ADVANCES IN CARDIAC ARRHYTHMIAS *and* GREAT INNOVATIONS IN CARDIOLOGY

XXVI Giornate Cardiologiche Torinesi

AZIENDA OSPEDALIERO-UNIVERSITARIA
Città della Salute e della Scienza di Torino

UNIVERSITÀ DEGLI STUDI DI TORINO

From Caliper to Catheter

JMC JOINT MEETING OF CARDIOLOGY

Building the core of a successful VT ablation: the importance of substrate location

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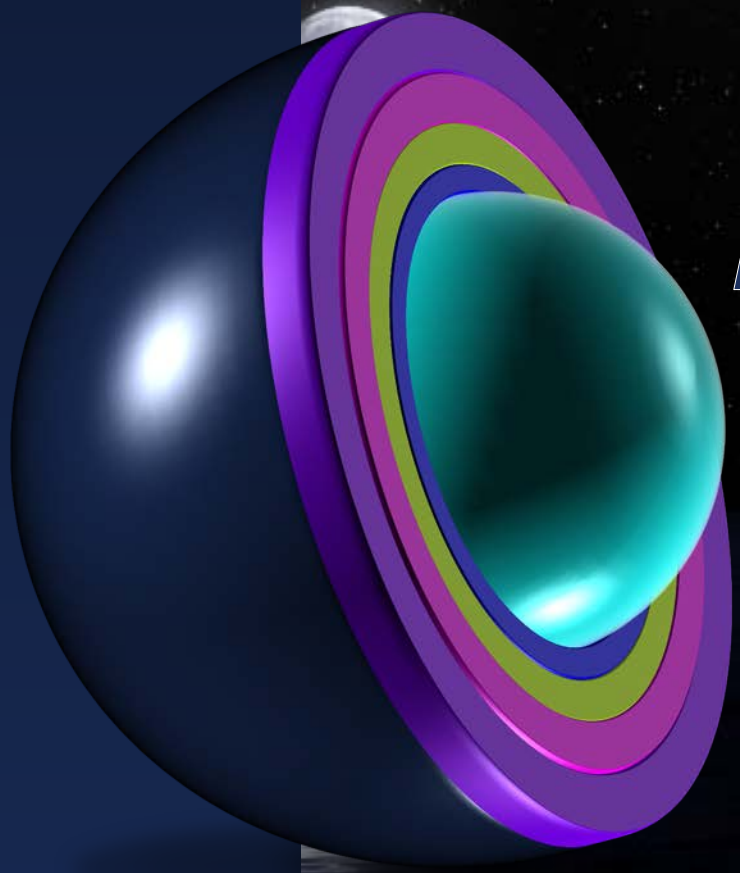
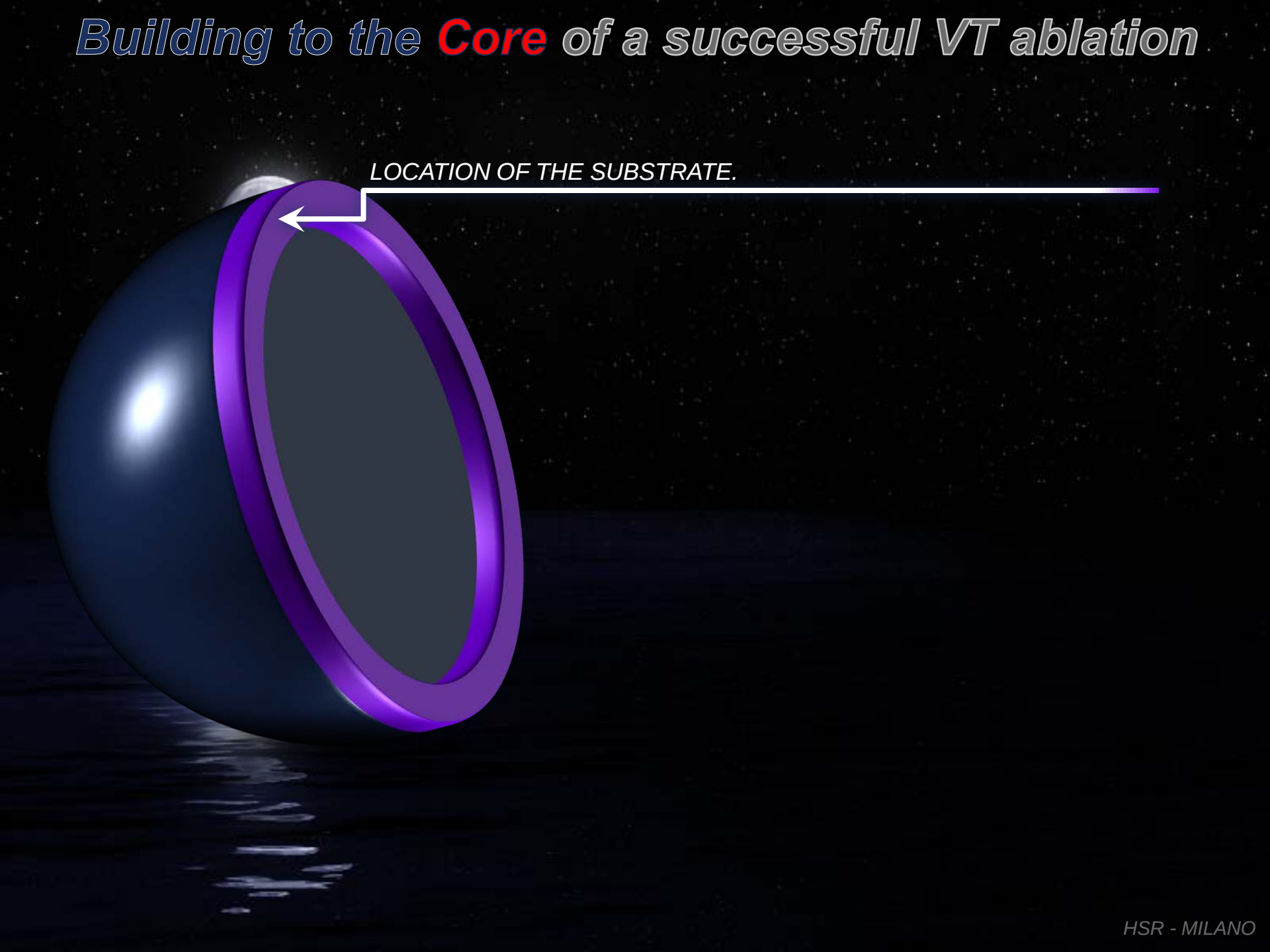
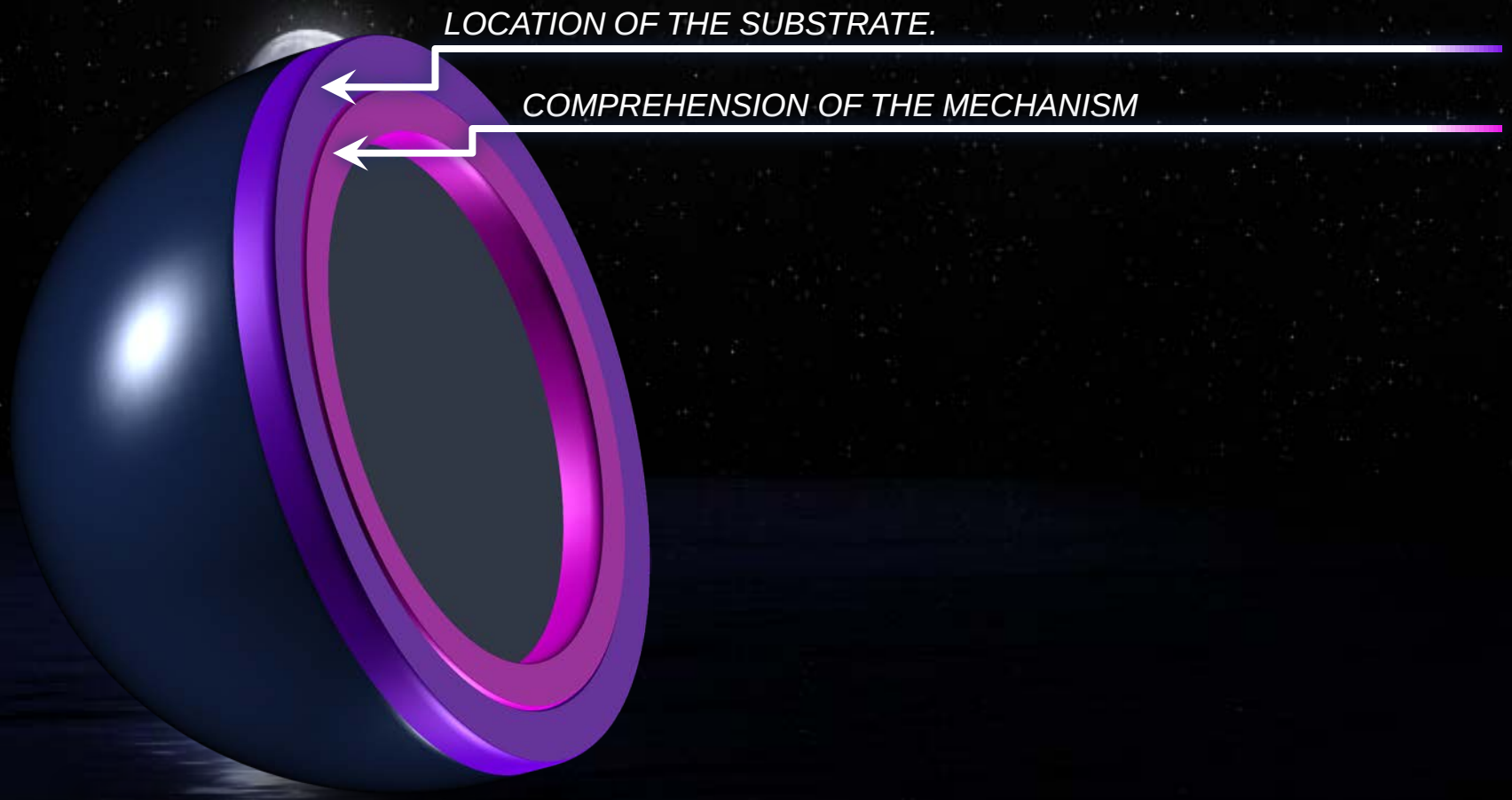


