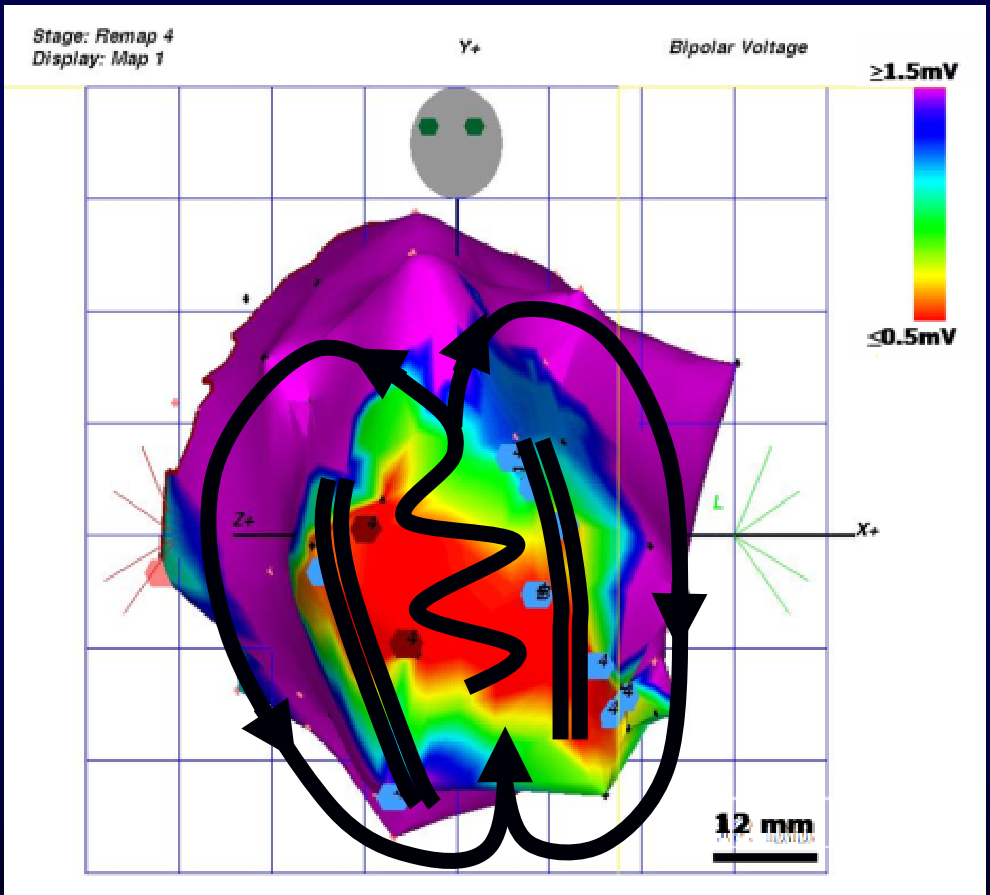
Step # 2 = VT induction & VT mapping



Post-infarct mappable VT ablation

Step # 3 = VT circuit reconstruction





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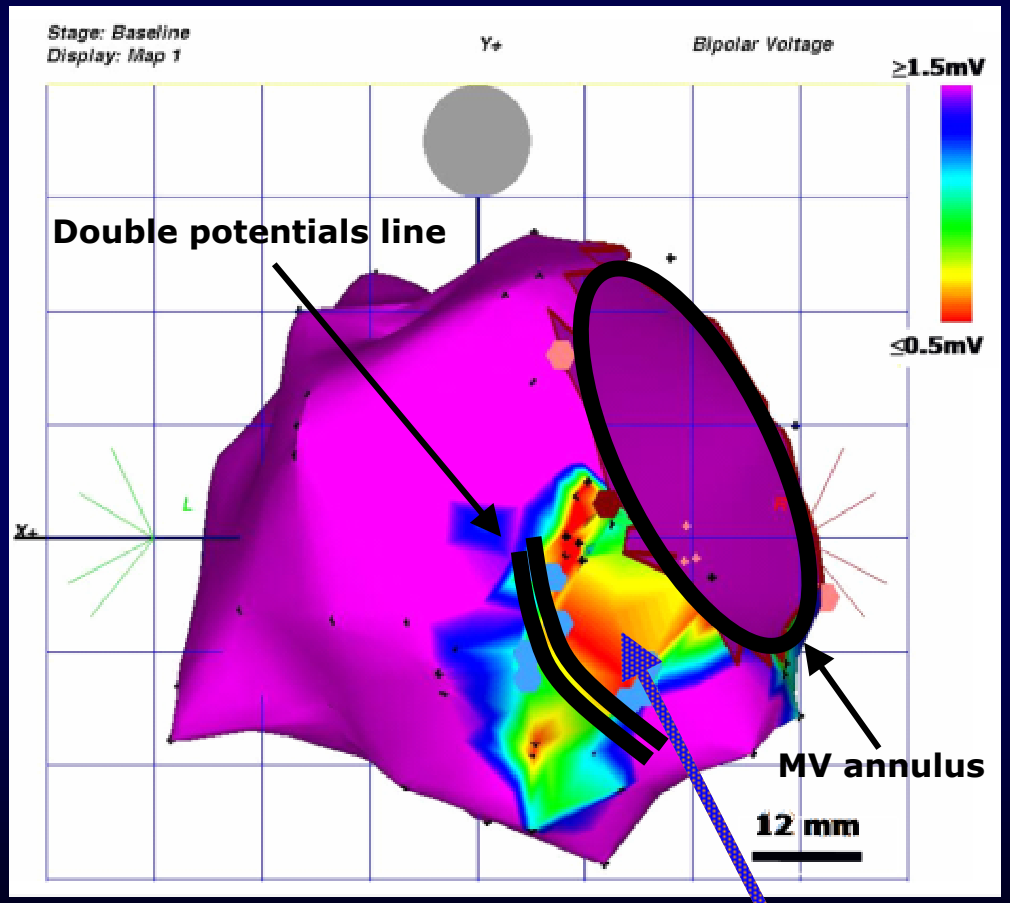
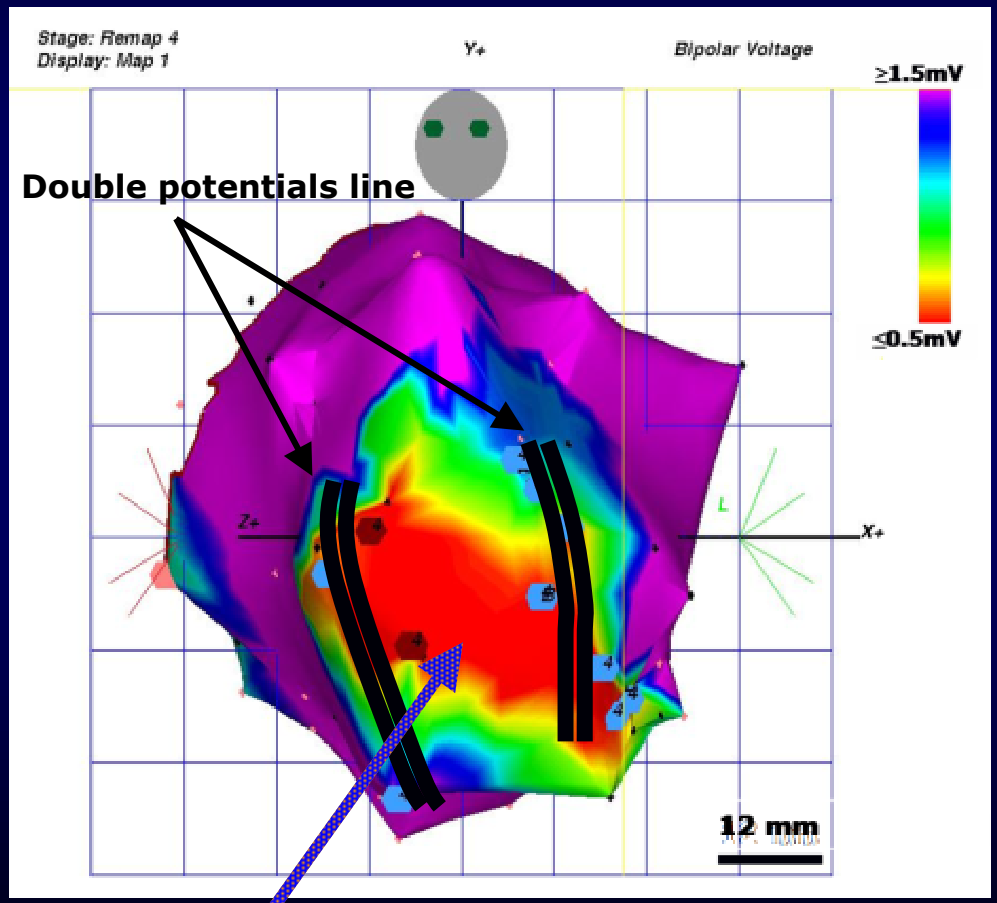
Post-infarct mappable VT ablation

Step # 4 = VT protected isthmus delineation



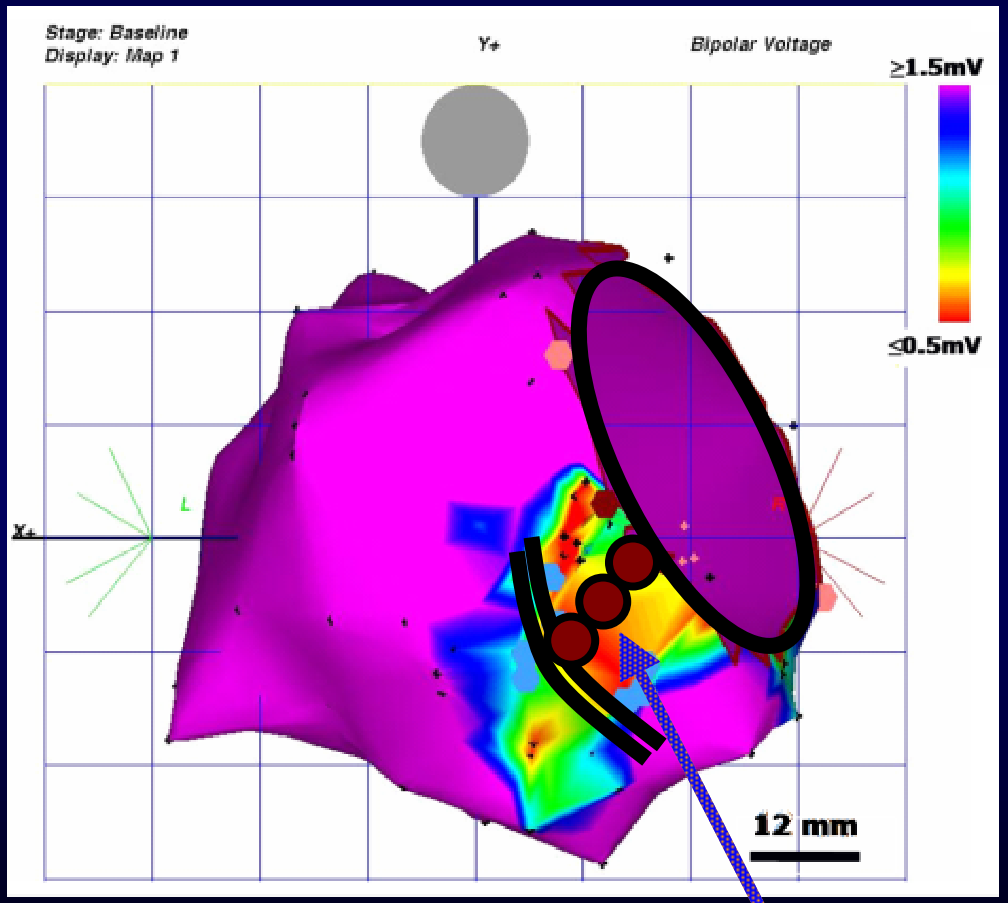


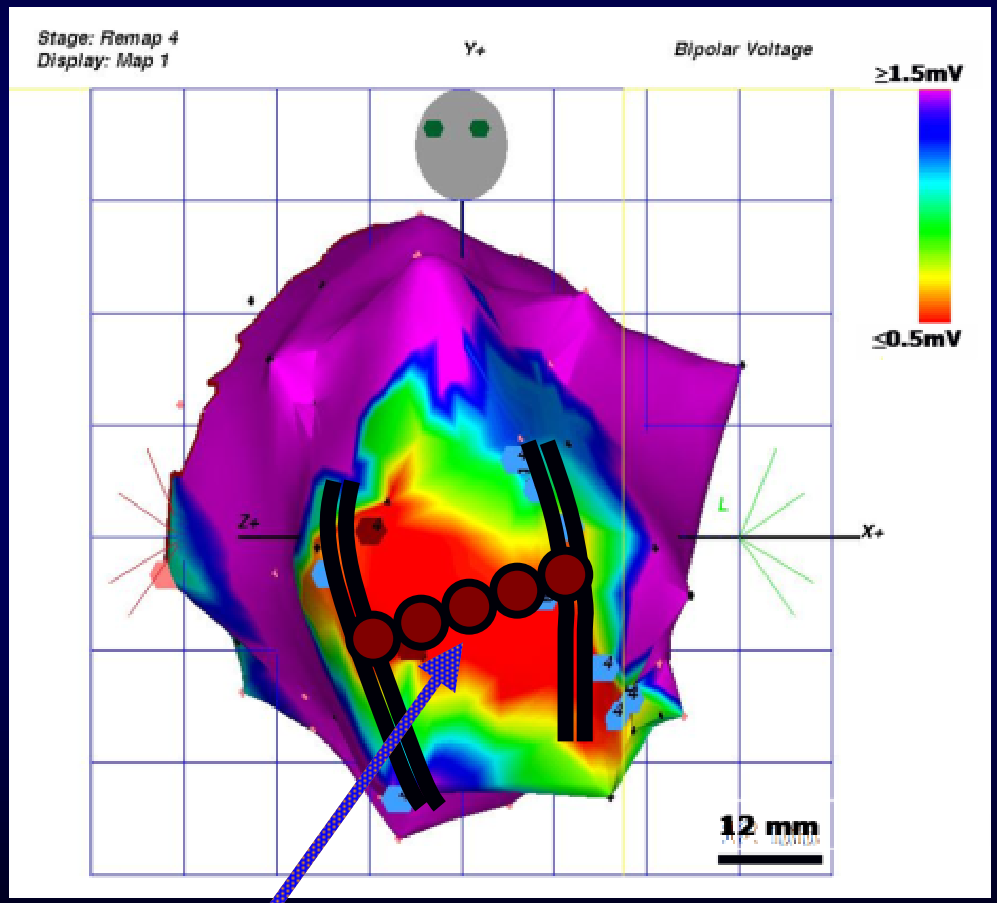
Protected VT isthmus

1

Post-infarct mappable VT ablation

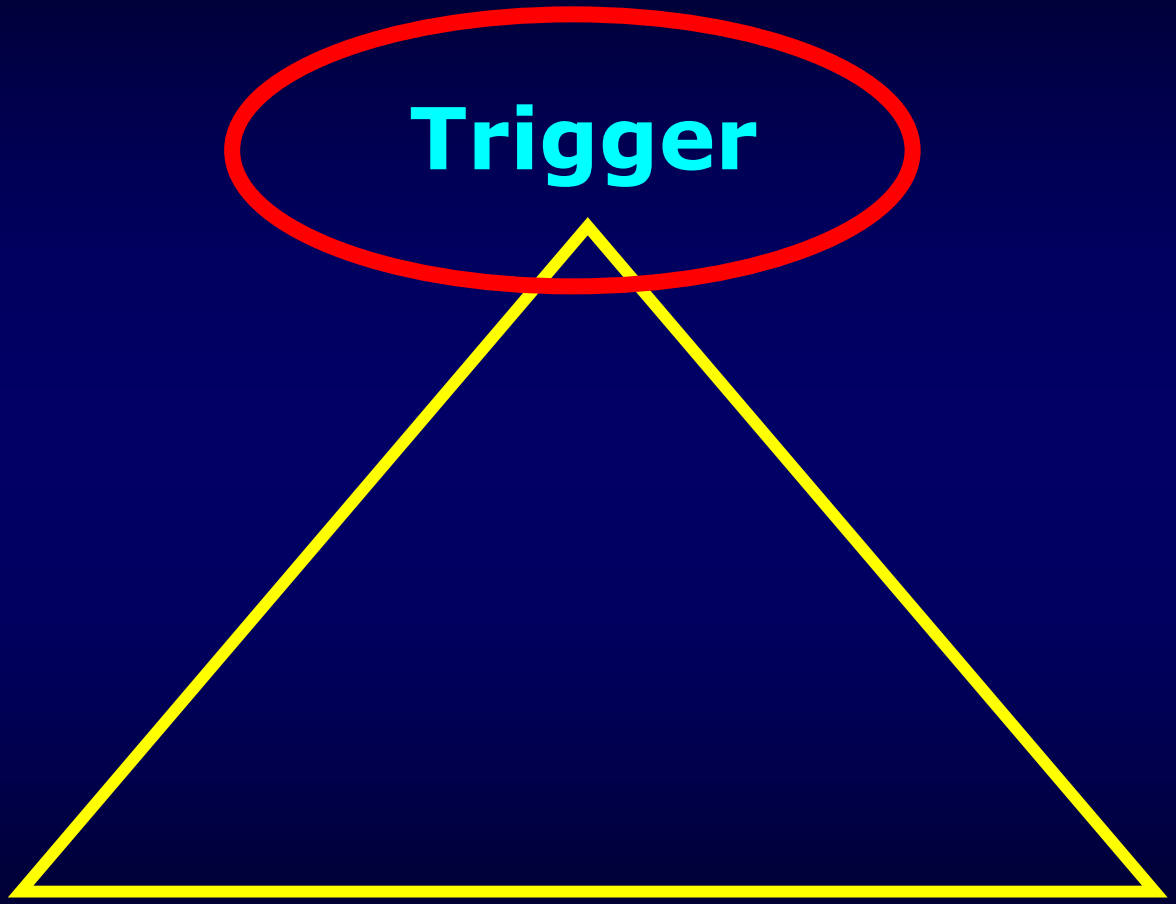
Step # 5 = Ablation ➔ isthmus 'transection'