Building to the Core of a successful VT ablation

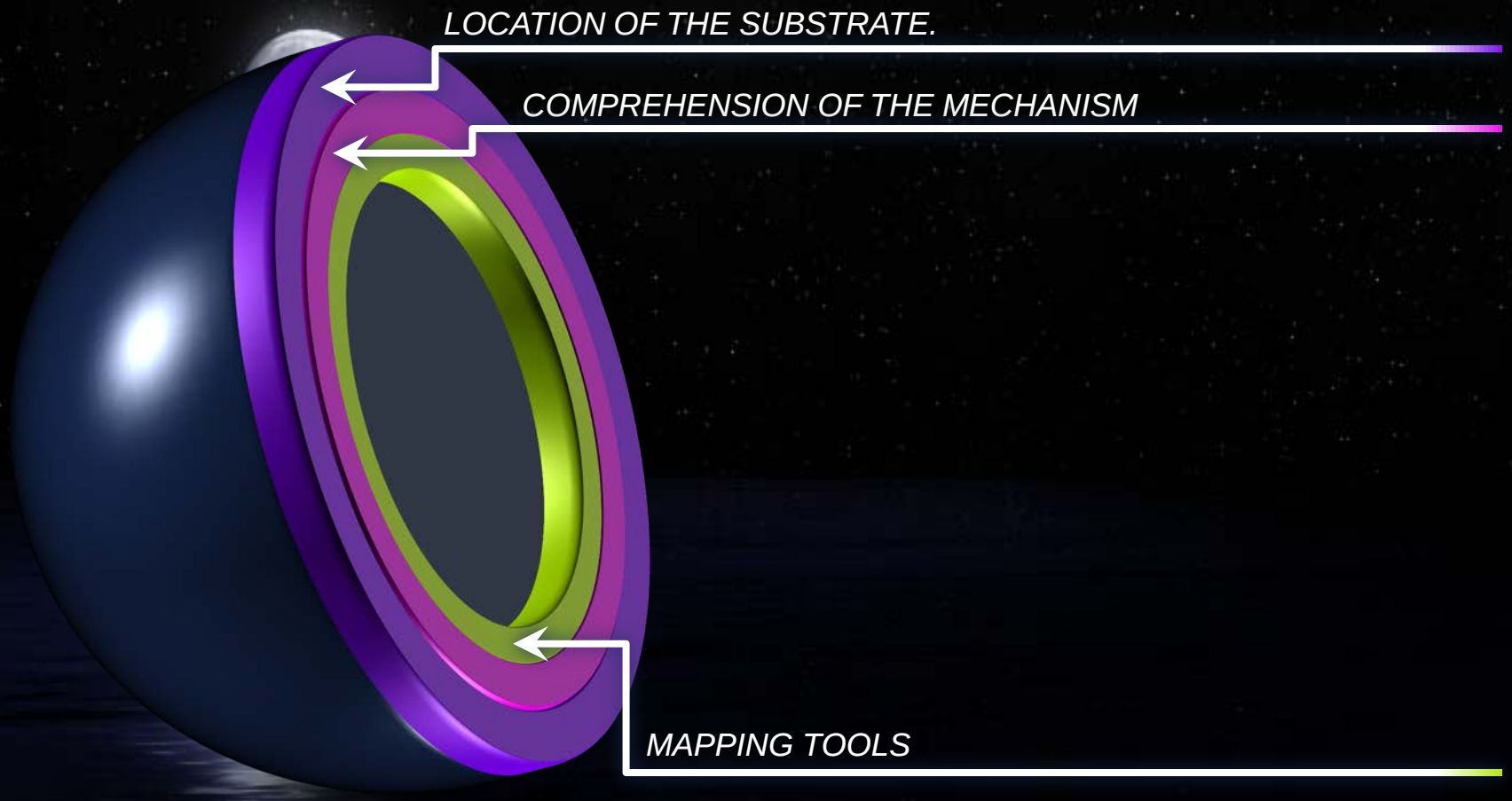
Building to the Core of a successful VT ablation



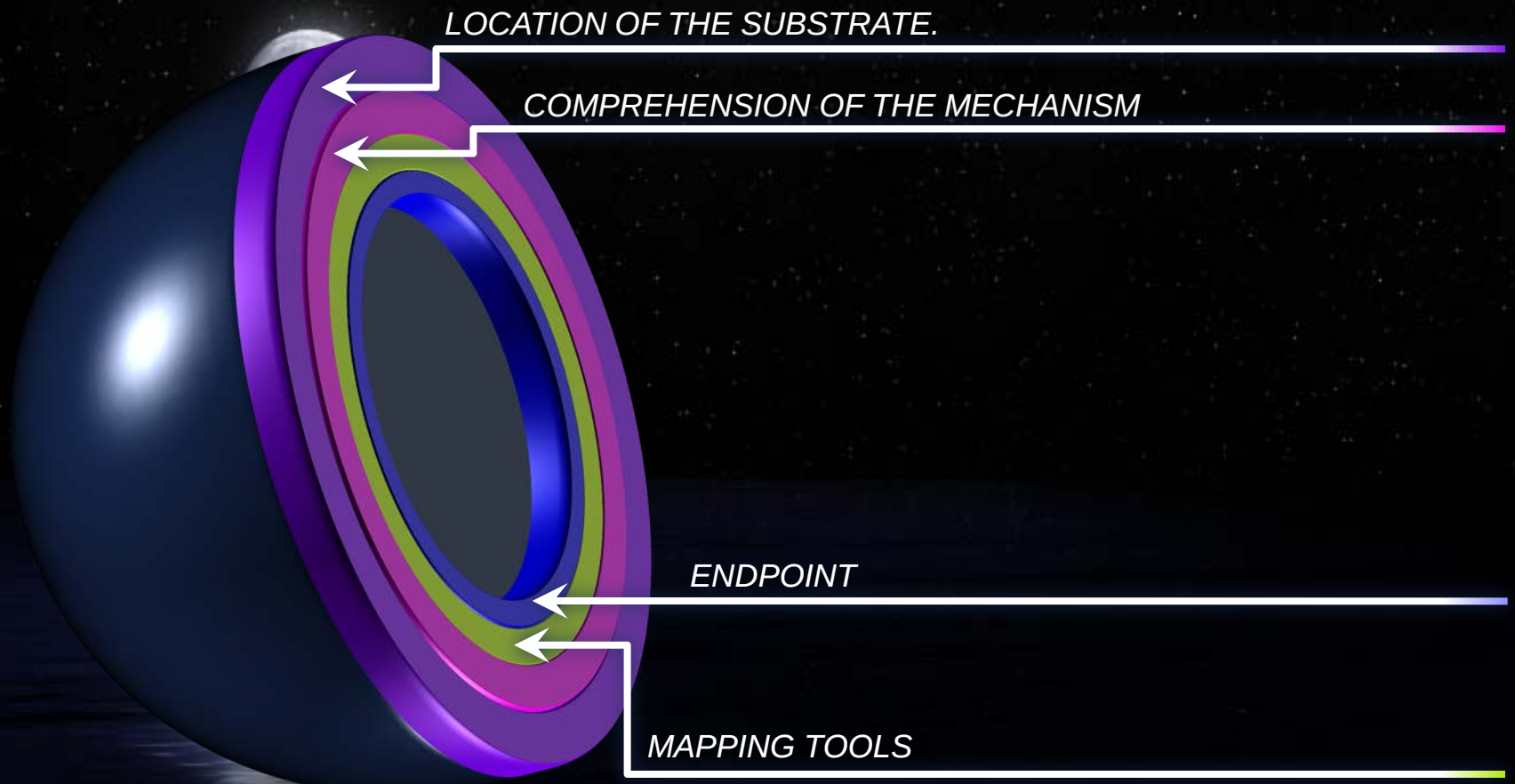
Building to the Core of a successful VT ablation



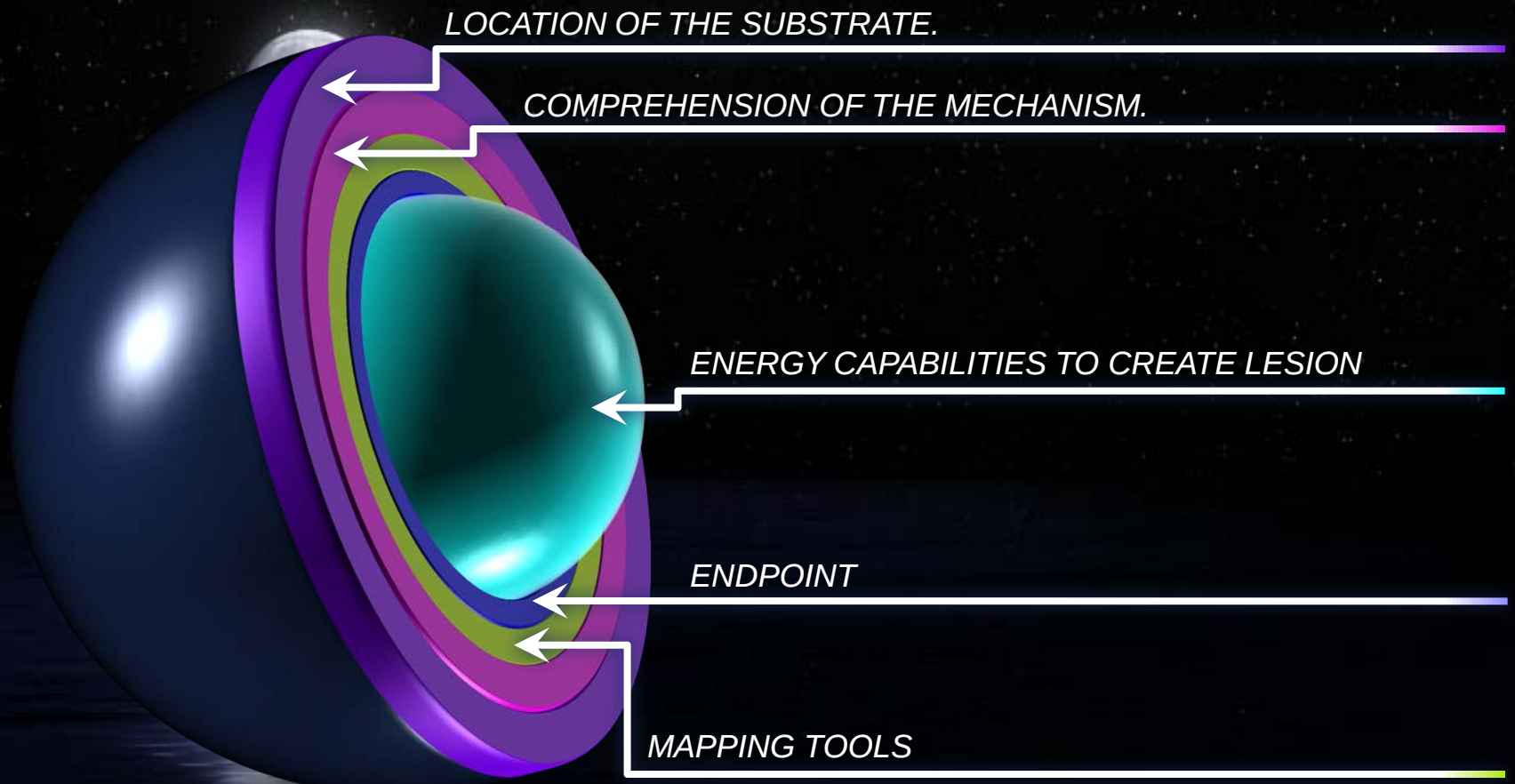
Building to the Core of a successful VT ablation



Building to the Core of a successful VT ablation



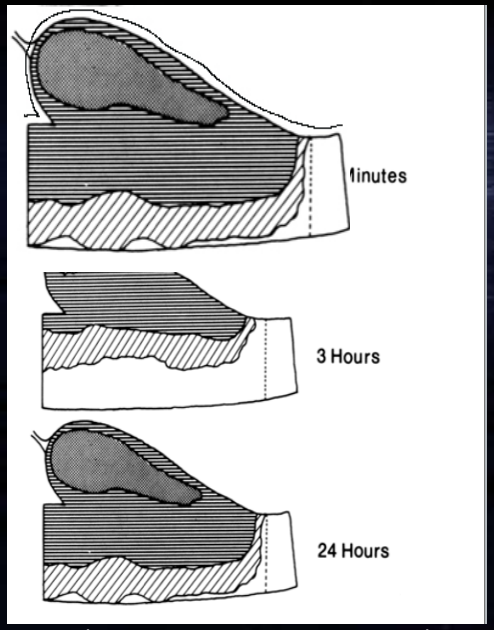
Building to the Core of a successful VT ablation



ISCHEMIC CARDIOMYOPATHY - *prerecanalization era*

The concept of “wavefront phenomenon” of myocardial death

Myocardial necrosis progresses with the duration of coronary occlusion, extending from the subendocardium towards the subepicardium, as to involve the full thickness ventricular wall. Coronary occlusion leads to transmural AMI when exceeding 6 h.