Ablation lines

Coumel's Triangle

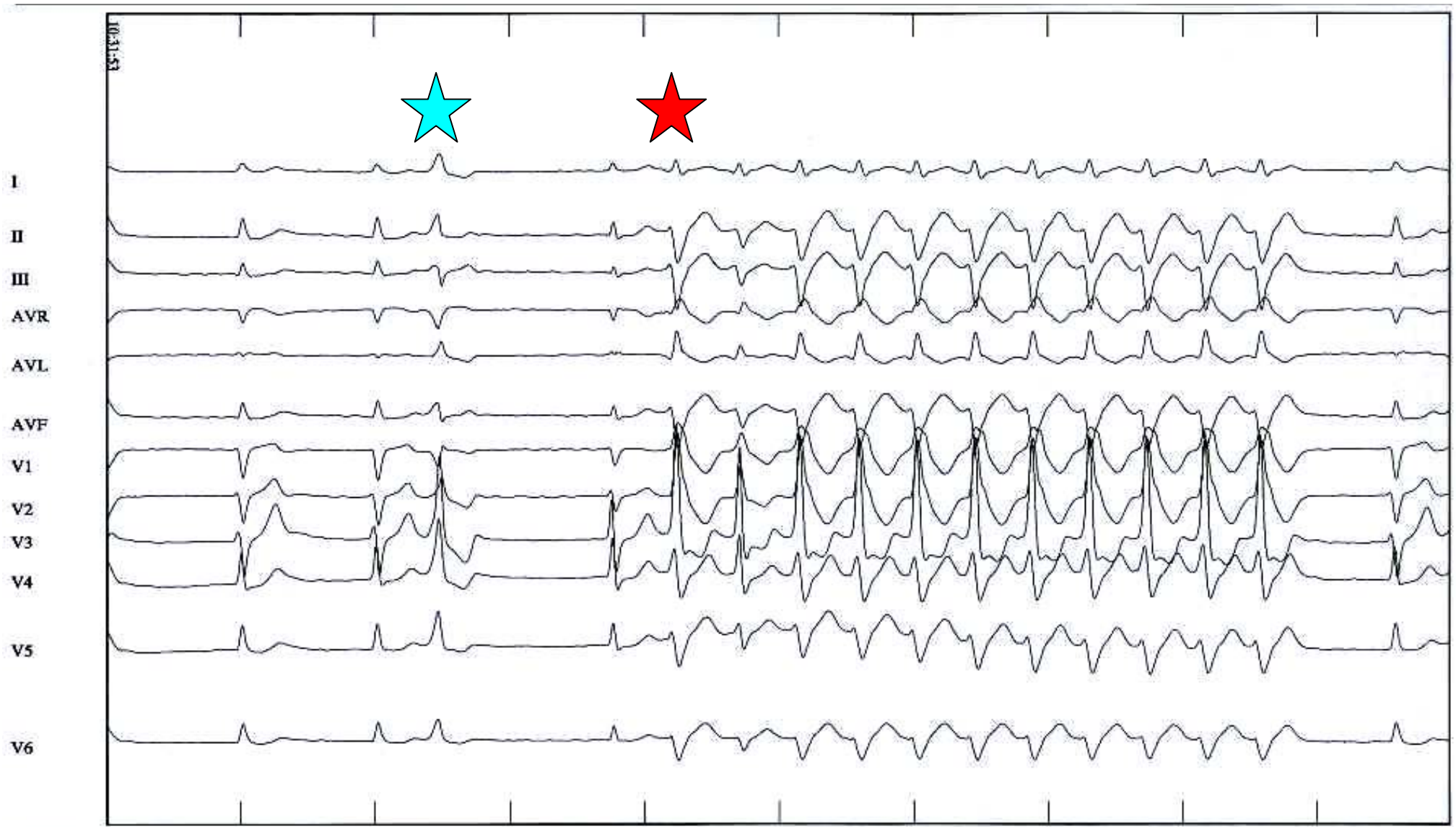


Case Report

- 70 year-old man
- **Chronic AFib** ➔ rate control (severe hyperthyroïdism with amiodarone)
- Acute **infero-lateral myocardial infarction** (04/2000)
- No acute revascularization procedure (late in-hospital admission)
- LVEF = 0.55 - One vessel disease = **Cx occluded** ➔ delayed PTCA
- Documented **syncopal VT** (220/mn) ➔ **ICD implantation** (03/2001)
- Many fast VT (>200/mn) recurrences efficiently treated by ATP
- Nadolol added: 80mg/d
- July 2001 = fast VTs (240/mn) ➔ **VT storms : 12 shocks / 5 days**

1

1



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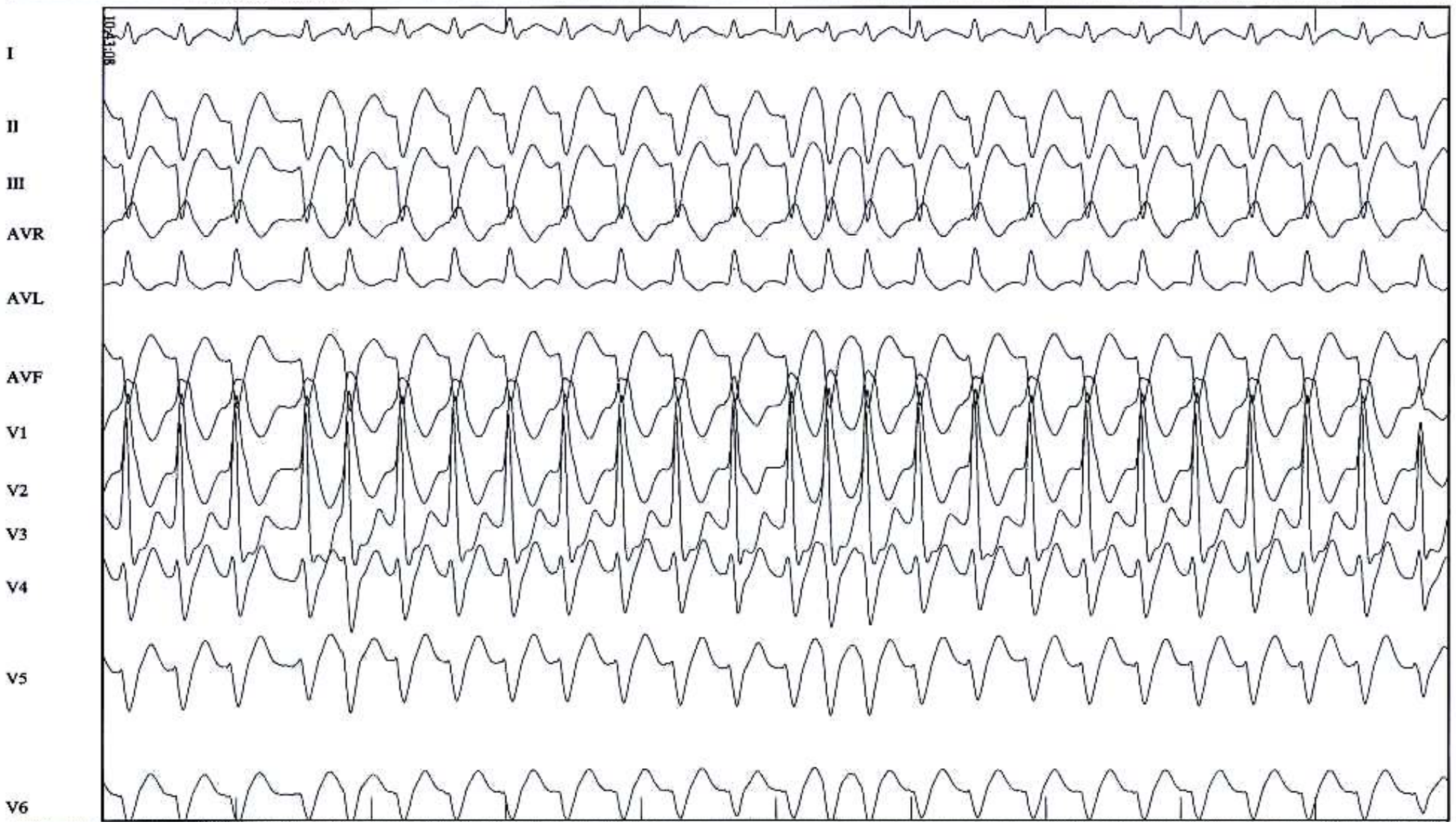
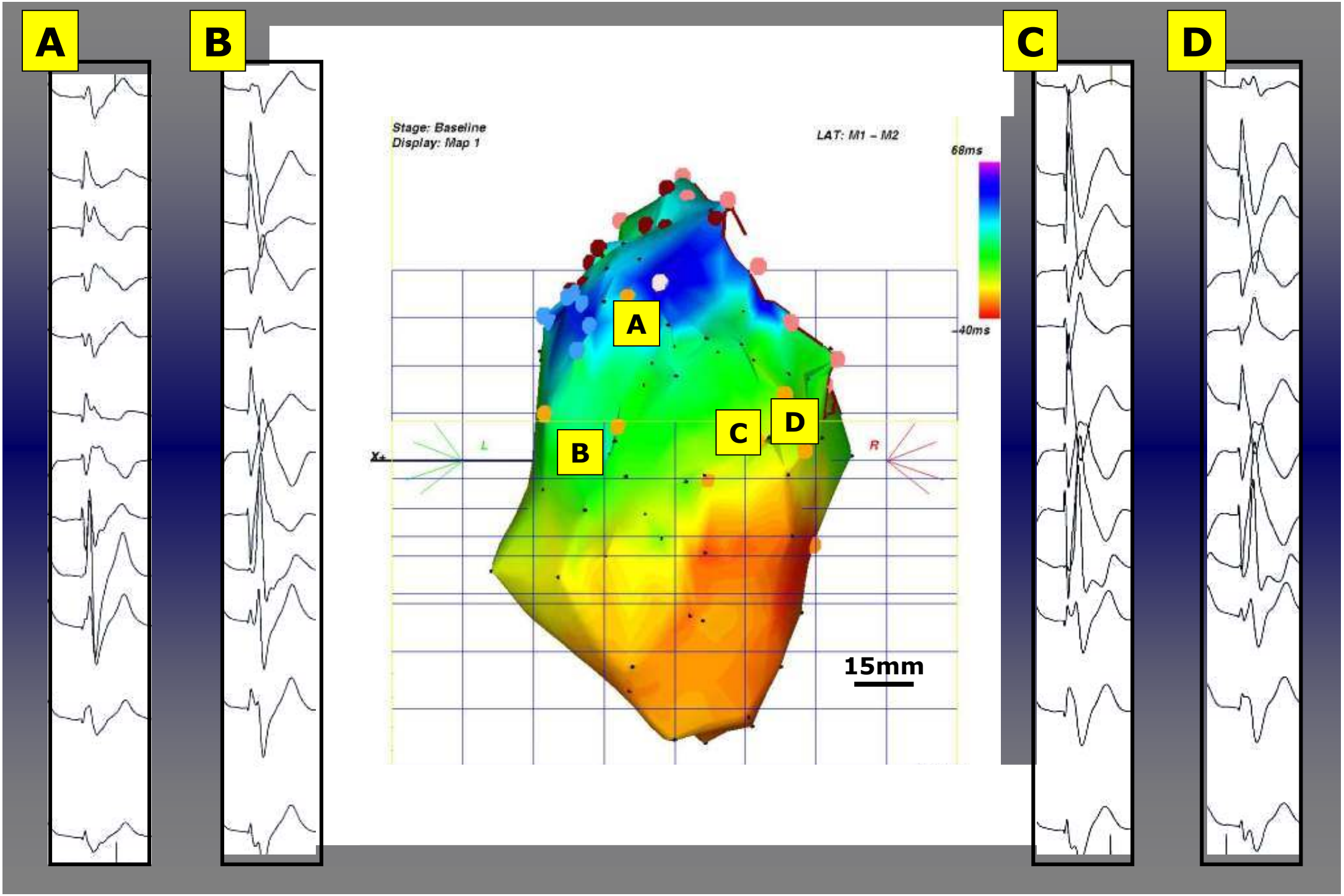
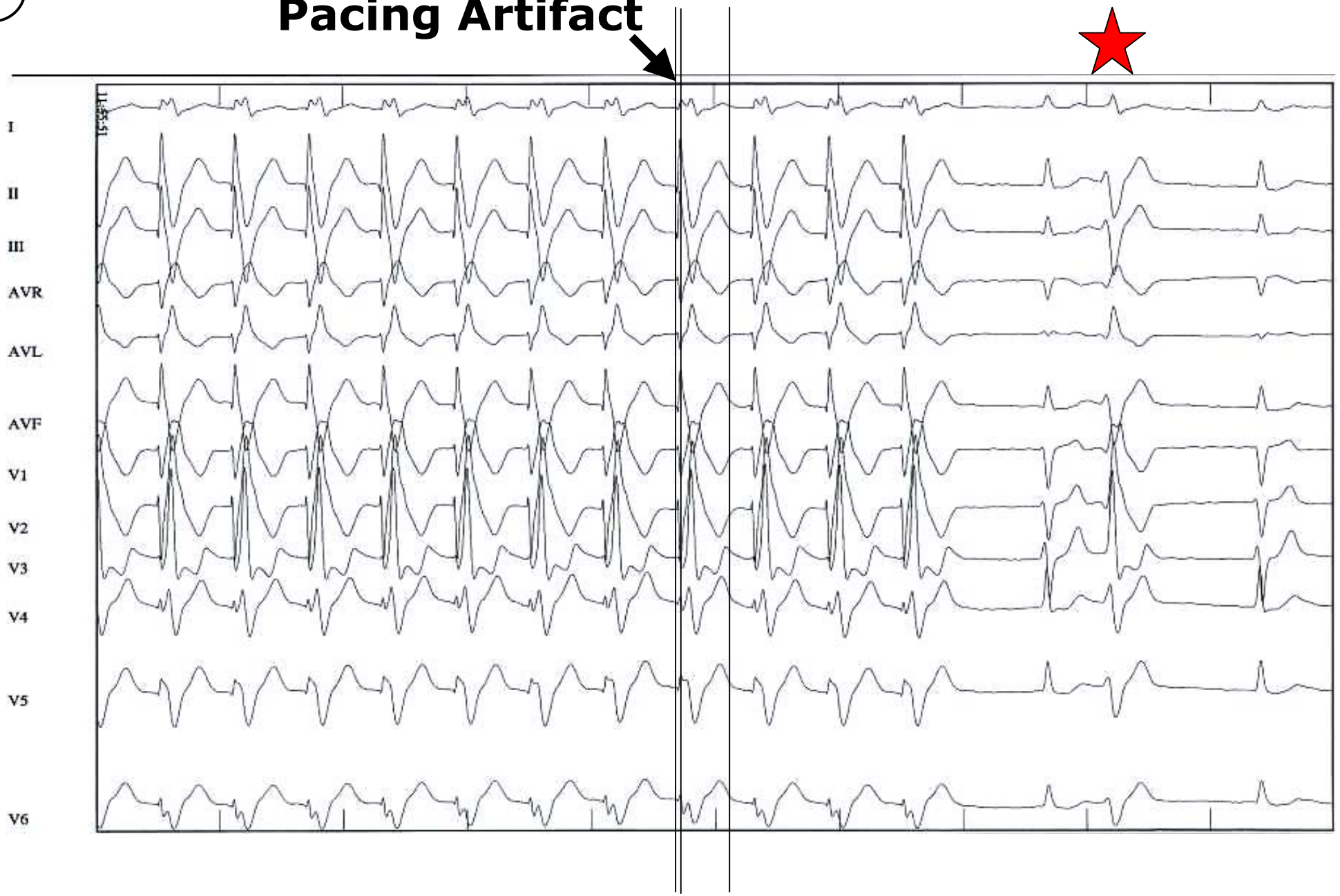