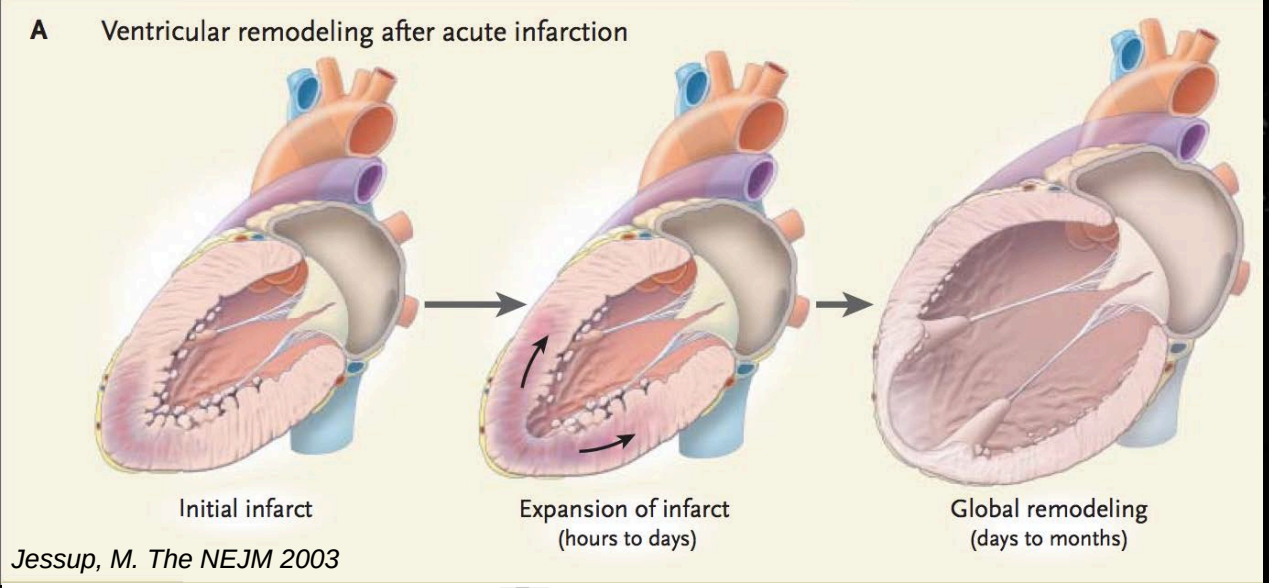


KA Reimer, JE Lowe, MM Rasmussen and RB Jennings
Circulation 1977, 56:786-794

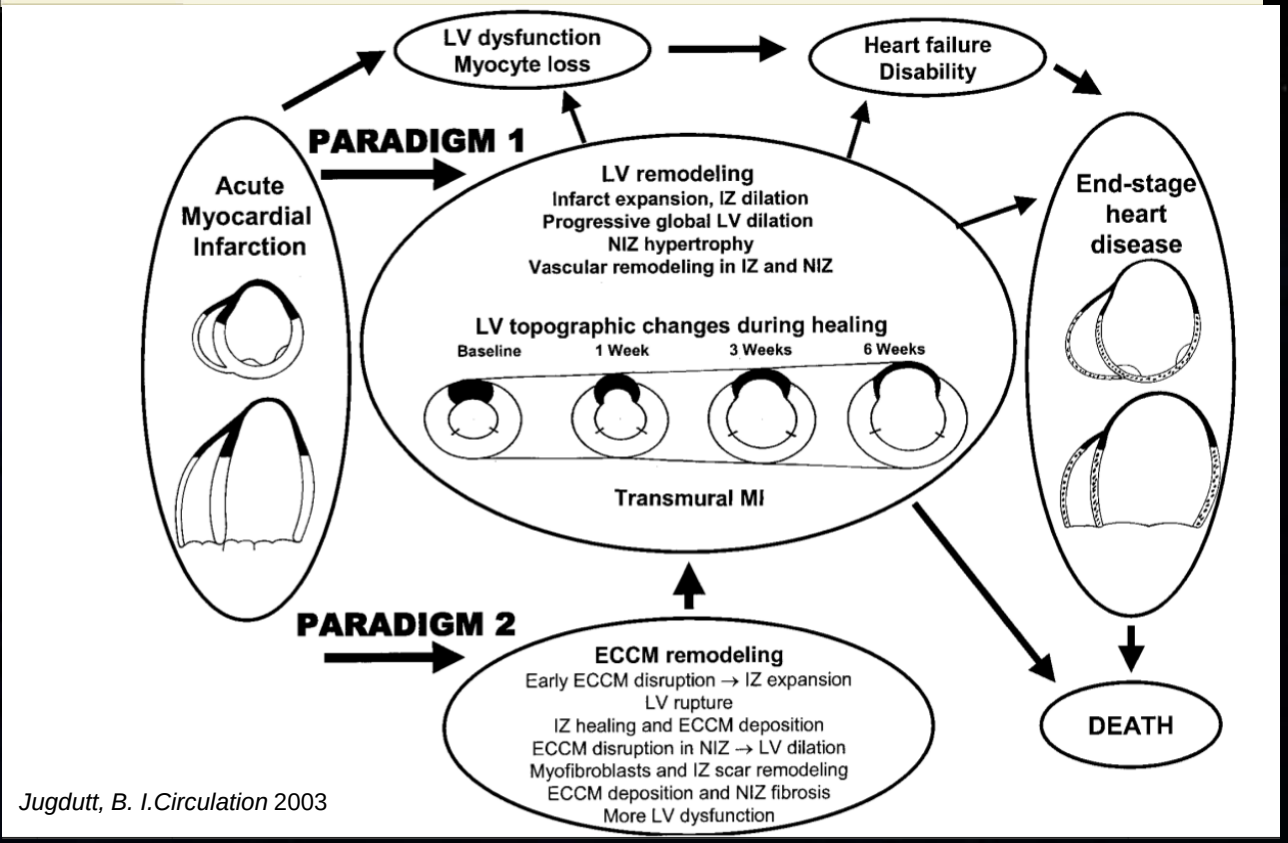
DIFFERENT SCENARIOS

1 WITHOUT RECANALIZATION

2 WITH RECANALIZATION

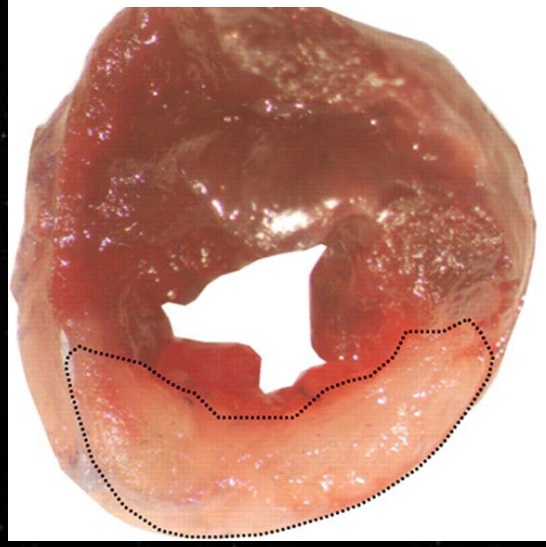


Jessup, M. The NEJM 2003

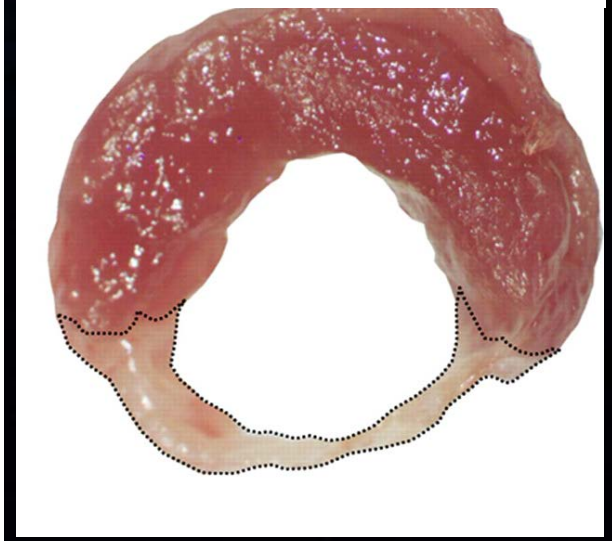


Jugdutt, B. I.Circulation 2003

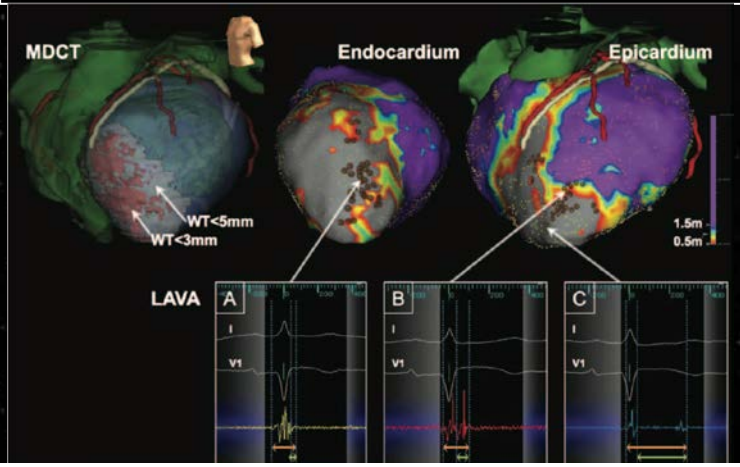
1 DAY

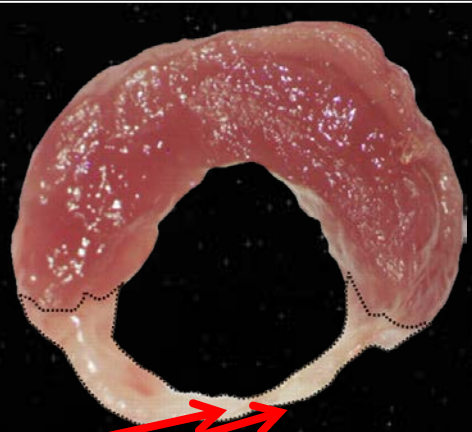


1 MONTH



Komatsu, Y.,. Regional Myocardial Wall Thinning at Multidetector Computed Tomography Correlates to Arrhythmogenic Substrate in Postinfarction Ventricular Tachycardia: Assessment of Structural and Electrical Substrate. Circulation: Arrhythmia and Electrophysiology 2013





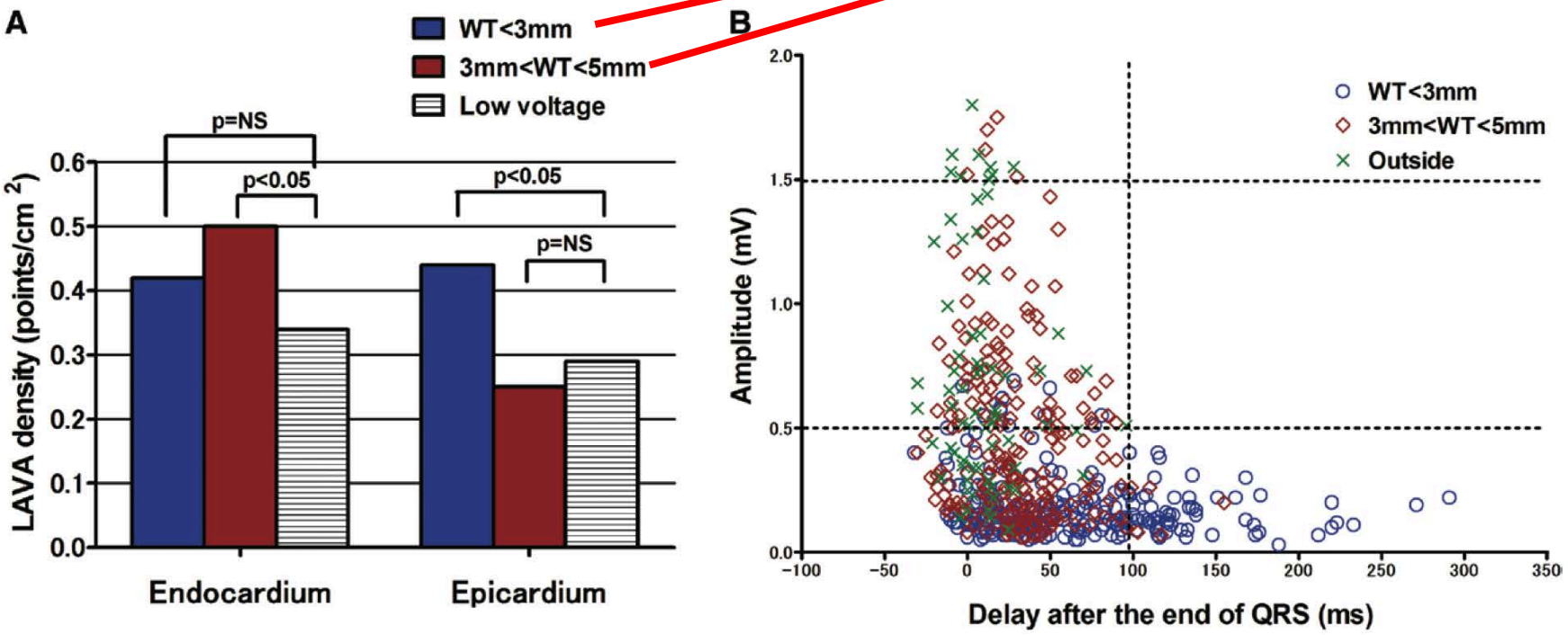


Figure 5. Distribution and characteristics of local abnormal ventricular activities (LAVA). The LAVA density in the low-voltage area was

ISCHEMIC CARDIOMYOPATHY - in the reperfusion era

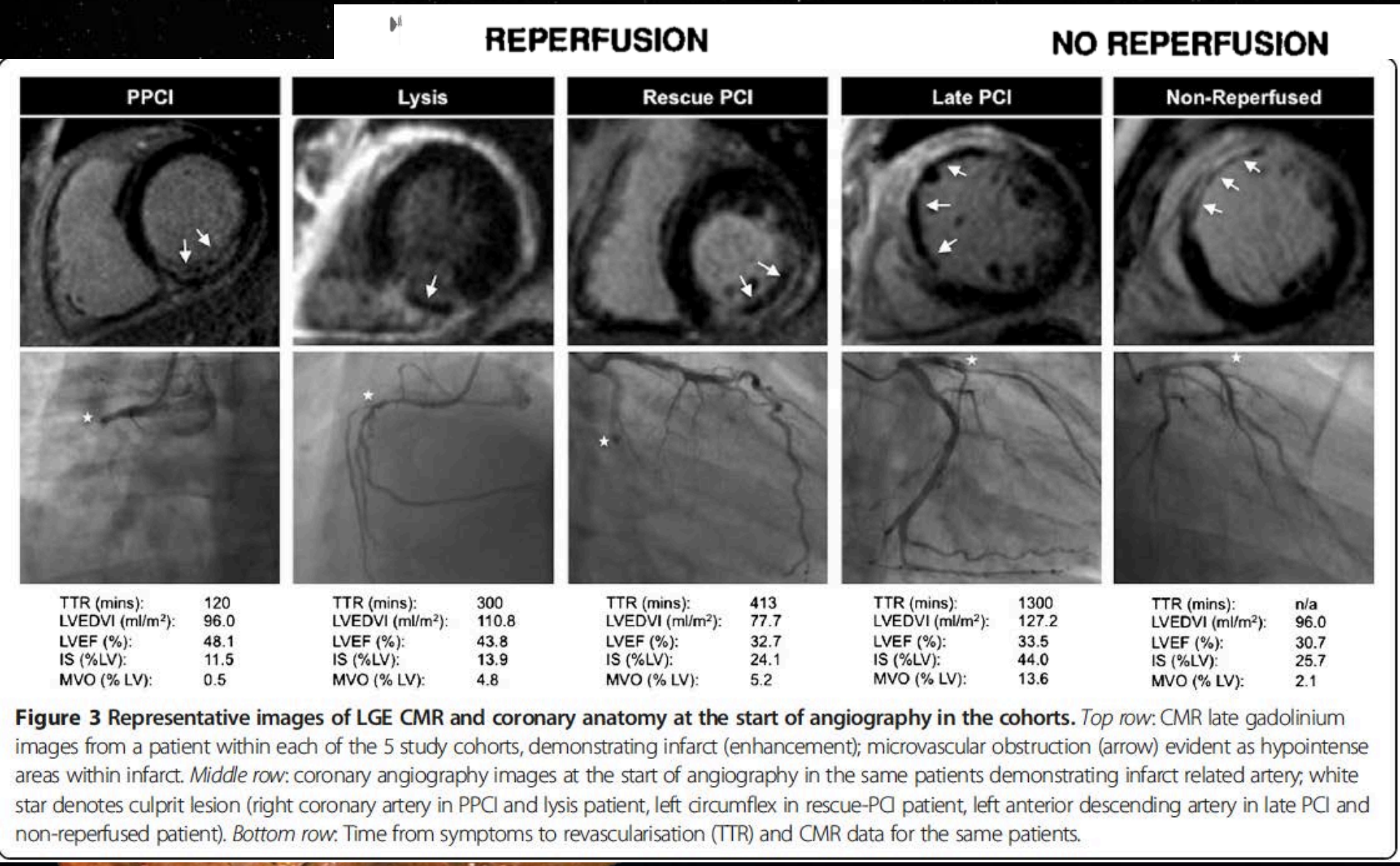


Figure 3 Representative images of LGE CMR and coronary anatomy at the start of angiography in the cohorts. *Top row*: CMR late gadolinium images from a patient within each of the 5 study cohorts, demonstrating infarct (enhancement); microvascular obstruction (arrow) evident as hypointense areas within infarct. *Middle row*: coronary angiography images at the start of angiography in the same patients demonstrating infarct related artery; white star denotes culprit lesion (right coronary artery in PPCI and lysis patient, left circumflex in rescue-PCI patient, left anterior descending artery in late PCI and non-reperfused patient). *Bottom row*: Time from symptoms to revascularisation (TTR) and CMR data for the same patients.

MJ Cowan HUM PATHOL 22:154-163. Copyright C 1991

ISCHEMIC CARDIOMYOPATHY

Sosa E, Nonsurgical trans-thoracic epicardial catheter ablation to treat recurrent ventricular tachycardia occurring late after myocardial infarction. J Am Coll Cardiol 2000;35:1442– 1449.

39% of all mappable VTs late after inferior MI were terminated by epicardial ablation.

Sacher F. Epicardial ventricular tachycardia ablation: a multicenter safety study. J Am CollCardiol 2010;55:2366–2372.

16% of patients with ischemic cardiomyopathy required epicardial mapping and/or ablation.

Nakahara S. Distribution of late potentials within infarct scars assessed by ultra high-density mapping. Heart Rhythm 2010;7: 1817–1824

4 of 14 patients (29%) with PI-MI and 0 of 7 patients (0%) with A-MI required epicardial ablation following endocardial and epicardial high-density mapping

Yoshiga, Y. Correlation between substrate location and ablation strategy in patients with ventricular tachycardia late after myocardial infarction. Heart rhythm : the official journal of the Heart Rhythm Society, *9*(8), 1192–1199. 2012

6 of 40 patients (15%) with PI-MI and none of the patients with A-MI required epicardial access after initial endocardial ablation

Silberbauer, J. Noninducibility and Late Potential Abolition: A Novel Combined Prognostic Procedural End Point for Catheter Ablation of Postinfarction Ventricular Tachycardia. Circulation: Arrhythmia and Electrophysiology, *7*(3), 424–435.

a combined endo-epicardial approach was used in 32 pts (21%) procedures.

ENDOCARDIAL ABLATION – THE GOLDEN ROLE IN ICM

Gao, P., Yee. Prediction of Arrhythmic Events in Ischemic and Dilated Cardiomyopathy Patients Referred for Implantable Cardiac Defibrillator: Evaluation of Multiple Scar Quantification Measures for Late Gadolinium Enhancement Magnetic Resonance Imaging. Circulation Cardiovascular Imaging, 2012

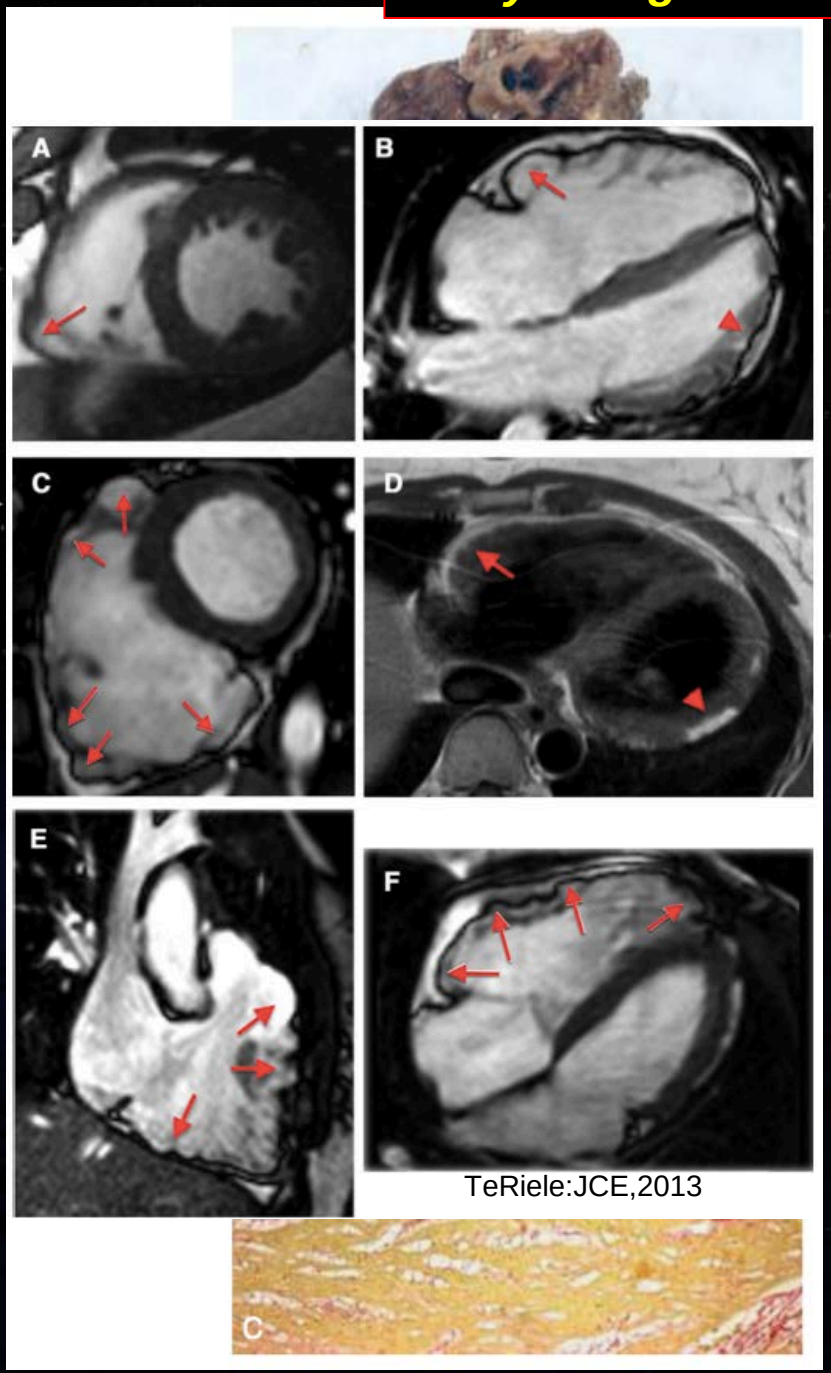
Table 2. Baseline Magnetic Resonance Imaging Characteristics of Study Population in Those With and Without the Primary Outcome Stratified According to Cardiomyopathy Etiology

	Total Population (N=124)			ICM (N=59)			DCM (N=65)		
Variable	PO– (N=106)	PO+ (N=18)	*P* Value	PO– (N=49)	PO+ (N=10)	*P* Value	PO– (N=57)	PO+ (N=8)	*P* Value
Non-HE variables									
LVEF (%)	26±7	25±7	0.471	27±8	23±8	0.072	26±7	28±7	0.350
LV EDV (mL)	251±68	261±73	0.557	244±65	283±66	0.087	257±71	234±76	0.394
LV ESV (mL)	186±61	195±56	0.554	180±61	216±32	0.075	192±62	170±69	0.356
Sub-epicardial HE	14 (13%)	4 (22%)	0.319	1 (2%)	0 (0%)	0.655	13 (23%)	4 (50%)	0.104
HE, total HE (g)									
STRM									
≥2SD	32±19	59±30	0.001	42±19	69±17	0.001	23±15	46±38	0.003
≥3SD	22±18	48±31	0.001	33±18	59±19	0.001	13±12	34±38	0.001
≥5SD	13±15	35±29	0.001	23±15	44±22	0.001	5±7	23±34	0.001
FWHM									
>50%				25±14	37±10	0.01			
HE, peri-infarct (g)									
STRM									
2–3 SD				9±5	11±6	0.270			
2–5 SD				19±10	25±11	0.107			
FWHM									
35%–50%				17±11	25±16	0.075			

COMPULSORY EPICARDIAL ABLATION

ENDOCARDIAL THROMBUS OR CALCIFICATION

Arrhythmogenic Right Ventricular Dysplasia



TeRiele:JCE,2013

The most striking morphological feature of the disease is the diffuse or segmental loss of RV myocytes, with replacement by fibrofatty tissue and thinning of the RV wall.

Patchy inflammatory infiltrates can be present in areas of myocardial damage.

Fibrofatty replacement usually begins in the subepicardium or midmural layers and progresses to the subendocardium.
Only the endocardium and myocardium of the trabeculae may be spared.

The sites of involvement can be localized and in early disease may show a characteristic pattern involving the basal inferior and anterior RV, (and the posterolateral LV). The RV apex is only involved in advanced ARVD/C, typically as a part of global RV involvement .
RV aneurysms, and segmental RV hypokinesia are typical. Diffuse myocardial involvement leads to global RV dilation.
However, the fibrofatty pattern of ARVD is limited not only to the RV; the disease also can migrate to the LV,with a predilection for the posteroseptal and posterolateral areas, with relative sparing of the septum

Arrhythmogenic Right Ventricular Dysplasia

Epicardial Substrate and Outcome With Epicardial Ablation of Ventricular Tachycardia in Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia
GARCIA . CIRCULATION 2009

Table 2. Substrate Characterization

	Sinus Rhythm RV Points		Abnormal Bipolar RV Voltage Area, cm^2		Endocardial-Epicardial Distance, mm		
Patient	Endocardial	Epicardial	Endocardial	Epicardial	Basal RV	Mid Free Wall RV	Opposing RF Lesions
1	154	332	43.4	72.1	7	7	7
2	267	260	53.8	141.7	15	8	9
3	183	195	40.2	78.4	7	8	7
4	297	260	31.3	82.8	7	8	7
5	318	316	136.3	187.7	11	8	6
6	326	431	34.4	178.6	12	7	11
7	458	415	34.8	51.6	23	7	12
8	355	443	17.0	90.8	15	7	13
9	590	527	43.5	67.5	20	8	16
10	386	695	12.2	106	6	7	...
11	185	311	13.2	28.3	9	5	4
12	552	356	26.4	73.7	7	5	...
13	319	605	6.1	77.8	5	5	6
Mean±SD	337±134	396±145	37.8±32.8	95.2±47.1	11.1±5.6	6.9±1.2	8.9±3.6

Opposing RF lesions indicates sites ablated at endocardial/epicardial sites directly opposite from each other.