A
B
C
D
Stage: Baseline
Display: Map 1
LAT: M1 – M2
68ms
-40ms
A
B
C
D
L
R
15mm

1



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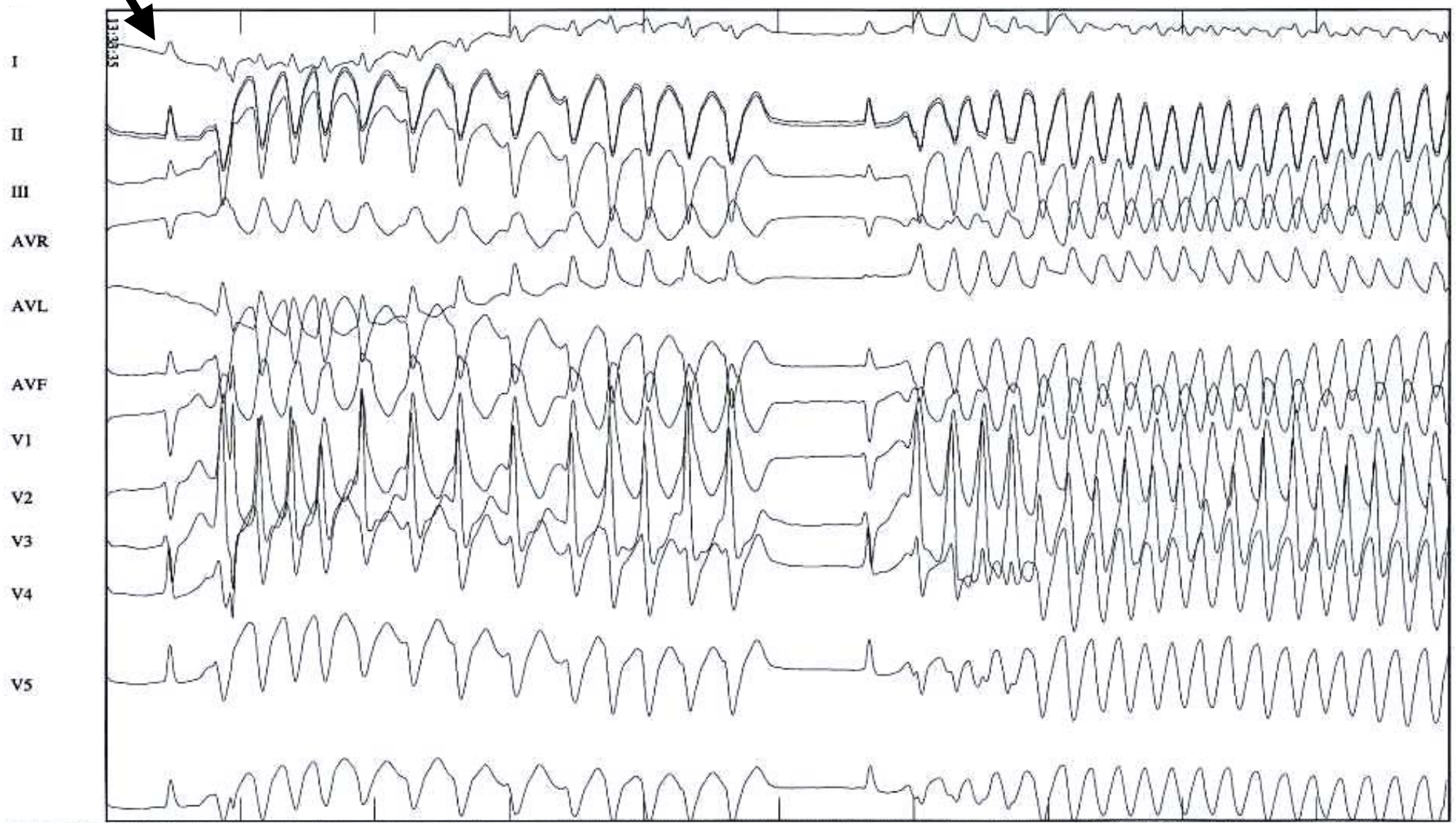
RF onset



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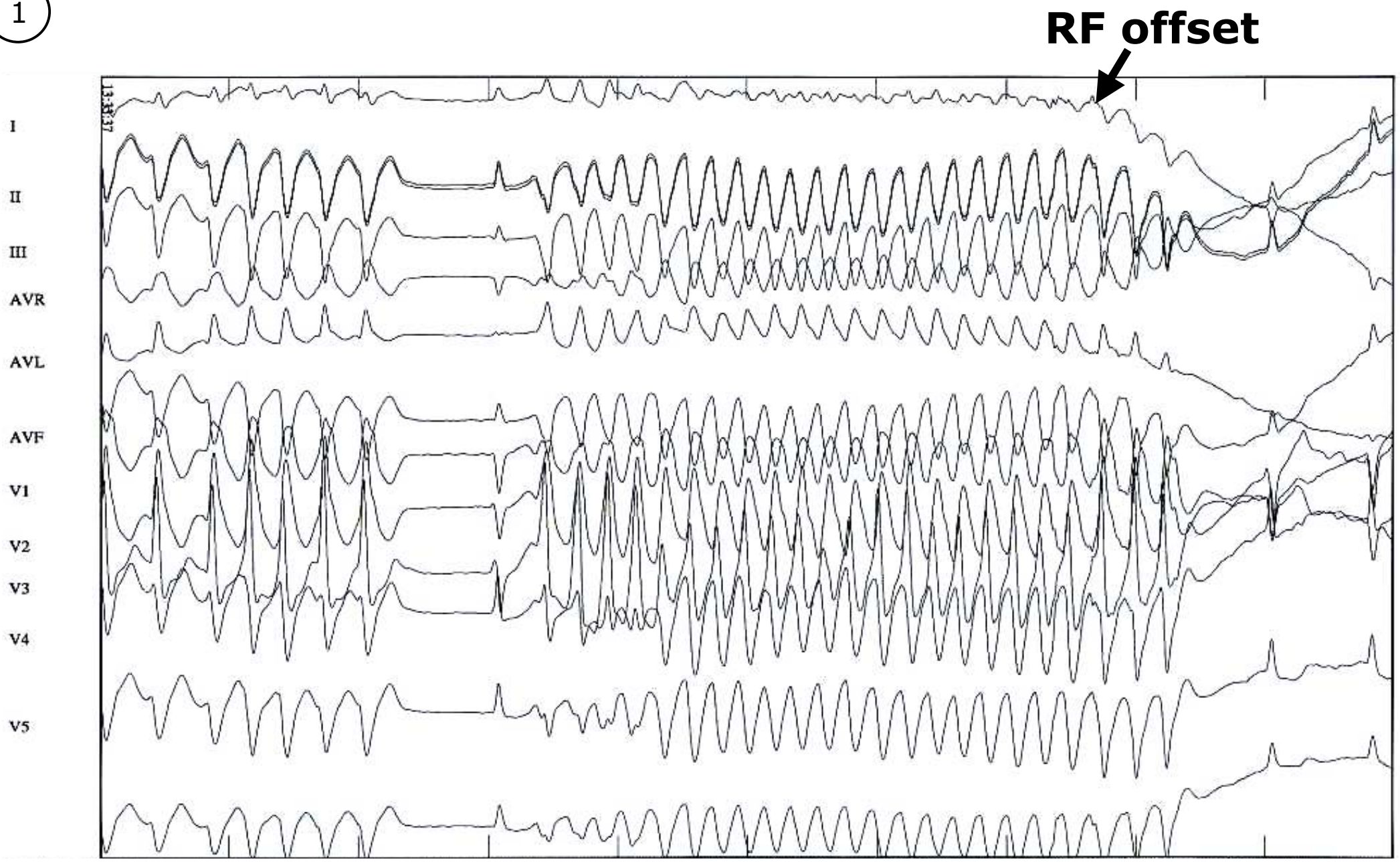

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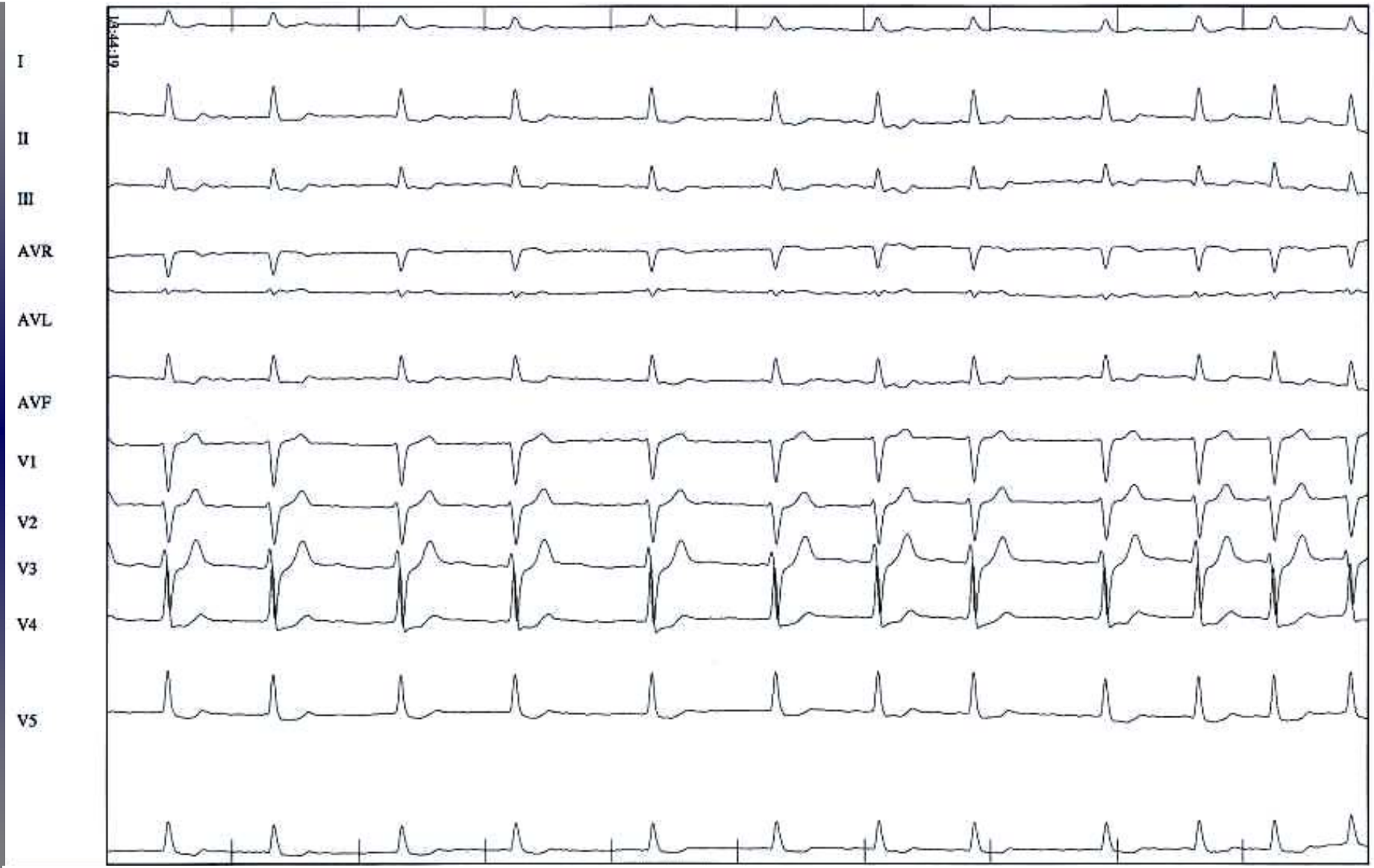
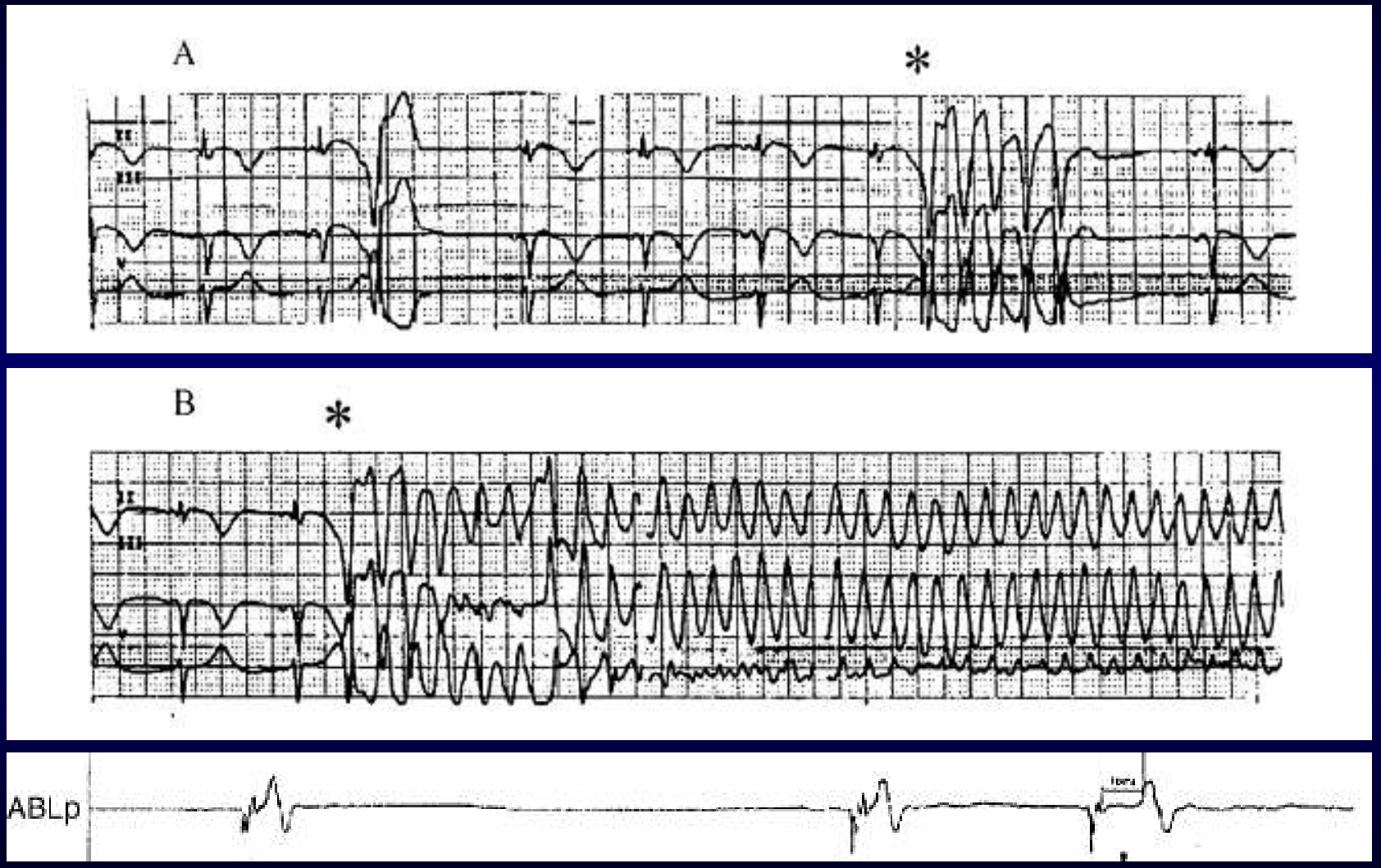


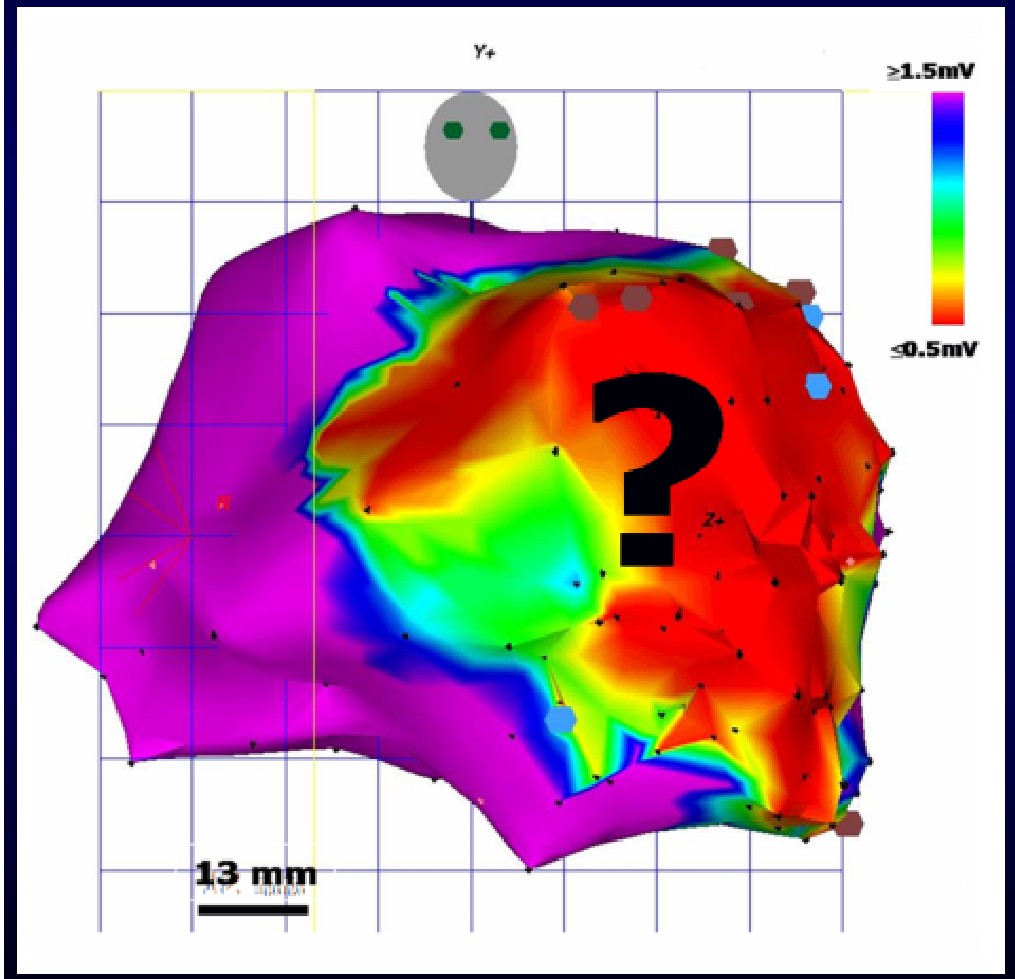
Myocardial infarction : late phase

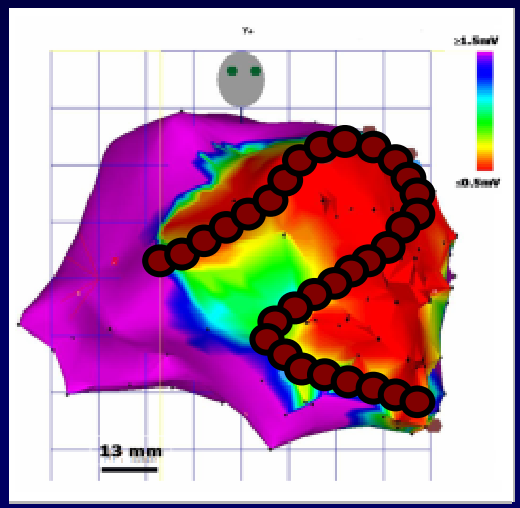


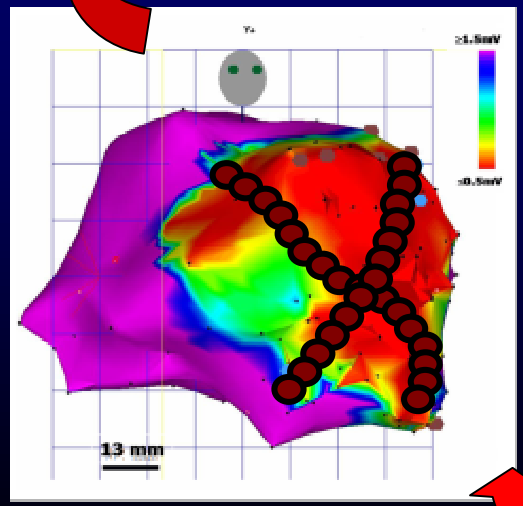
Mode of initiation and ablation of VF storms in patients with ischemic cardiomyopathy
Marrouche N. et al., J Am Coll Cardiol 2004;43:1715-20