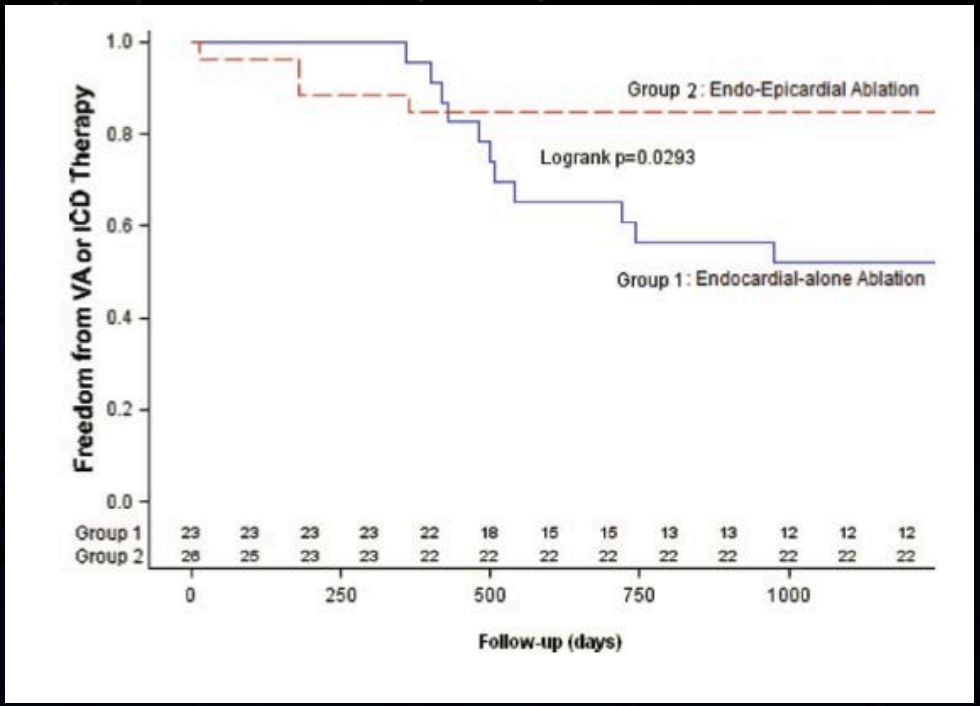
Epicardial VTs were targeted opposite normal endocardium in 10 patients (77%) and/or opposite ineffective endocardial ablation sites in 11 patients (85%). During 18±13 months, 77% of patients had no VT, with 2 patients having only a single VT at 2 and 38 months, respectively.

Arrhythmogenic Right Ventricular Dysplasia

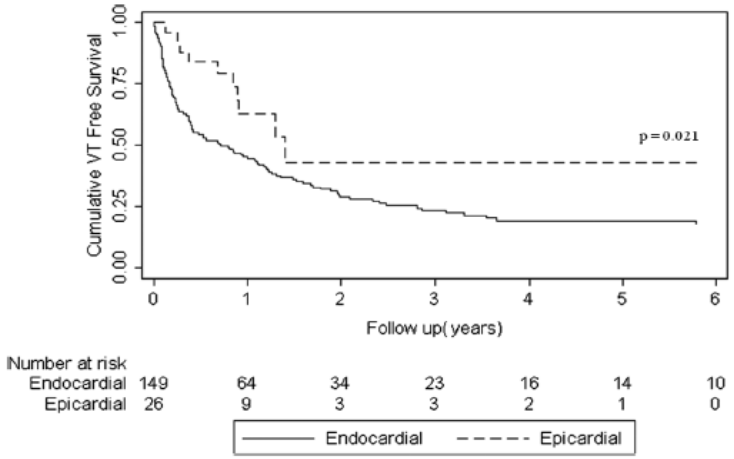
Ablation of Ventricular Arrhythmias in Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy Arrhythmia-Free Survival After Endo-Epicardial Substrate Based Mapping and Ablation
Bai. ***Circ Arrhythm Electrophysiol. 2011;4:478-485.***

Forty-nine patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy

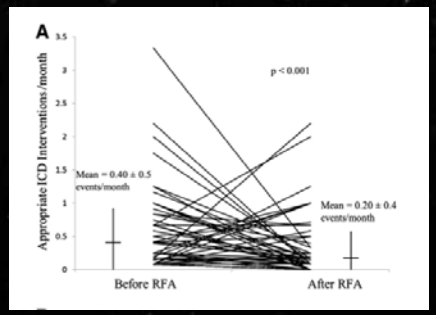
Follow-up: 3 years	Group 1	Group 2	
Freedom from VAs or ICD therapy	52.2% (12/23)	84.6% (22/26)	P=0.029
Off antiarrhythmic drugs	with 21.7% (5/23	69.2% (18/26)	P=0.001



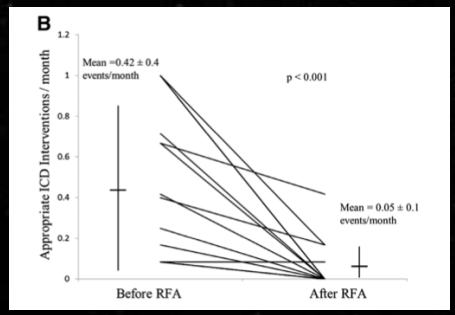
Philips, B. Outcomes of Catheter Ablation of Ventricular Tachycardia in Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy. Circulation: Arrhythmia and Electrophysiology, 2012



N° ICD SHOCKS

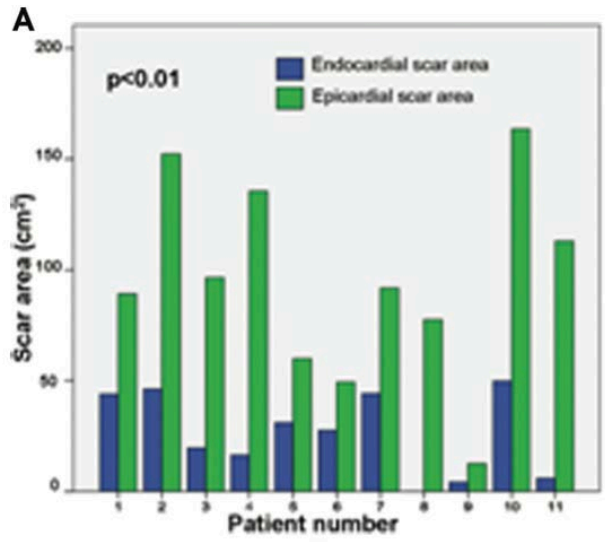


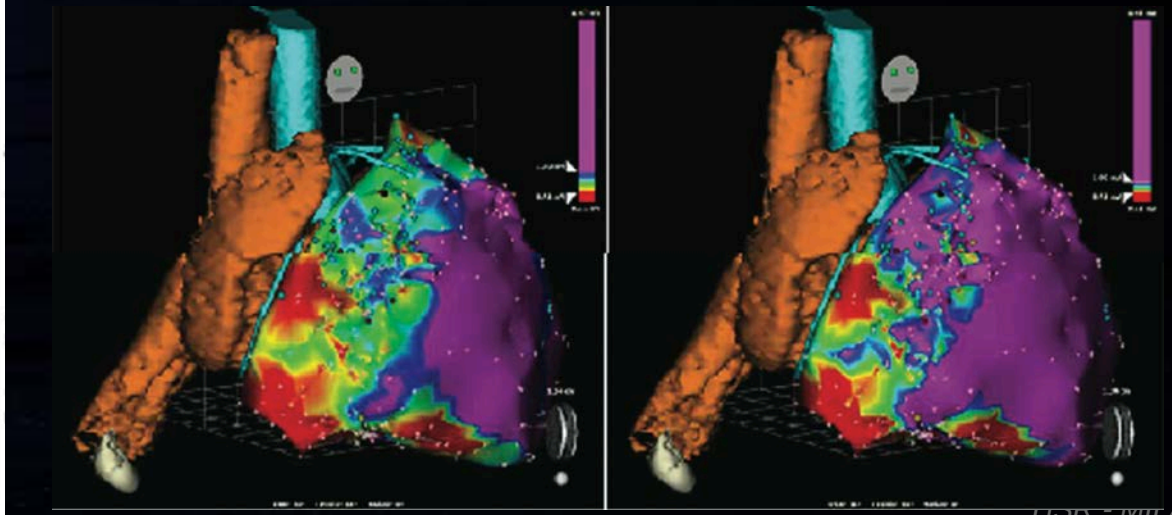
Endocardial



Endo-epicardial

Berruezo, A. Combined Endocardial and Epicardial Catheter Ablation in Arrhythmogenic Right Ventricular Dysplasia Incorporating Scar Dechanneling Technique. Circulation: Arrhythmia and Electrophysiology, 2012





Arrhythmogenic Right Ventricular Dysplasia

V.V. 44 y, female, initial ARVD dilated cardiomyopathy with mild LV dysfunction

9/2011 marked weakness during aerobic exercise -> syncope -> the ECG shows VT (strip not available) -> treated with DC shock

Coronary angiography: normal

EPS: inducibility of VT -> Endocardial ablation (RV) -> partial success

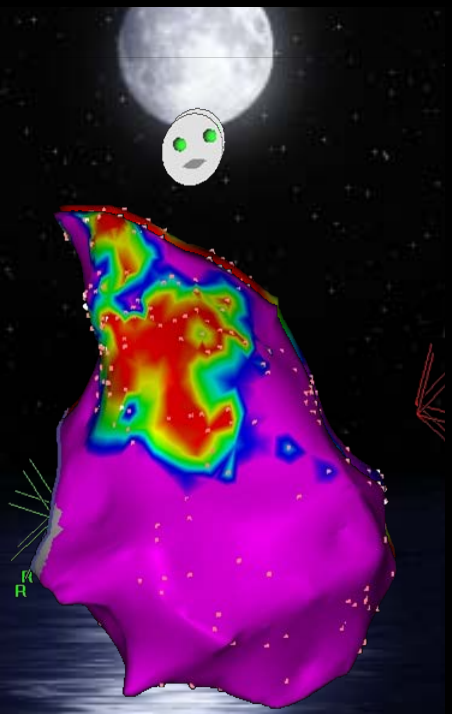
Discharged: Metoprolol

10/2011 2 spontaneous VT episode

REFERRED TO OUR CENTER

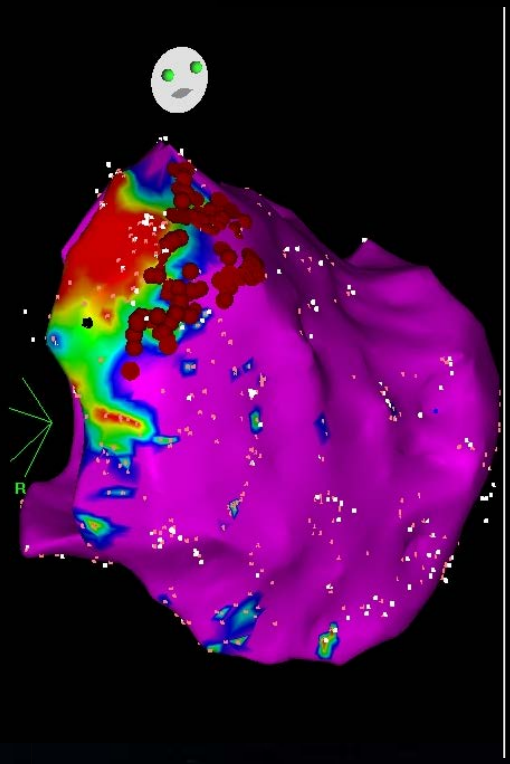
Arrhythmogenic Right Ventricular Dysplasia