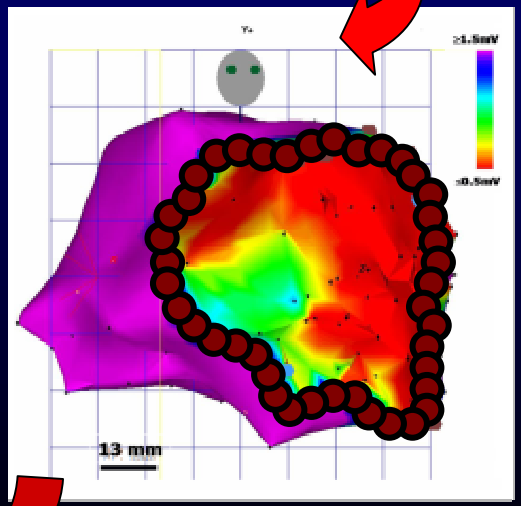
Post-infarct *non mappable* VT ablation

Different possible empiric approaches



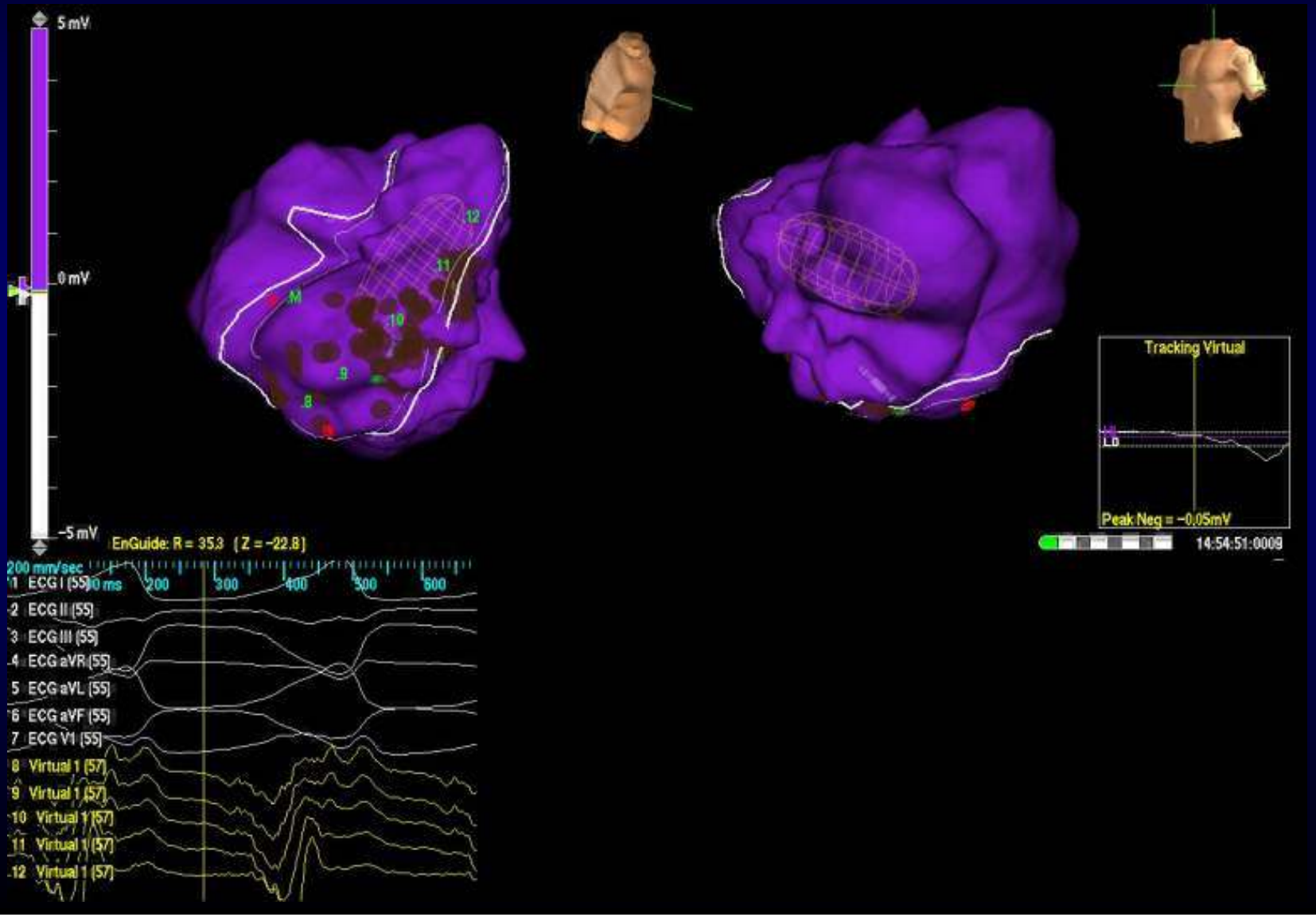






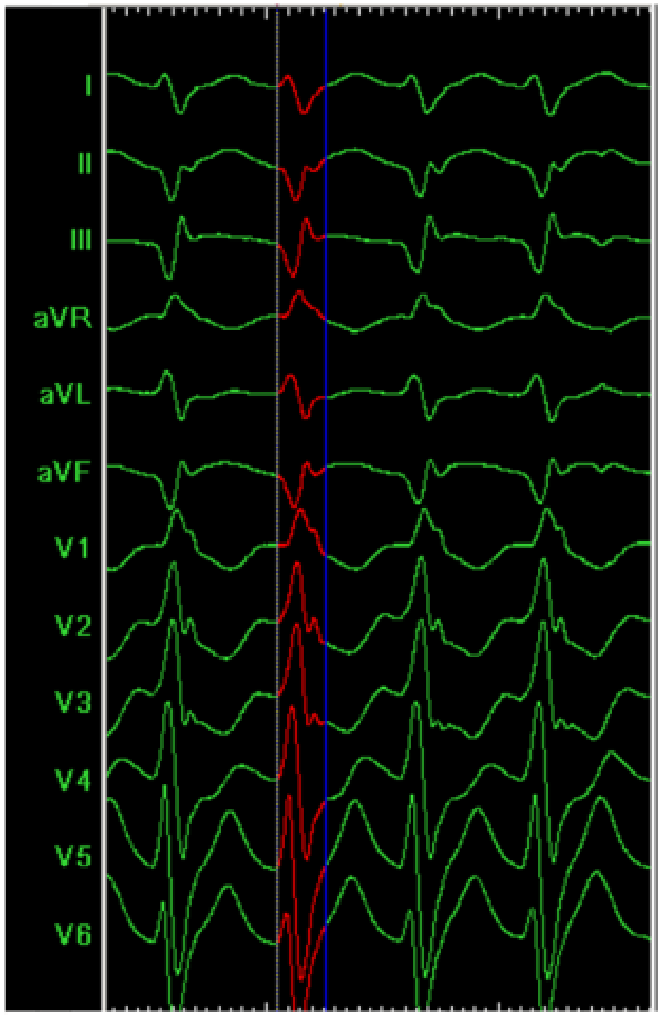
1

Post-infarct non mappable VT ablation
➔ non contact mapping

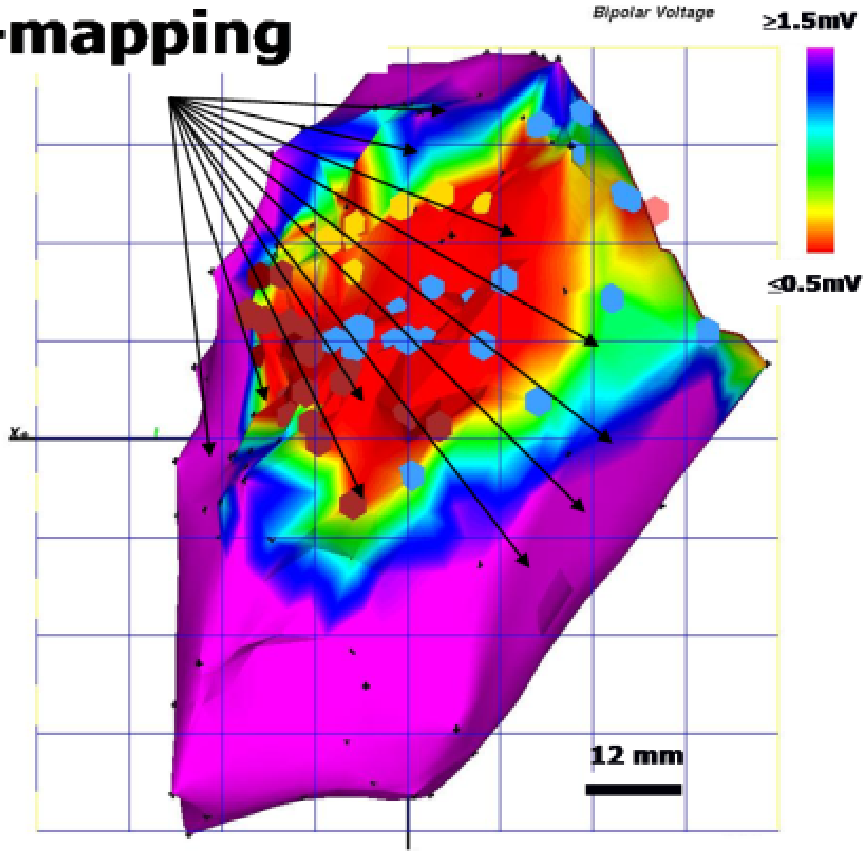


Post-infarct *non mappable* VT ablation

Step # 1 = substrate mapping & pace mapping



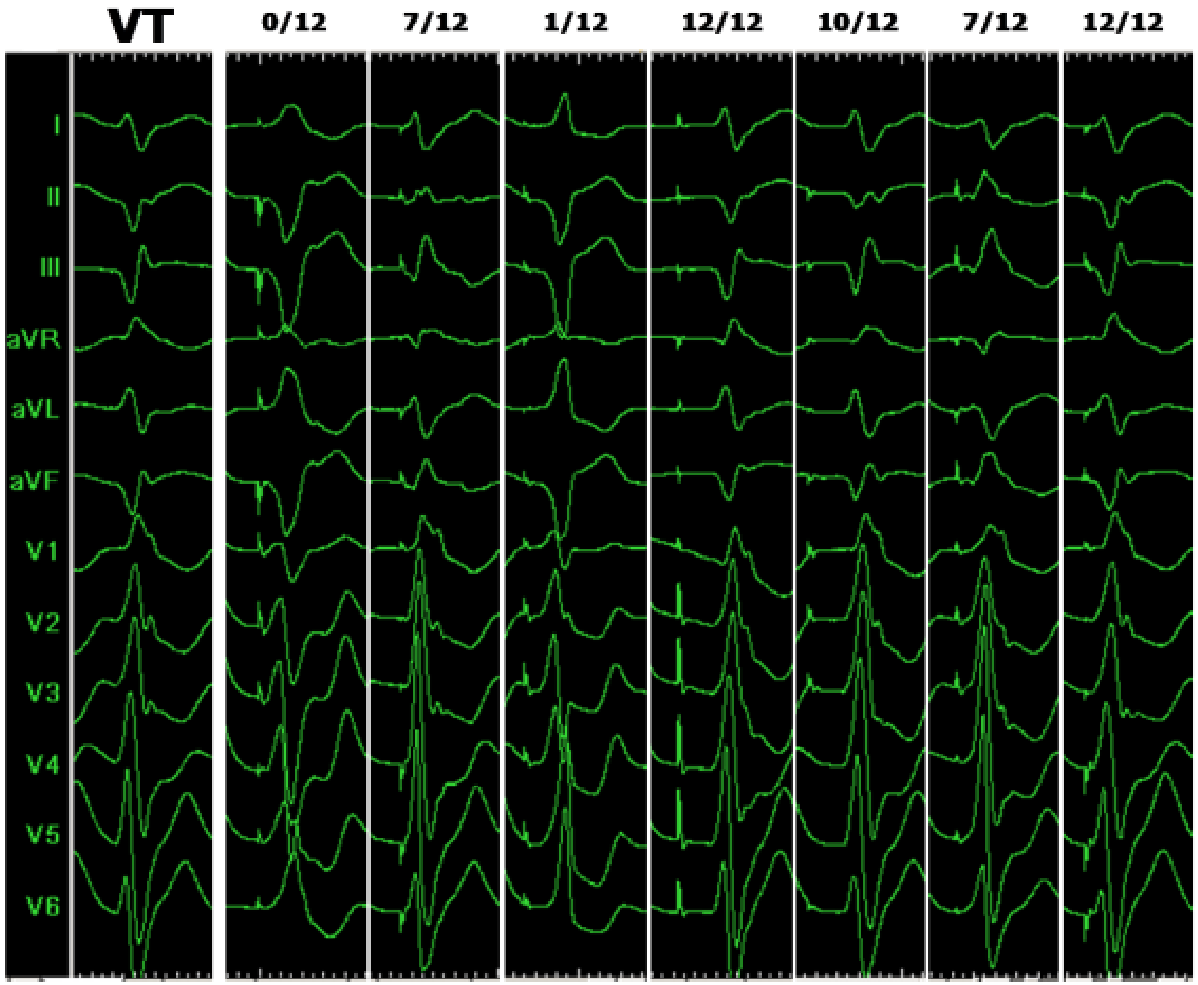
Pace-mapping

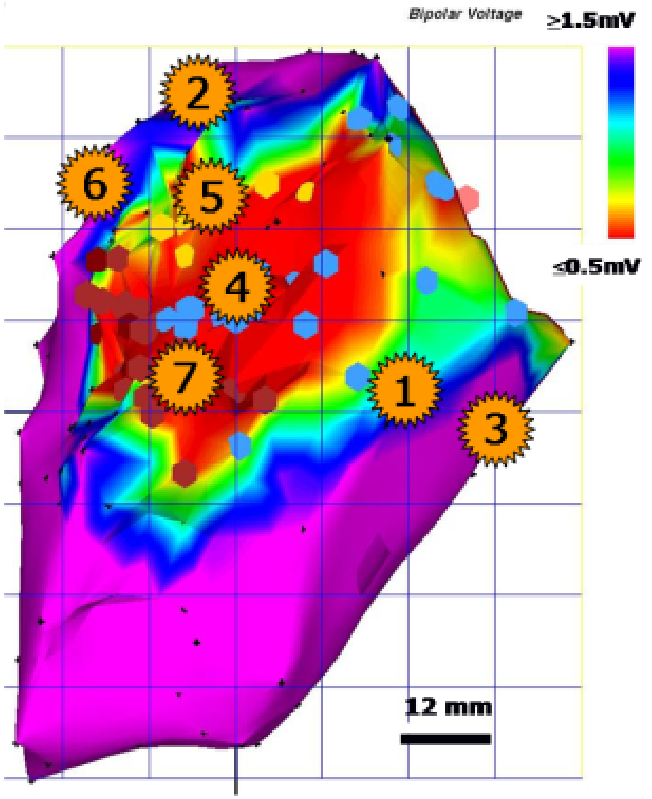


Kottkamp et al. JCE 2003;14:675-81
Brunckhorst et al. Circulation 2004;14:675-81

Post-infarct *non mappable* VT ablation

Step # 2 = matching w. clinical VT 12-lead ECG

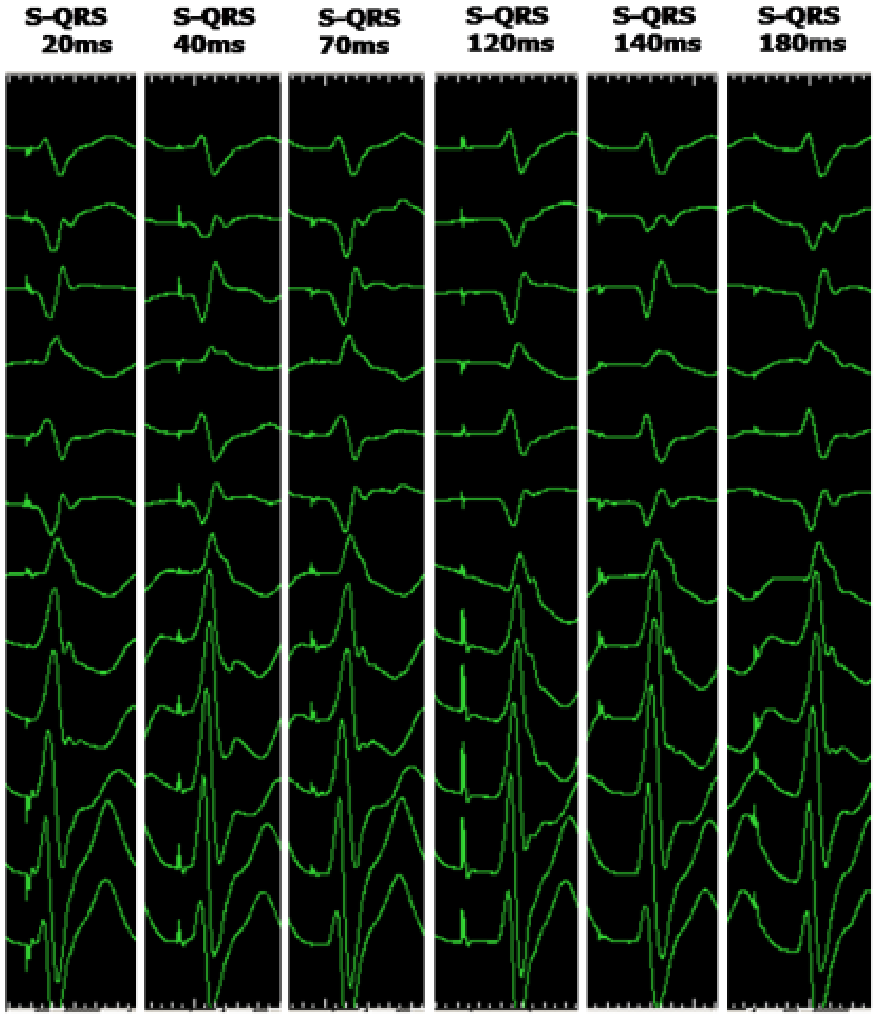


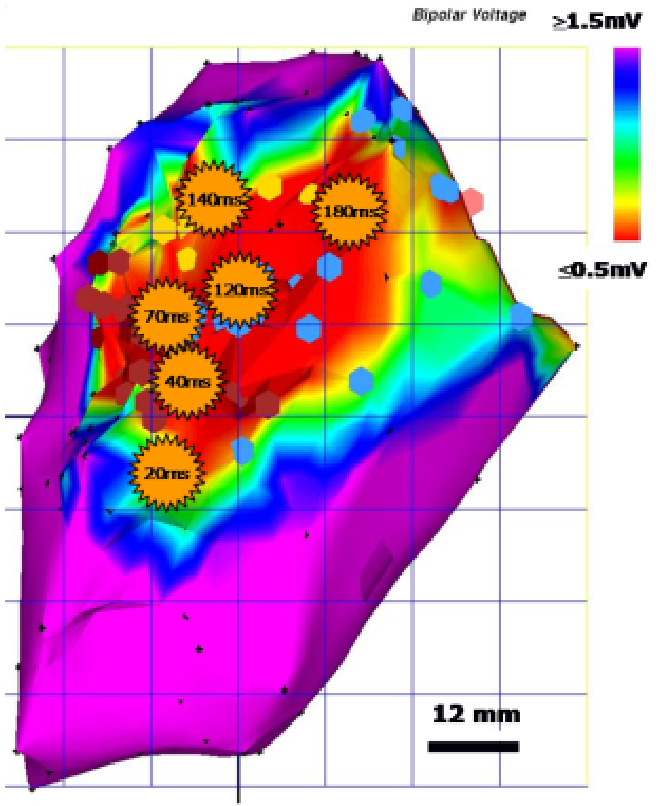


Post-infarct *non mappable* VT ablation

Step # 3 = Ranking the matching mapping sites

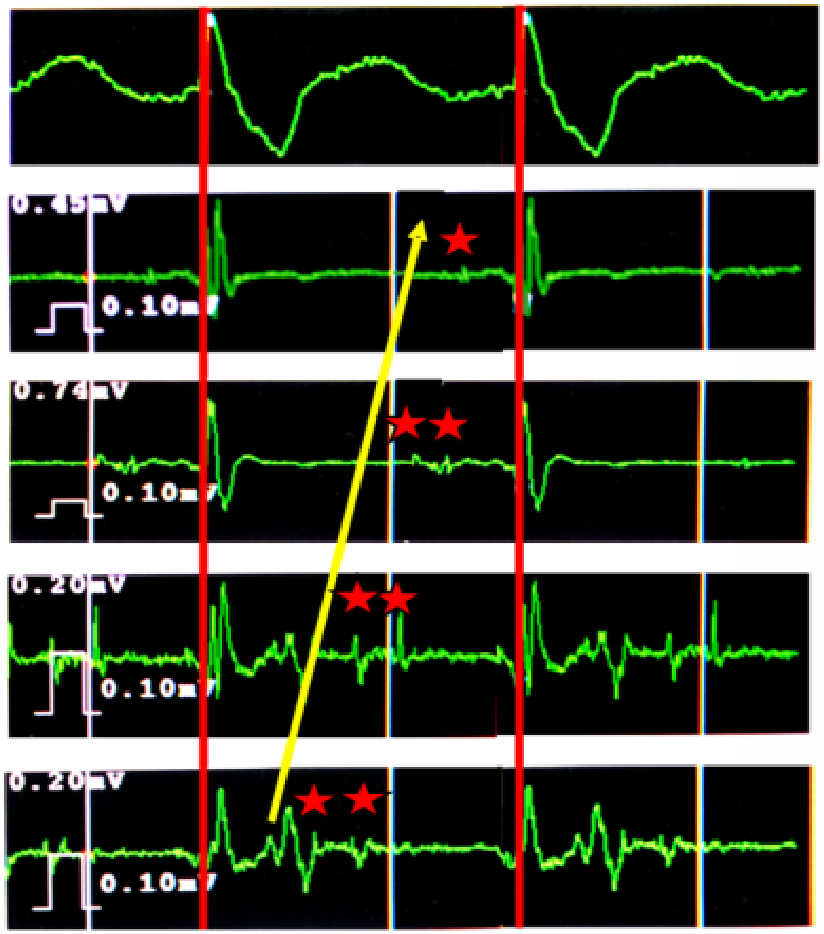
RANKING



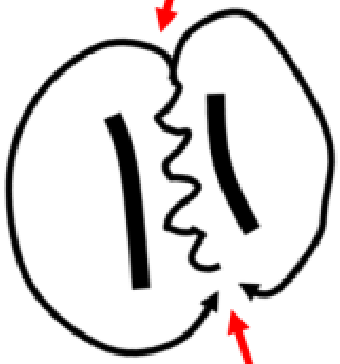


Post-infarct *non mappable* VT ablation

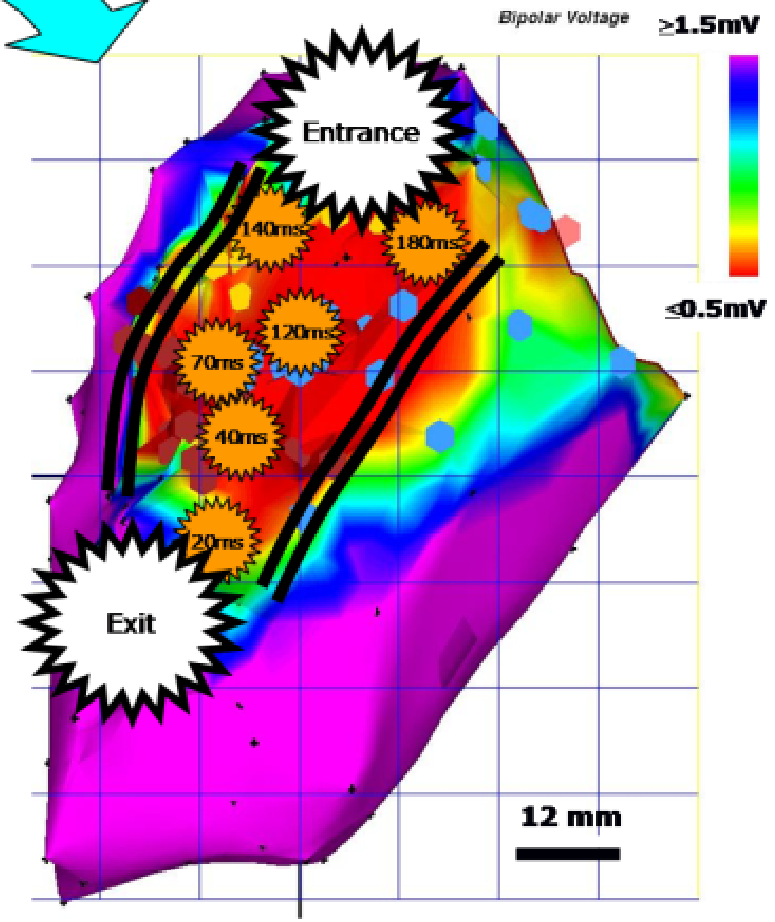
Step # 4 = VT protected isthmus definition



Exit
Short S-QRS

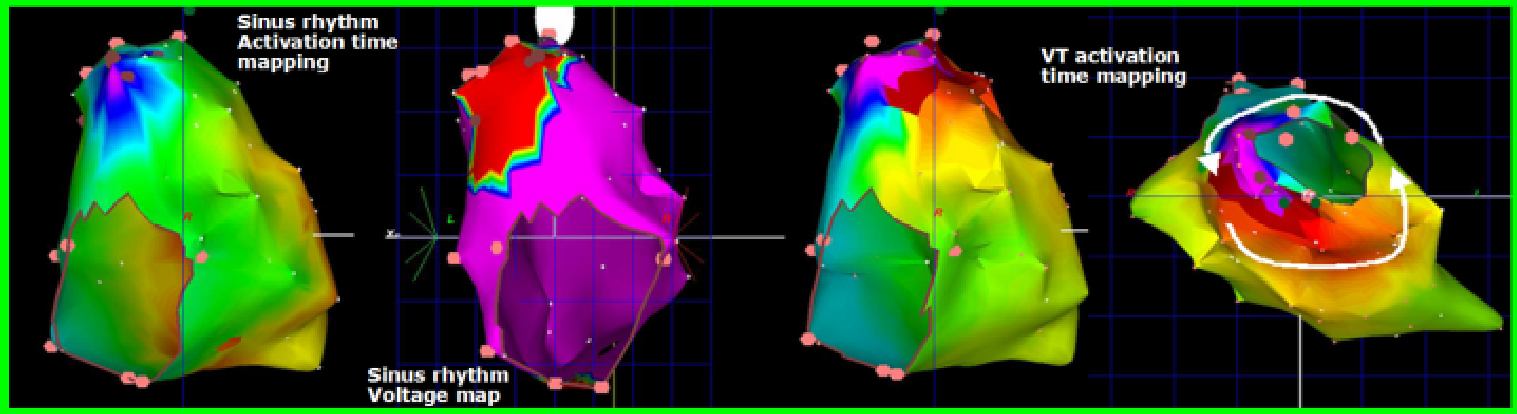


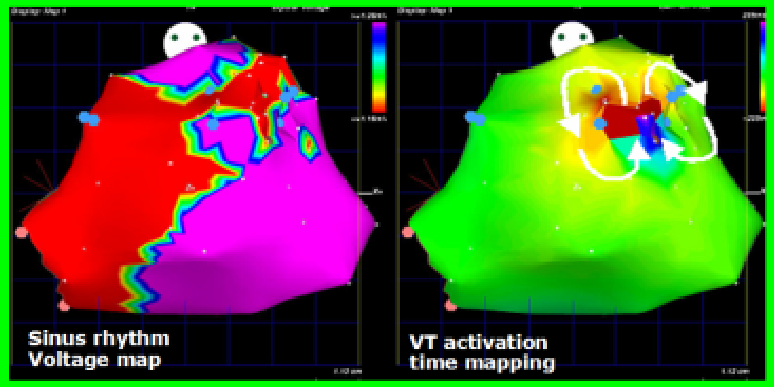
Entrance
Long S-QRS



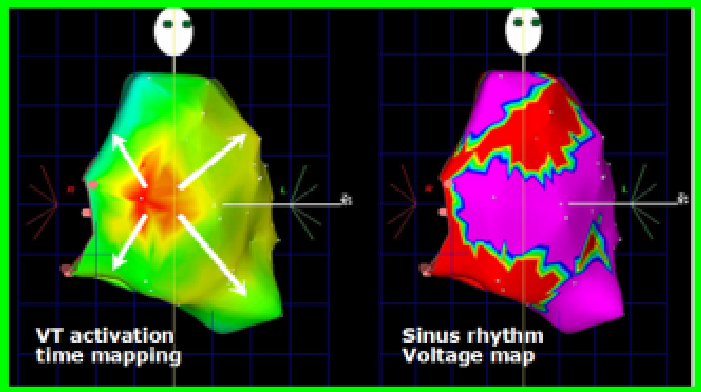
Endocardial reentrant circuit = 66% of ARVD/C related mappable VTs

Reentrant VT pattern = 66%



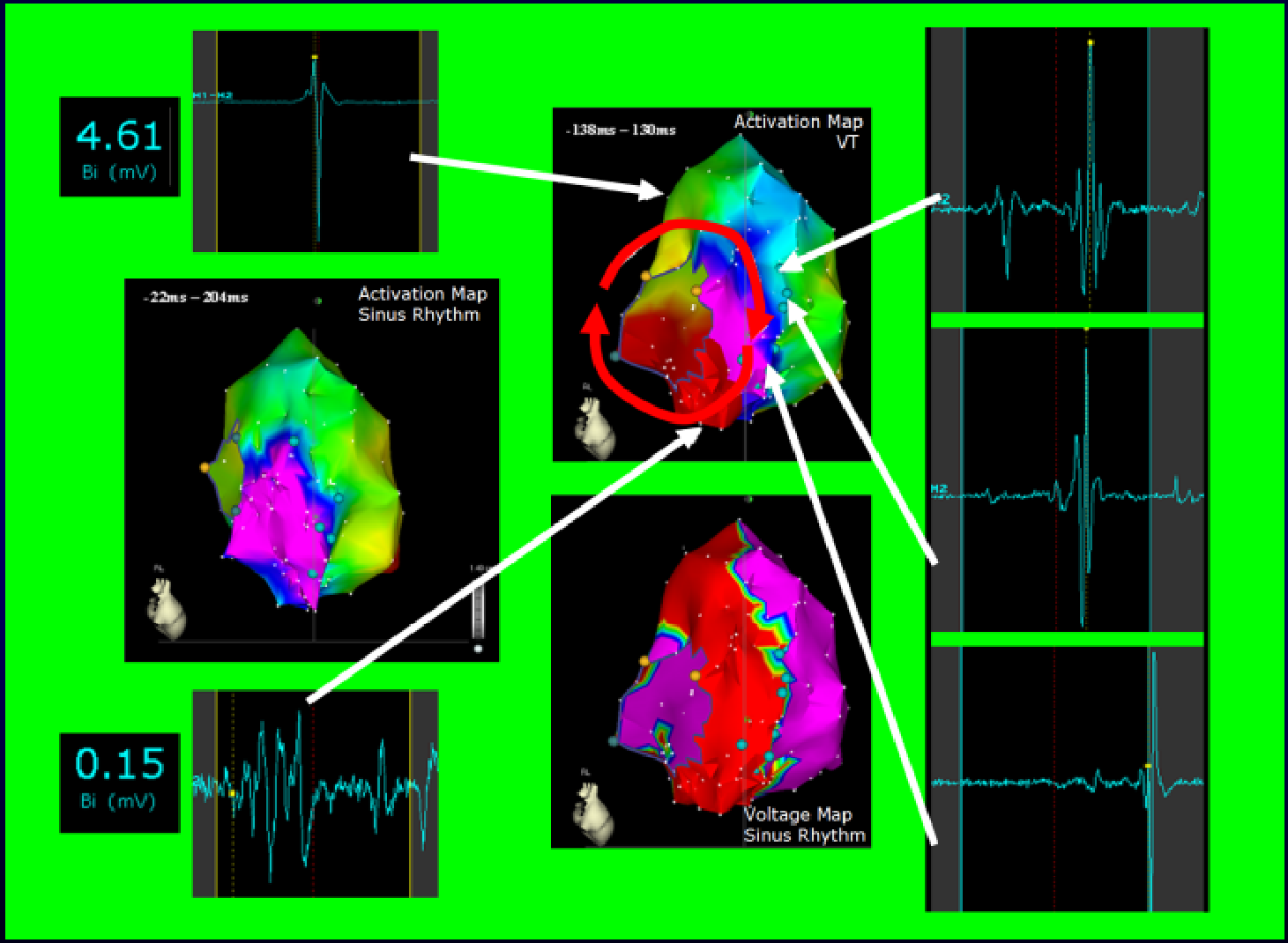


Focal VT pattern = 33%



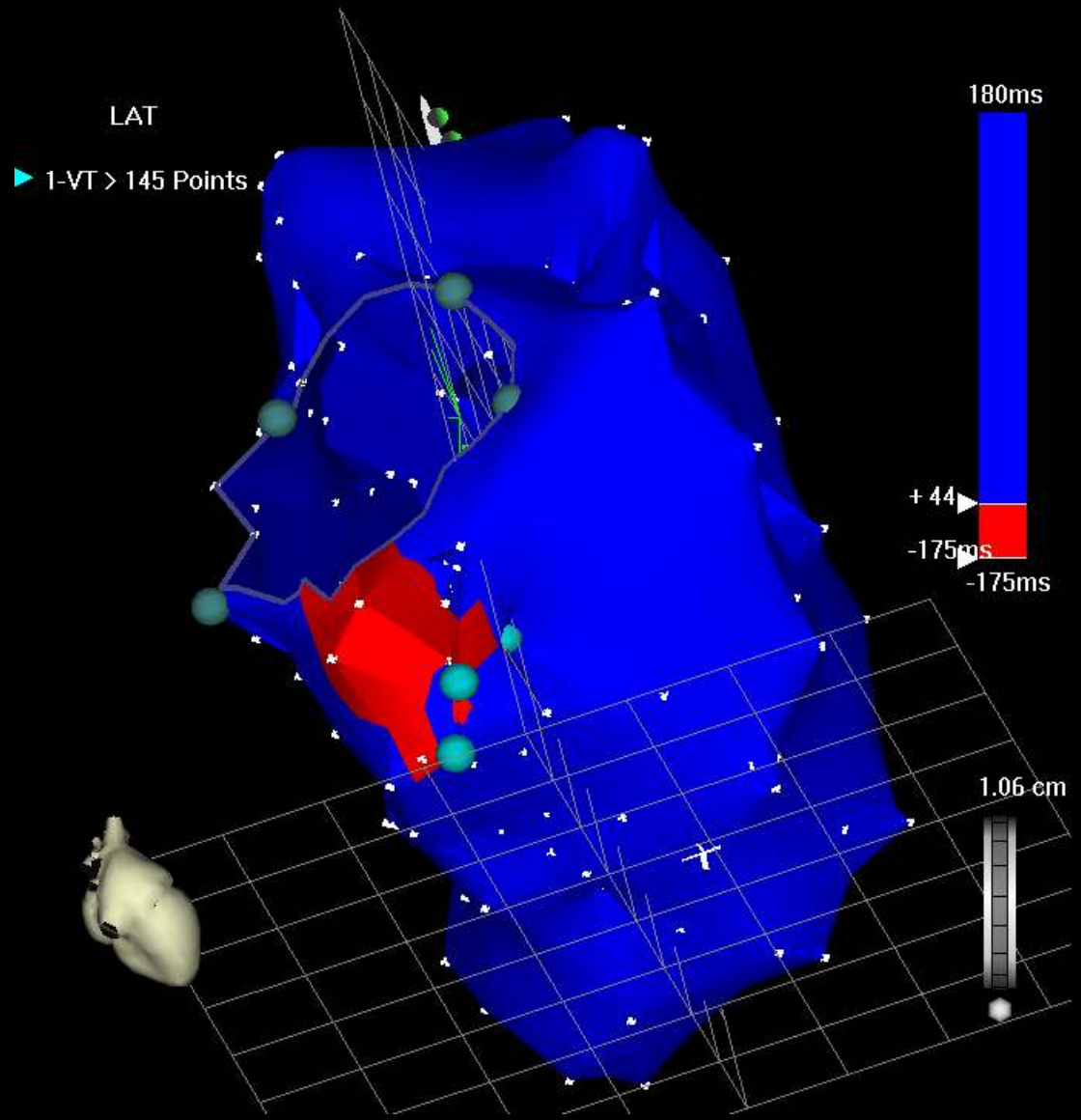
Miljoen H, de Chillou C et al. Europace 2005;7:516-24