ENDOCARDIAL RV

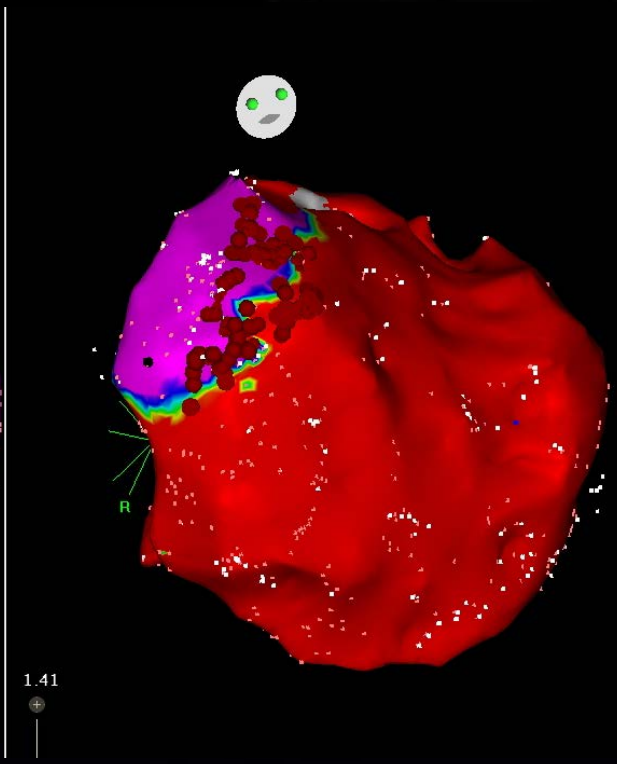


BIPOLAR

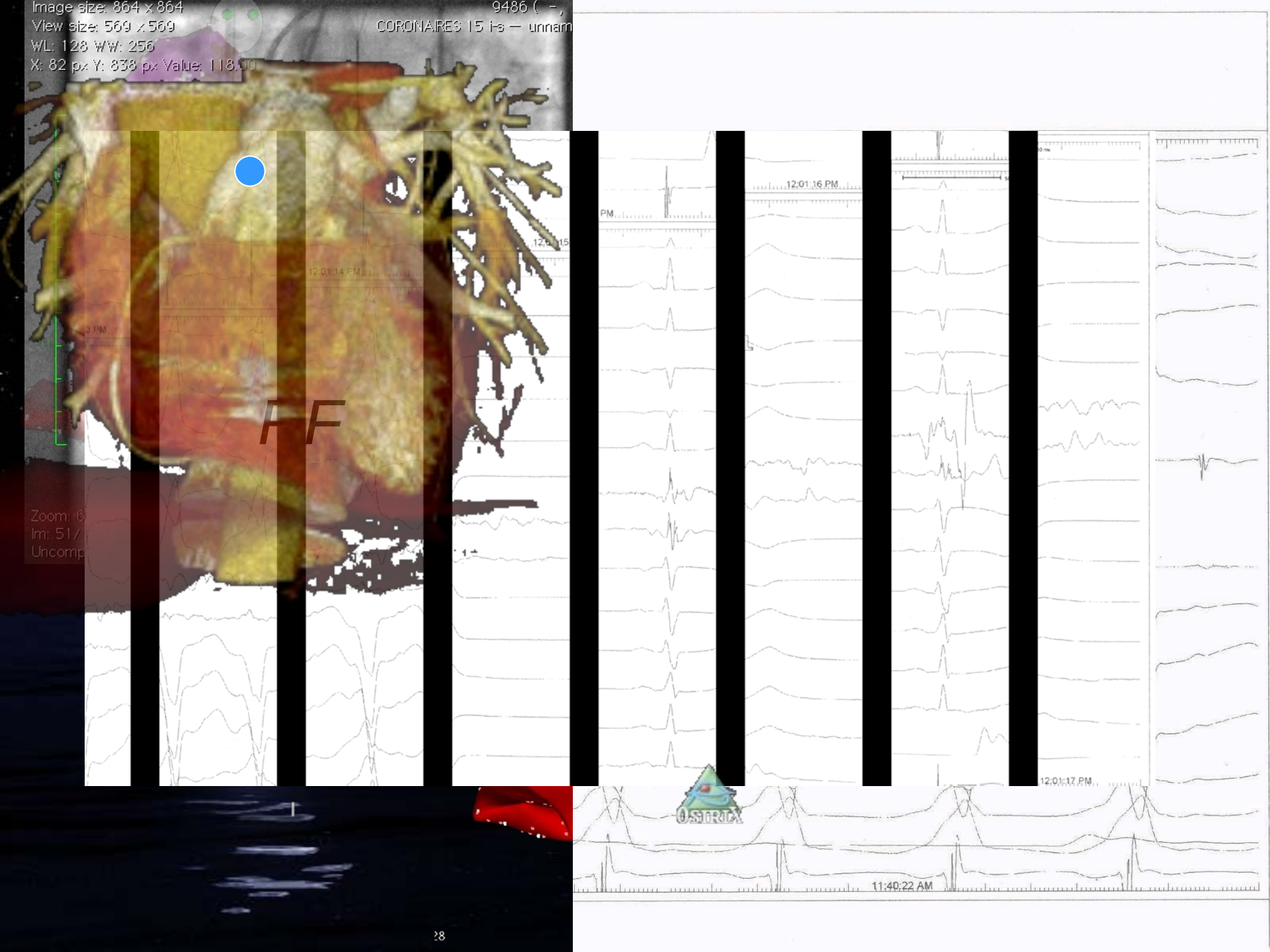
EPICARDIAL RV



BIPOLAR



LATE POTENTIALS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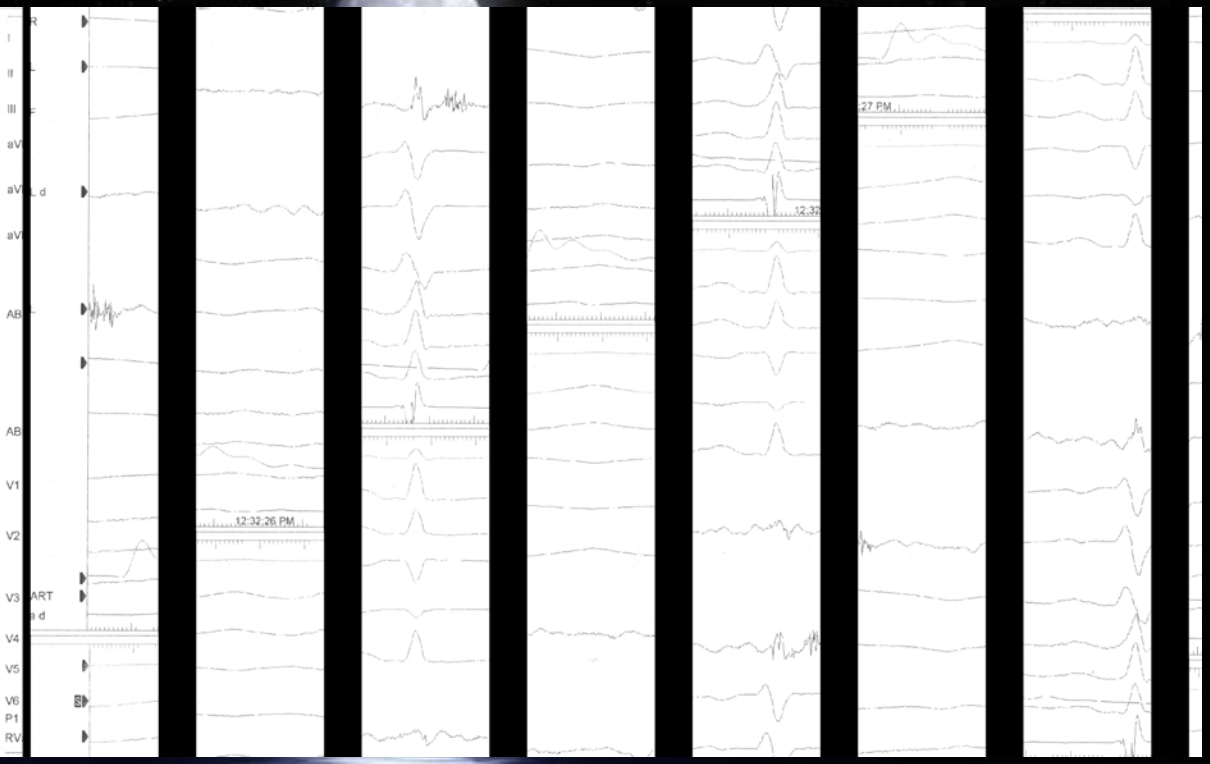
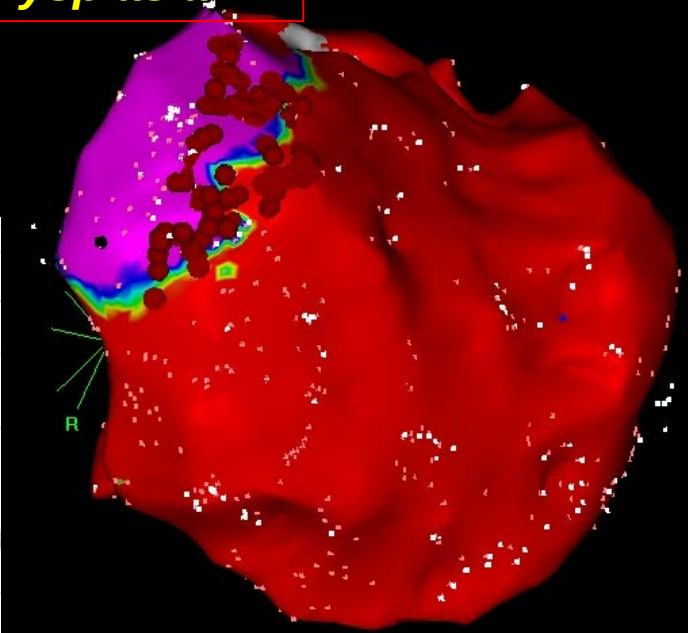
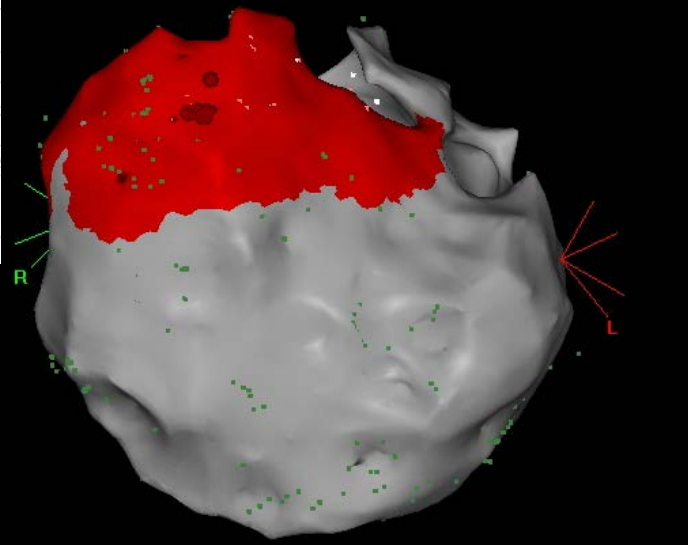
Arrhythmogenic Right Ventricular Dysplasia