Peri-tricuspid reentrant VT : a frequent mechanism in ARVD/C reentrant VTs (5 of 12 VTs)

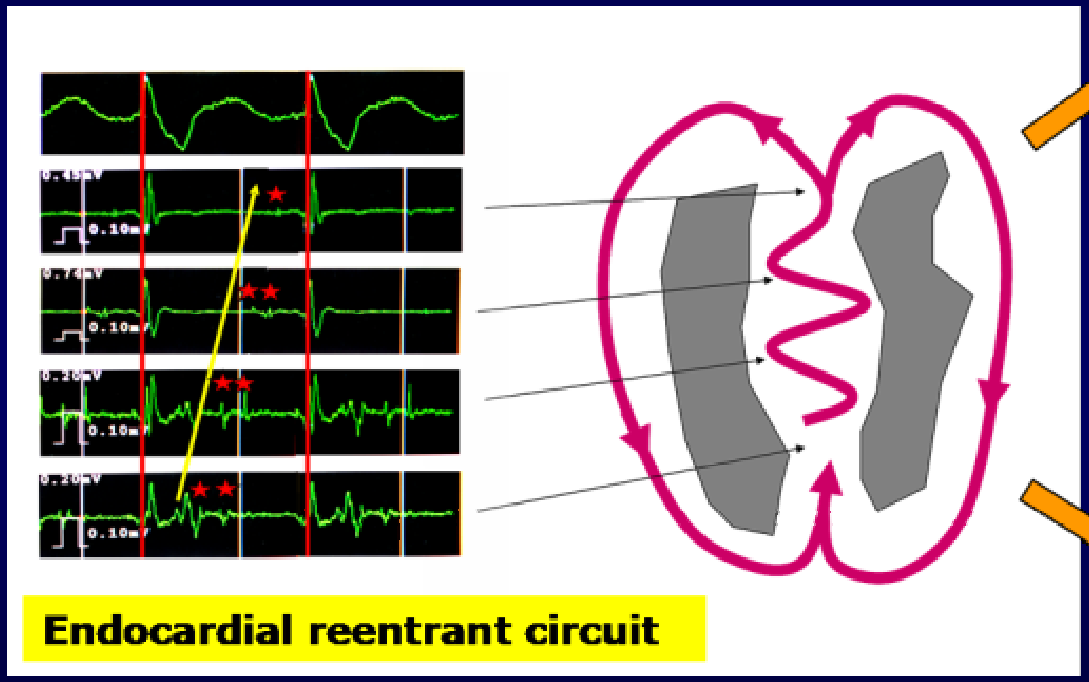


Miljoen H, de Chillou C et al. Europace 2005;7:516-24

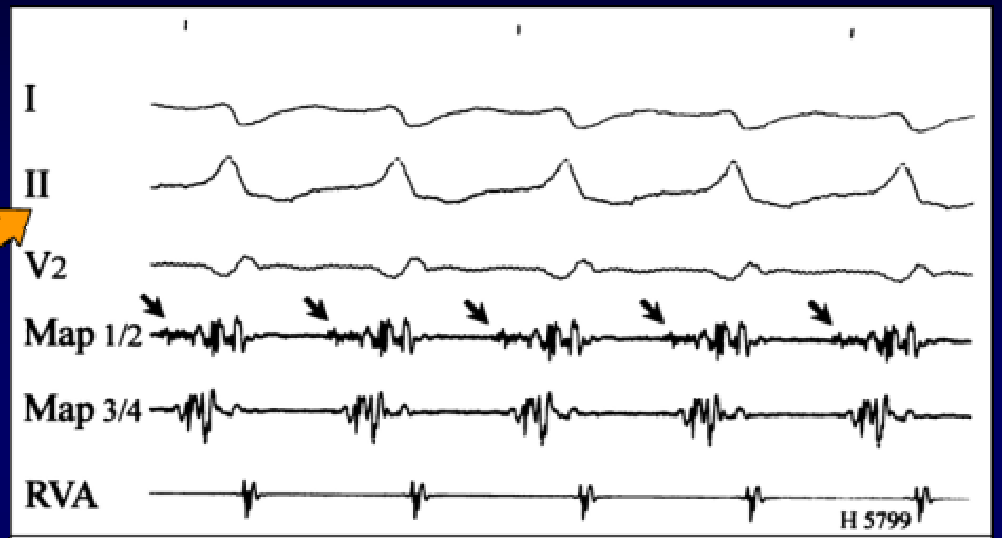
2

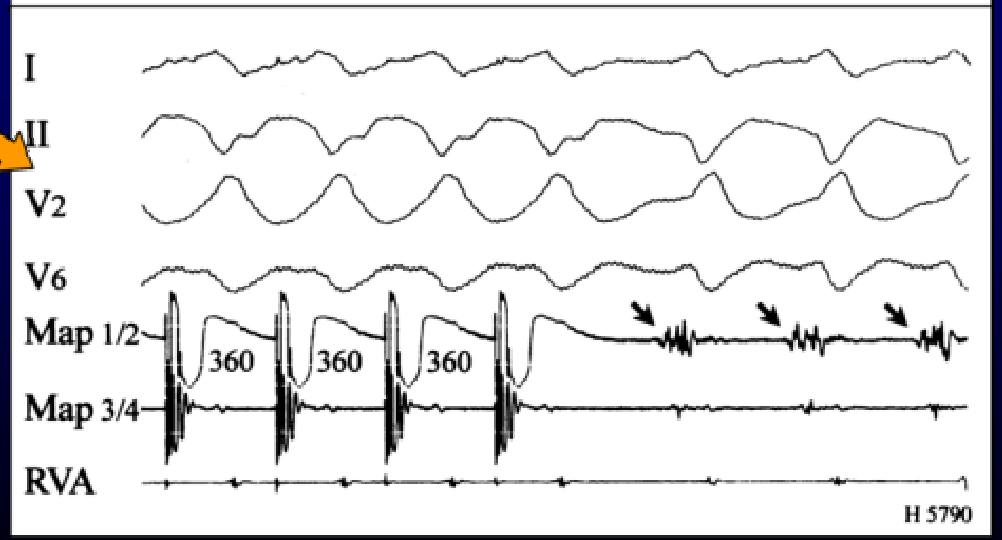


Non BBR-related VT mapping in patients with idiopathic DCM



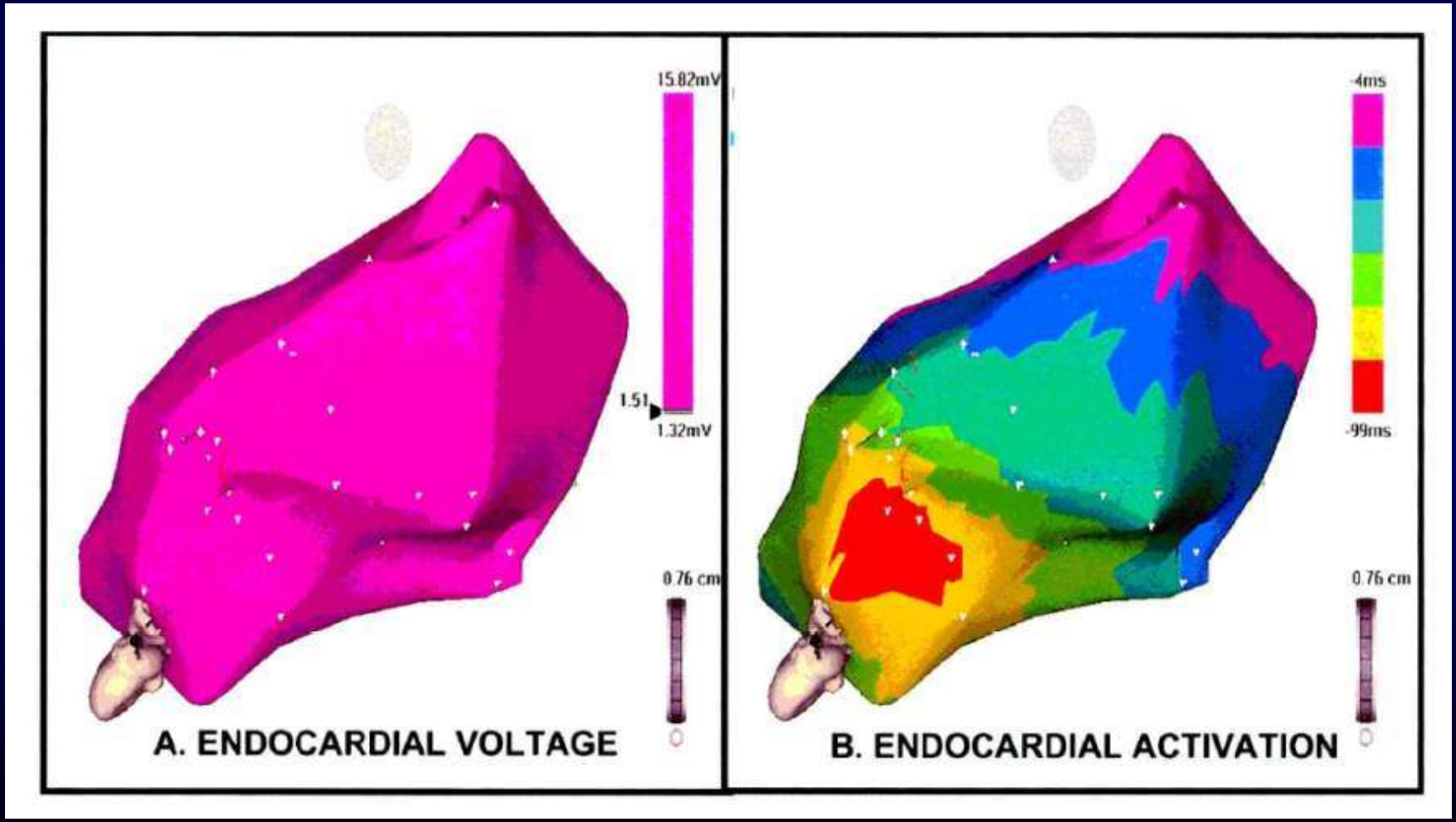
Endocardial reentrant circuit





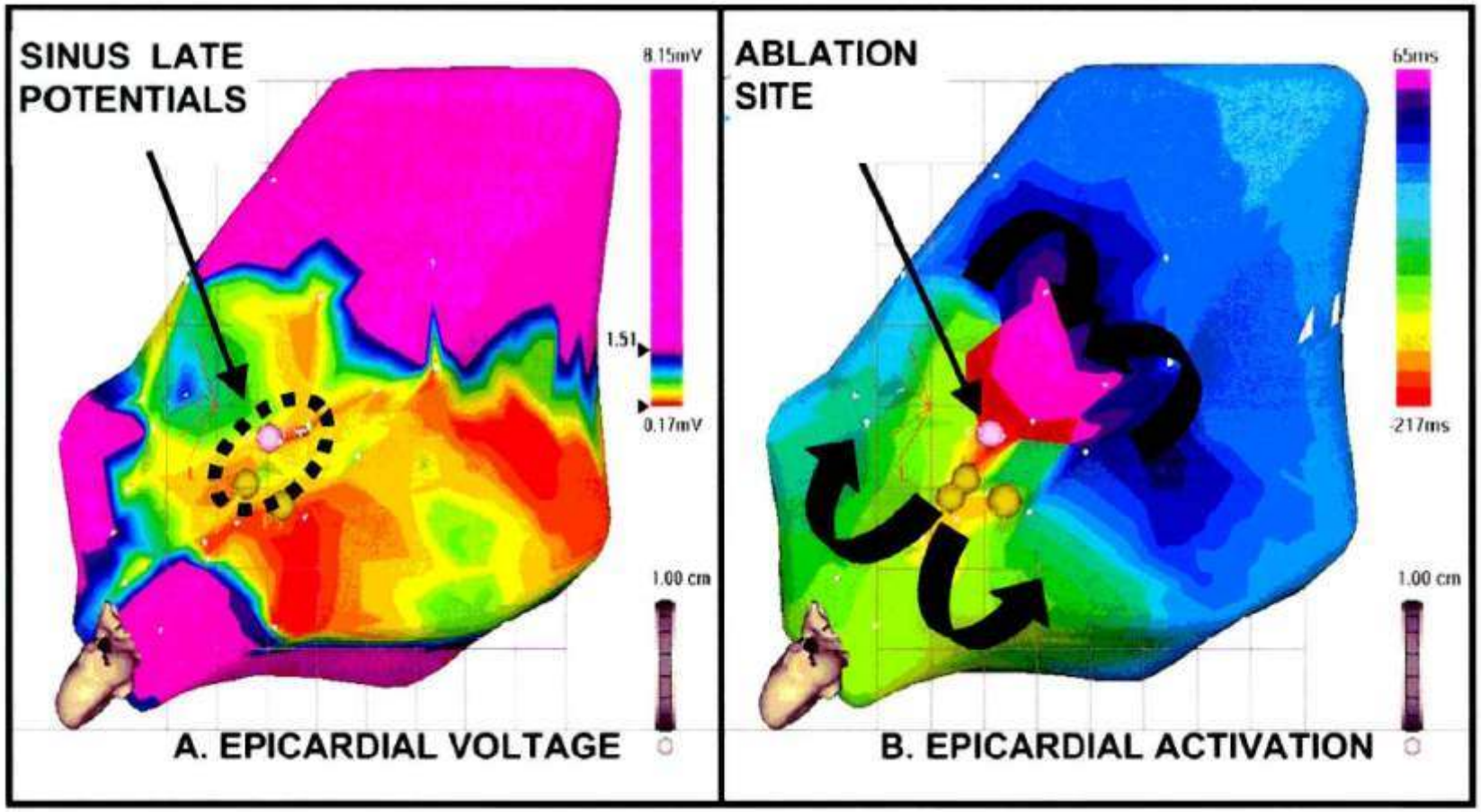
Kottkamp H et al. Circulation 1995;92:1159-68
Delacretaz E et al. J Cardiovasc Electrophysiol 2000;11:11-7
Hsia HH et al. Circulation 2003;108:449-52

Non BBR-related VT mapping in patients with idiopathic DCM



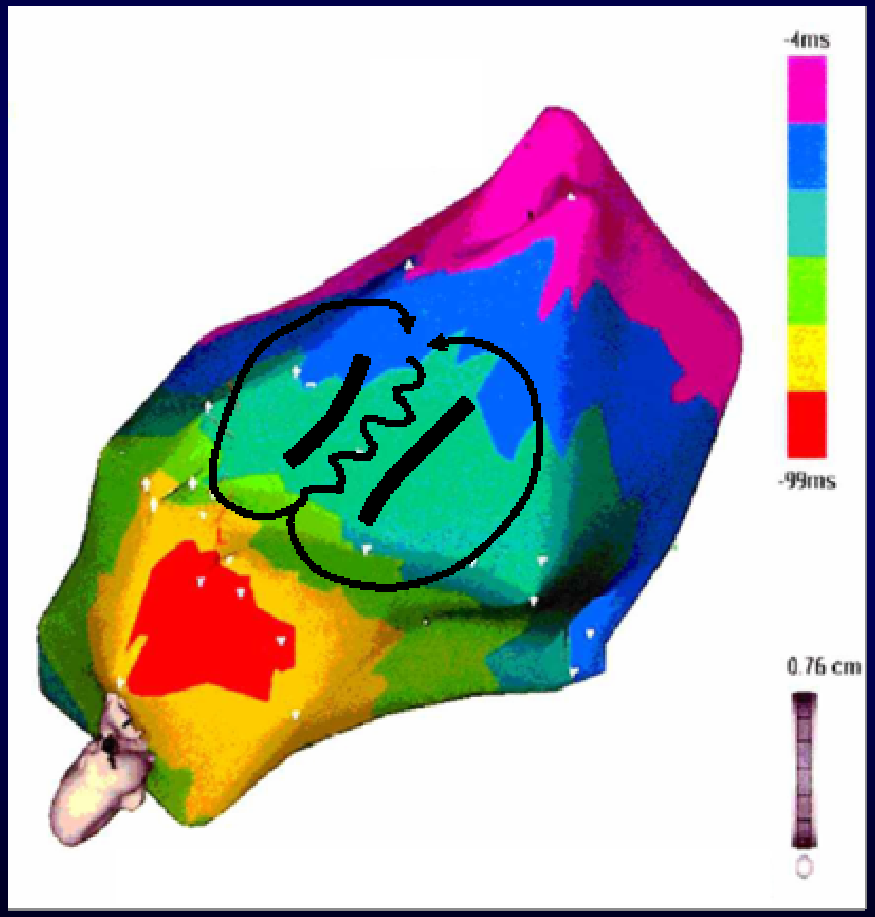
Swarup V et al. J Cardiovasc Electrophysiol 2002;13:1164-8

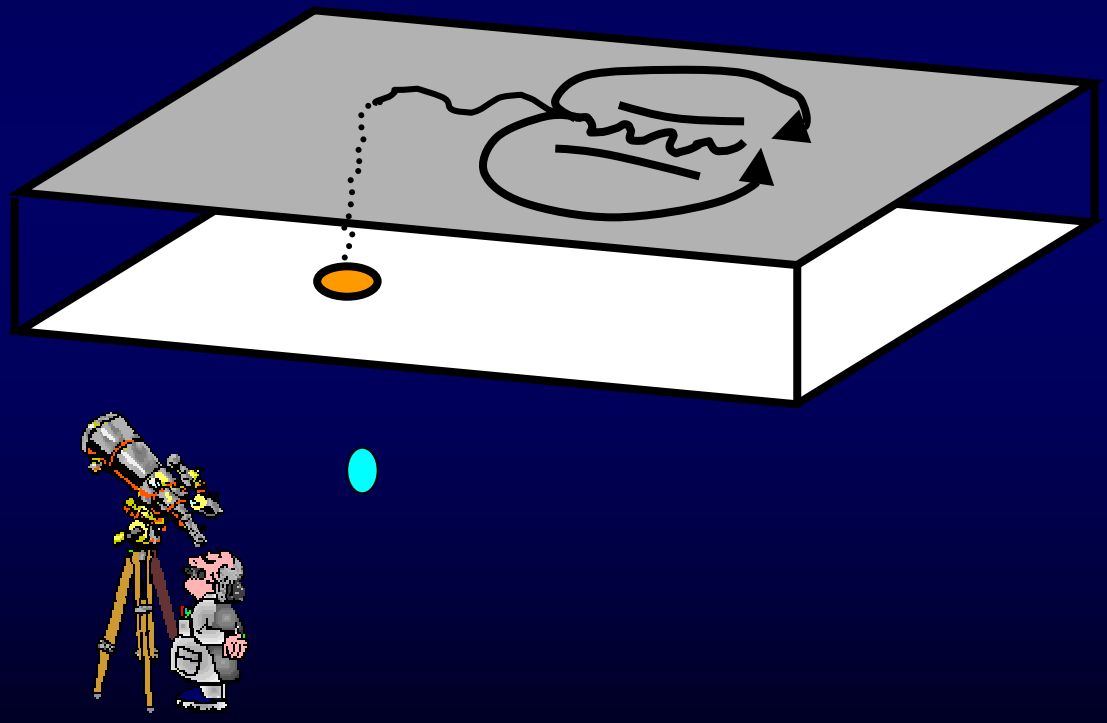
Non BBR-related VT mapping in patients with idiopathic DCM



Swarup V et al. J Cardiovasc Electrophysiol 2002;13:1164-8

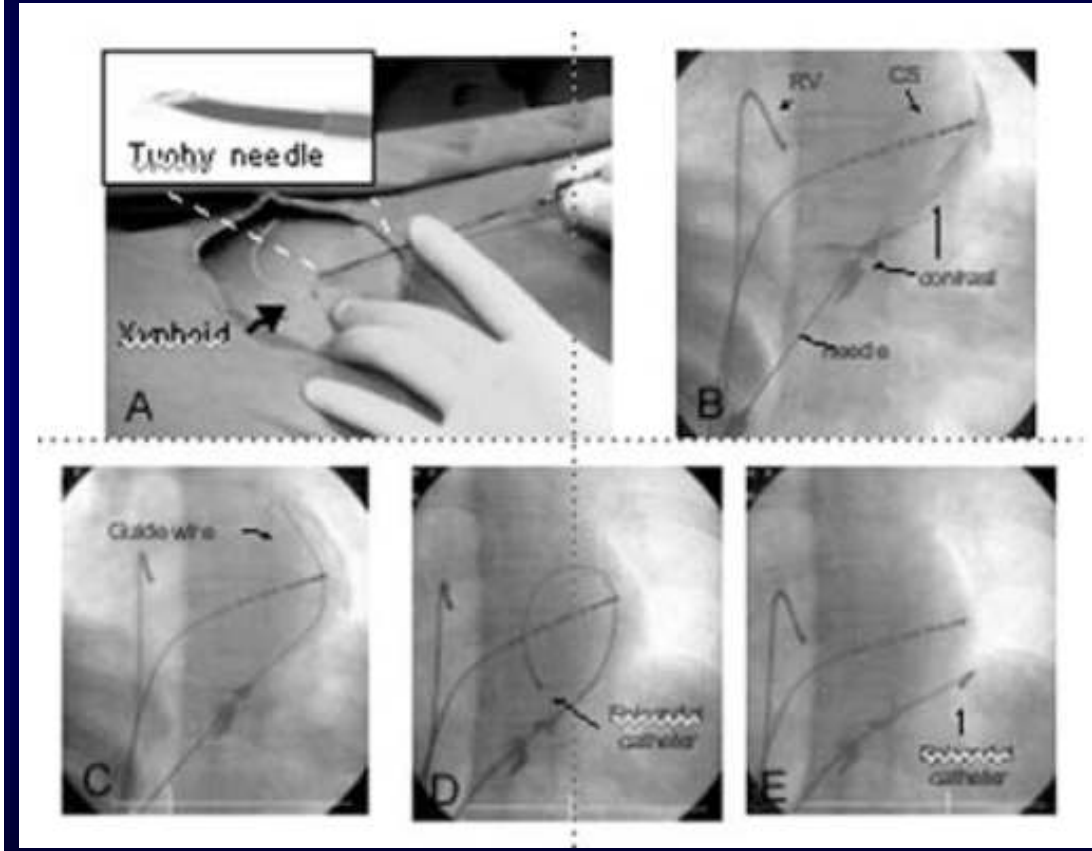
Non BBR-related VT mapping in patients with idiopathic DCM

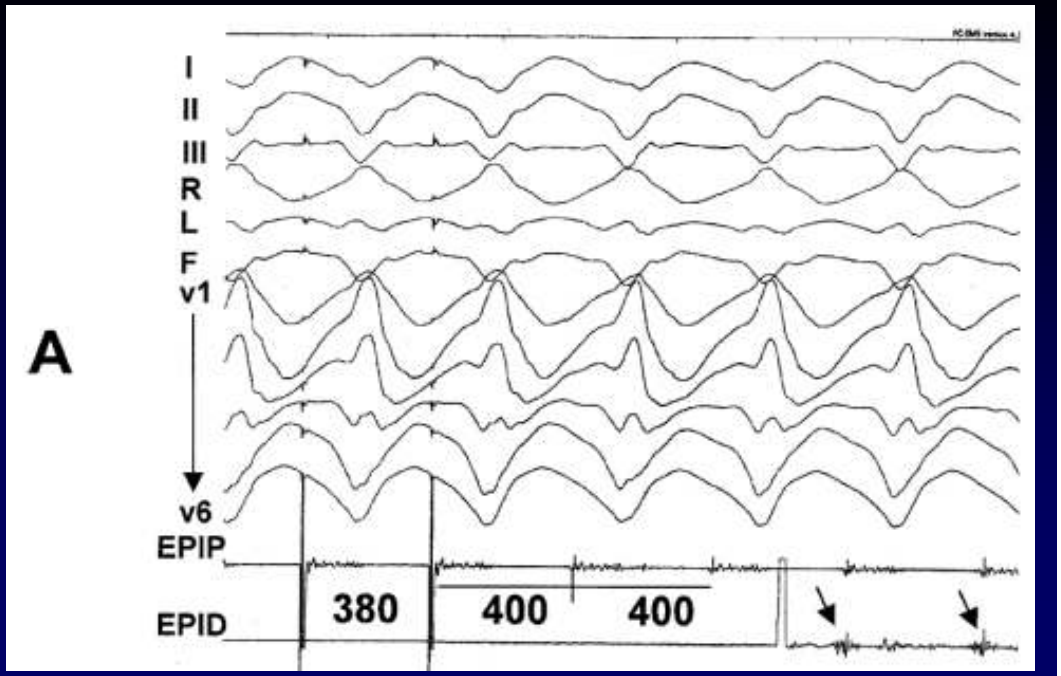


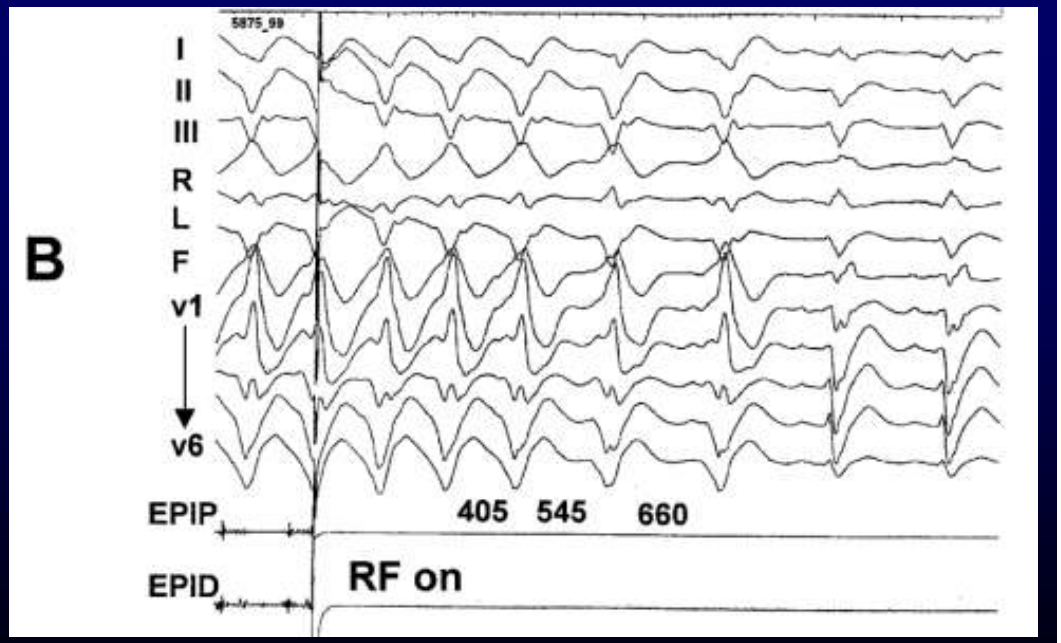


Swarup V et al. J Cardiovasc Electrophysiol 2002;13:1164-8

Epicardial approach for VT ablation







Sosa E et al. J Cardiovasc Electrophysiol 2005;16:449-52

Substrate mapping

VT mapping

Ablation results

VT ablation in patients with a structural heart disease

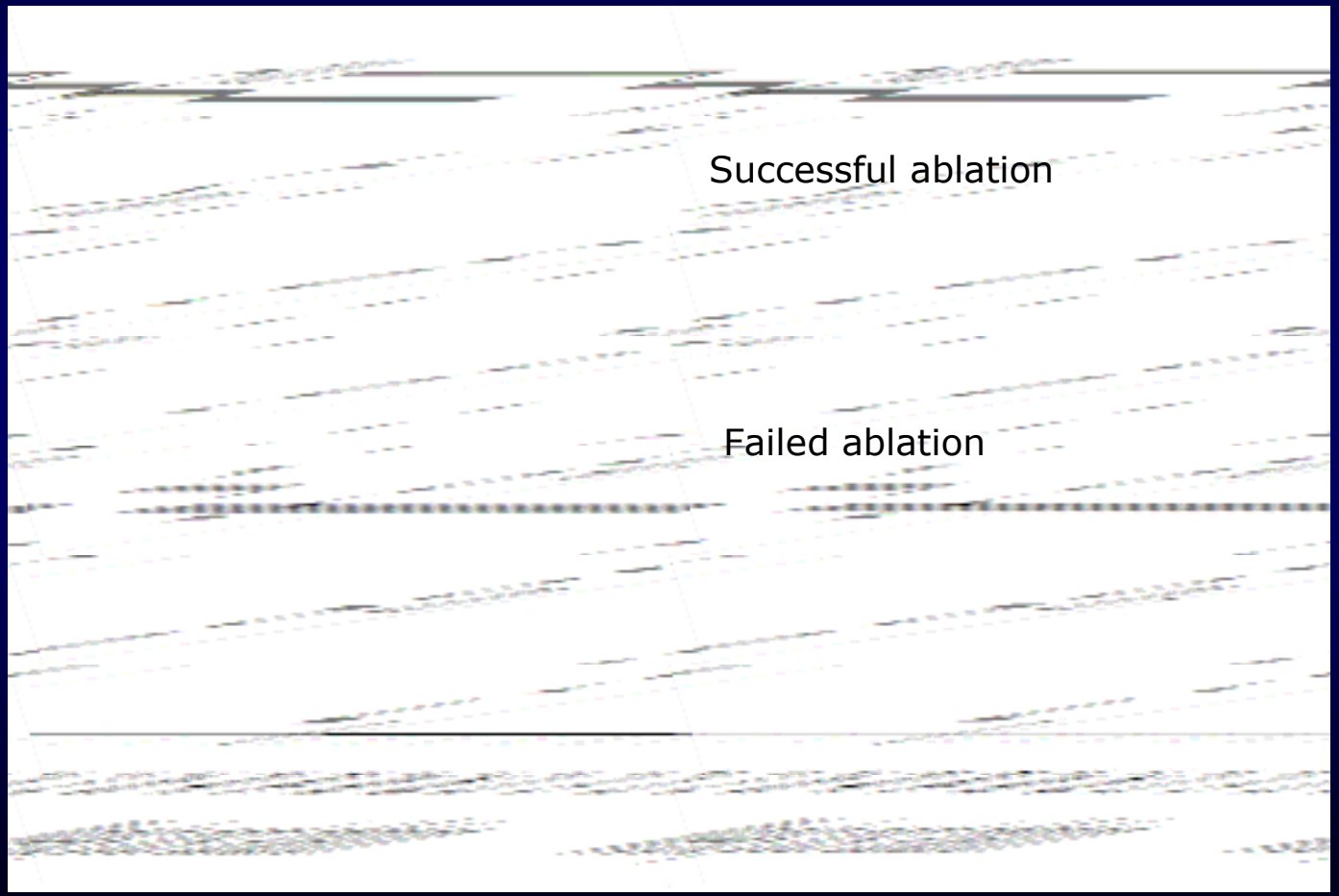
Literature review (case reports excluded)

Results for both ablation of stable and unstable VTs are shown

Heart disease	Studies	Years	Patients	Acute success	Mean FU (mo)	Recurrences*
Post-MI	29	1993 - 2007	1093	76%	19.2	30%
ARVD/C	11	1998 - 2007	211	72%	31.5	33%
Idiopathic DCM	6	1995 - 2006	77	73%	13	41%

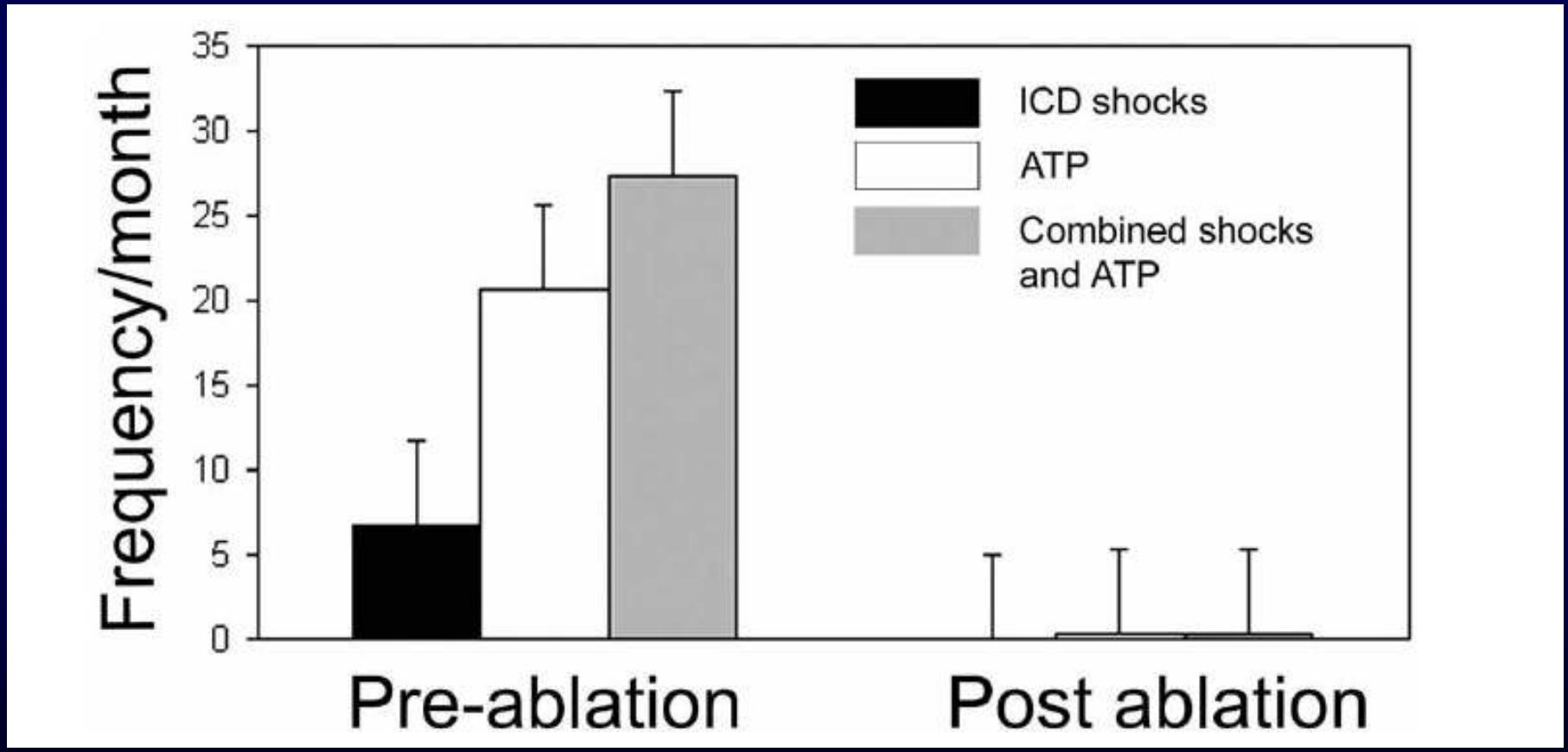
* Global recurrence rate, mixing patients with a successful ablation and those with a failed one

VT ablation : review of the literature
(case reports excluded)



O'Donnell D et al. Eur Heart J 2002;23:1699-1705

VT ablation : review of the literature
(case reports excluded)

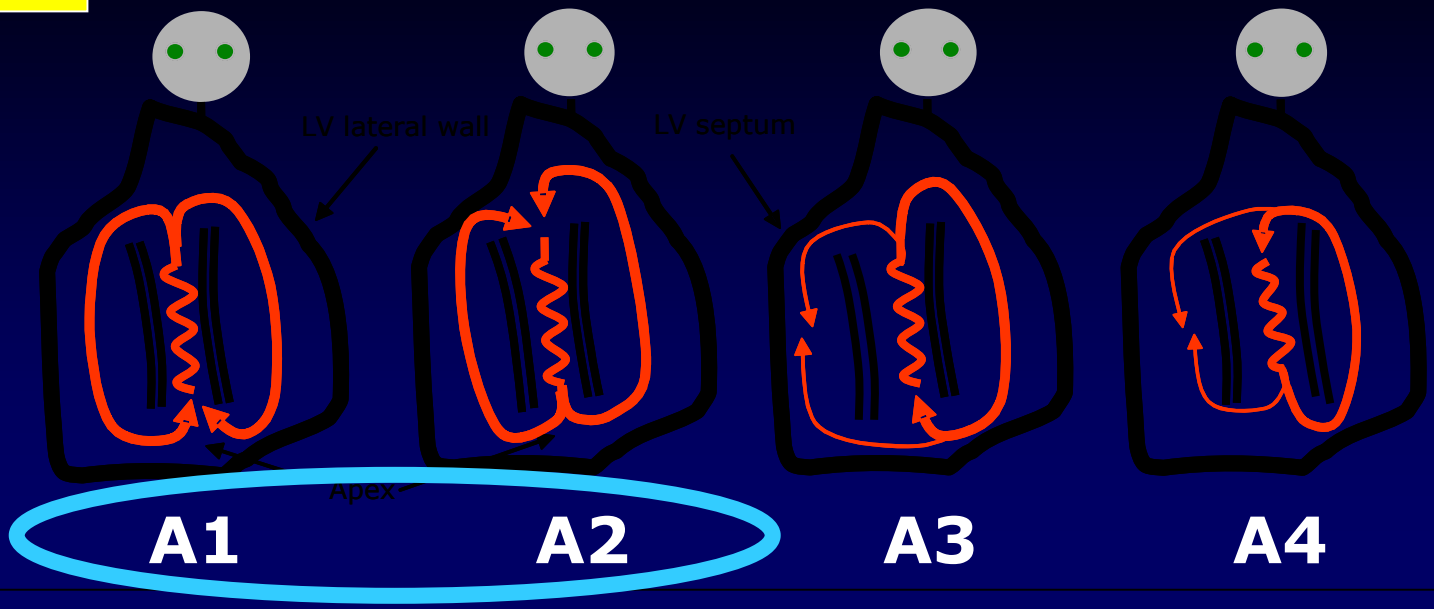


Segal OR et al. Heart Rhythm 2005;2:474-82

1

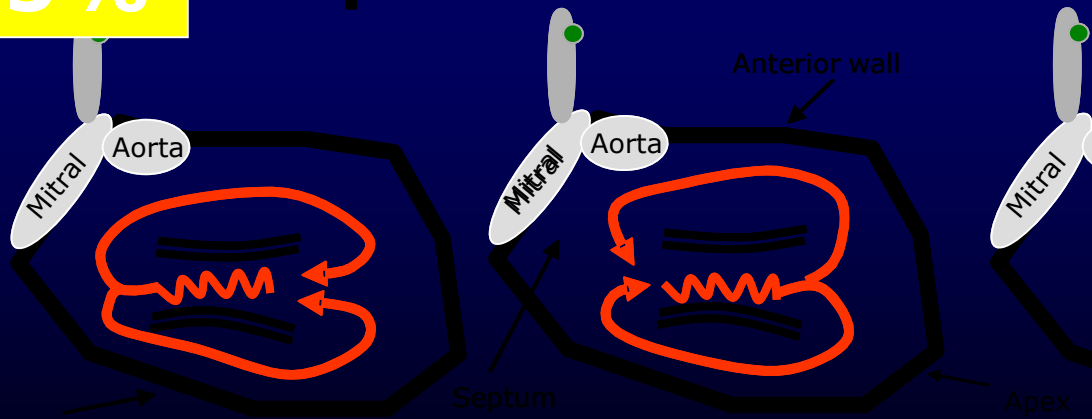
85%

Antero-apical circuits

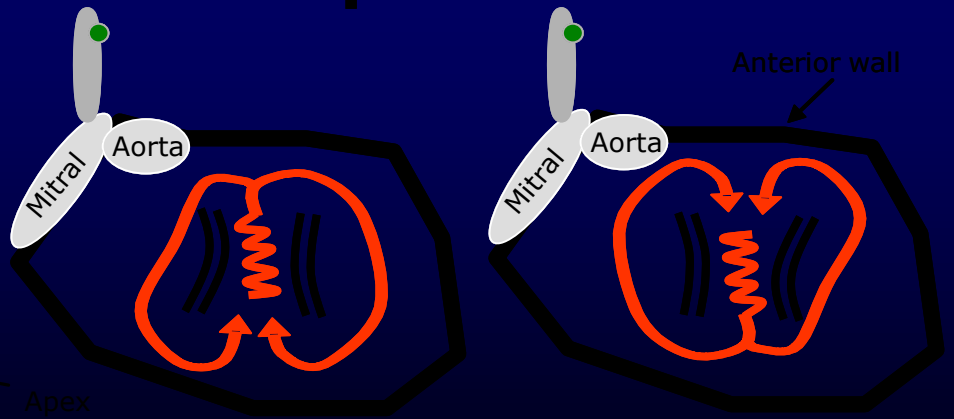


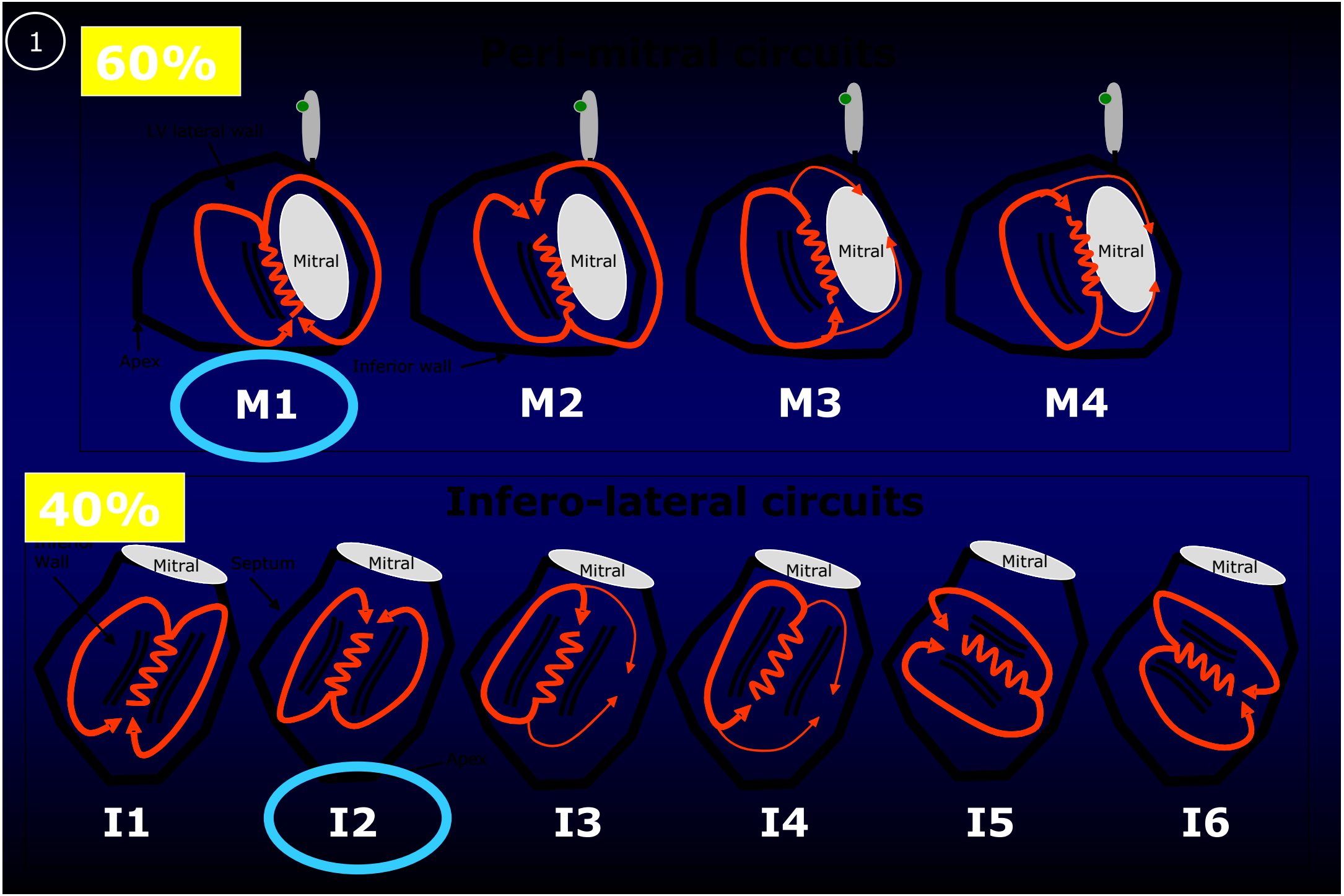
15%

Septal circuits

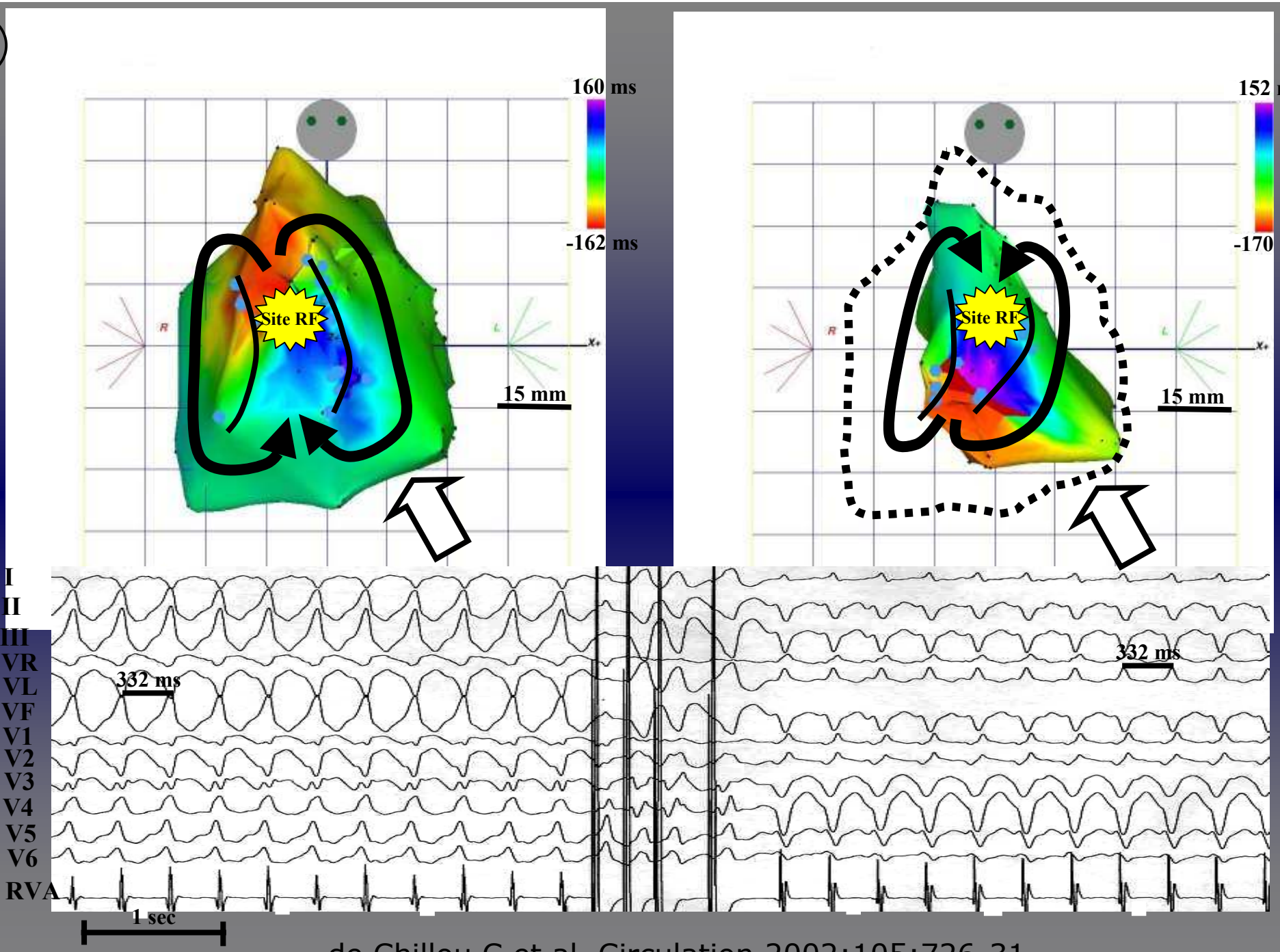


Septal circuits




1
60%
Peri-mitral circuits
LV lateral wall
Mitral
Apex
Inferior wall
M1
M2
M3
M4
40%
Infero-lateral circuits
Wall
Septum
Apex
I1
I2
I3
I4
I5
I6

1



de Chillou C et al. Circulation 2002;105:726-31

VT ablation in patients with ARVD/C

Author	Year	Patients	VTs	Mapping	Acute success	Follow-up (months)	Recurrences
Harada T	1998	7	8	Entrainment	100%	19±7	0%
Ellison KE	1998	5	19	Entrainment	42%	17±5	0%
Fontaine G	2000	50	Unknown	Unknown	46%	65 (mean)	54%
Reithmann C	2003	5	5	CARTO™ & Entrainment	80%	7±3	40%
O'Donnell D	2003	17	31	Activation & Entrainment	41%	56 (mean)	47%
Marchlinski FE	2004	21	66	CARTO™ & Entrainment	74%	27±22	16%
Miljoen H	2005	11	12	CARTO™	75%	36±13	54%
Verma A	2005	22	66	CARTO™	82%	37 (median)	36%
Satomi K	2006	17	26	CARTO™	82%	26±15	24%
Total		**155**	**233**		**69%**	**32**	**30%**

Harada T et al. PACE 1998;21(11 Pt2):2547-50
Ellison KE et al. J Am Coll Cardiol 1998;32:724-8
Fontaine G et al. Curr Cardiol Rep 2000;2:498-506
Reithmann C et al. PACE 2003;26:1308-16
O'Donnell D et al. Eur Heart J 2003;24:801-10
Marchlinski FE et al. Circulation 2004;110:2293-8
Miljoen H et al. Europace 2005;7:516-24
Verma A et al. Circulation 2005;111:3209-16
Satomi K et al. J Cardiovasc Electrophysiol 2006;17:469-76

The VT isthmus and/or successful ablation site were found around the tricuspid annulus or RVOT in 90 (78%) out of the 115 VTs for which this information was available

VT ablation in patients with Idiopathic DCM

Author	Year	Patients	VTs	Mapping	Acute success	Follow-up (months)	Recurrences
Kottkamp H	1995	8	9	Activation & Entrainment	66%	8±5	75%
Marchlinski FE	2000	7	17	**Substrate Mapping**	57%	8±4	43%
Delacretaz E	2000	7	8	Activation & Entrainment	75%	17±10	14%
Hsia HH	2003	19	57	CARTO & Entrainment	74%	22±12	58%
Soejima K	2004	28	82	CARTO & Entrainment	65%	11±9	34%
Cesario DA	2006	8	NA	**Substrate Mapping**	100%	12±4	25%
Total		**77**	**173**		**73%**	**13**	**41%**

Kottkamp H et al. Circulation 1995;92:1159-68
Marchlinski FE et al. Circulation 2000;101:1288-96
Delacretaz E et al. J Cardiovasc Electrophysiol 2000;11:11-7
Hsia HH et al. Circulation 2003;108:704-10
Soejima K et al. J Am Coll Cardiol 2004;43:1834-42
Cesario DA et al. Heart Rhythm 2006;3:1-10

The VT isthmus and/or successful ablation site were found close to the LV base (anterior, lateral or posterior wall) in >90% of cases

Complications of VT Ablation in Patients with Structural Heart Disease

Literature data = mainly post-infarct VT

Acute complications = 7.4% !!

Death = 0.9%
Transient Ischemic Attack or Stroke = 0.9%
Pericardial Effusion or Tamponade = 1.8%