BEFORE RF

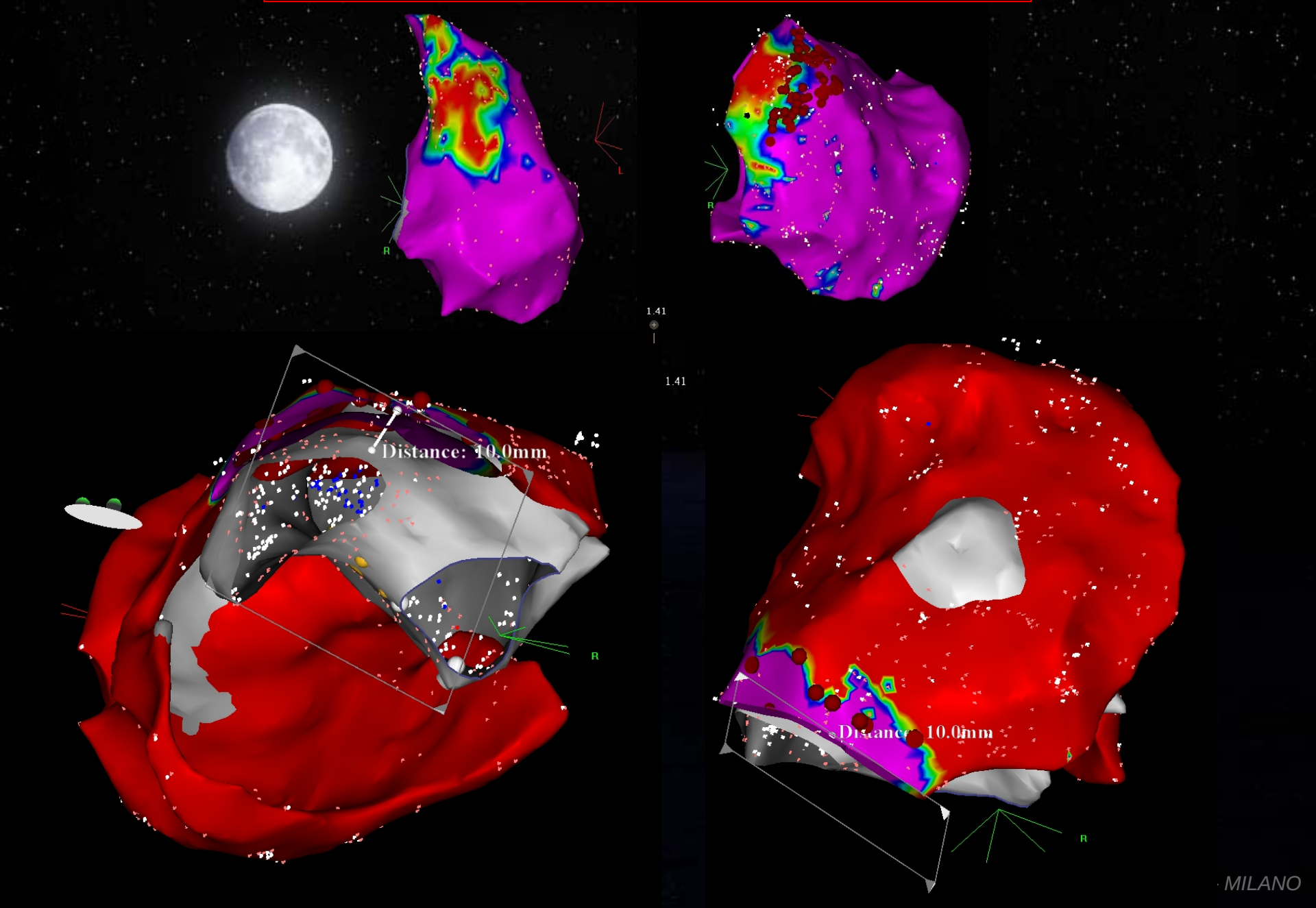




REMAP AFTER RF



Arrhythmogenic Right Ventricular Dysplasia



Cardiomyopathies are traditionally defined on the basis of structural and functional phenotypes,notably dilated,hypertrophic,and restrictive.

THE DILATED CARDIOMYOPATHY PHENOTYPE

is the most common and is often viewed as a "final common pathway" of numerous types of cardiac injuries

	HCM	DCM	ARVC	RCM	Unclassified
Familial	Familial, unknown gene Sarcomeric protein mutations ß myosin heavy chain Cardiac myosin binding protein C Cardiac troponin I Troponin-T α-tropomyosin Essential myosin light chain Regulatory myosin light chain Cardiac actin α-myosin heavy chain Titin Troponin C Muscle LIM protein Glycogen storage disease (e.g. Pompe; PRKAG2, Forbes', Danon) Lysosomal storage diseases (e.g. Anderson–Fabry, Hurler's) Disorders of fatty acid metabolism Carnitine deficiency Phosphorylase B kinase deficiency Mitochondrial cytopathies Syndromic HCM Noonan's syndrome LEOPARD syndrome Friedreich's ataxia Beckwith–Wiedermann syndrome Swyer's syndrome Other Phospholamban promoter Familial amyloid	Familial, unknown gene Sarcomeric protein mutations (see HCM) Z-band Muscle LIM protein TCAP Cytoskeletal genes Dystrophin Desmin Metavinculin Sarcoglycan complex CRYAB Epicardin Nuclear membrane Lamin A/C Emerin Mildly dilated CM Intercalated disc protein mutations (see ARVC) Mitochondrial cytopathy	Familial, unknown gene Intercalated disc protein mutations Plakoglobin Desmoplakin Plakophilin 2 Desmoglein 2 Desmocollin 2 Cardiac ryanodine receptor (RyR2) Transforming growth factor-β3 (TGFβ3)	Familial, unknown gene Sarcomeric protein mutations Troponin I (RCM +/− HCM) Essential light chain of myosin Familial amyloidosis Transthyretin (RCM + neuropathy) Apolipoprotein (RCM + nephropathy) Desminopathy Pseuxanthoma elasticum Haemochromatosis Anderson–Fabry disease Glycogen storage disease	Left ventricular non-compaction Barth syndrome Lamin A/C ZASP α-dystrobrevin
Non-familial	Obesity Infants of diabetic mothers Athletic training Amyloid (AL/prealbumin)	Myocarditis (infective/toxic/immune) Kawasaki disease Eosinophilic (Churg Strauss syndrome) Viral persistence Drugs Pregnancy Endocrine Nutritional — thiamine, carnitine, selenium, hypophosphataemia, hypocalcaemia Alcohol Tachycardiomyopathy	Inflammation?	Amyloid (AL/prealbumin) Scleroderma Endomyocardial fibrosis Hypereosinophilic syndrome Idiopathic Chromosomal cause Drugs (serotonin, methysergide, ergotamine, mercurial agents, busulfan) Carcinoid heart disease Metastatic cancers Radiation Drugs (anthracyclines)	Tako Tsubo cardiomyopathy

Table 1. Known Causes of Dilated Cardiomyopathy.

Toxins

Ethanol*
Chemotherapeutic agents (doxorubicin, bleomycin)
Cobalt*
Antiretroviral agents (zidovudine,* didanosine,* zalcitabine*)
Phenothiazines*
Carbon monoxide*
Lead*
Cocaine*
Mercury*

Metabolic abnormalities

Nutritional deficiencies (thiamine,* selenium,* carnitine*)
Endocrinologic disorders (hypothyroidism,* acromegaly,* thyrotoxicosis,* Cushing's disease, pheochromocytoma,* diabetes mellitus)
Electrolyte disturbances (hypocalcemia,* hypophosphatemia*)

Inflammatory or infectious causes

Infectious
- Viral (coxsackie virus, cytomegalovirus,* human immunodeficiency virus)
- Rickettsial
- Bacterial (diphtheria*)
- Mycobacterial
- Fungal
- Parasitic (toxoplasmosis,* trichinosis, Chagas' disease)

Noninfectious
- Collagen vascular disorders (scleroderma, lupus erythematosus, dermatomyositis)
- Hypersensitivity myocarditis*
- Sarcoidosis*
- Peripartum dysfunction*

Neuromuscular causes

Duchenne's muscular dystrophy
Facioscapulohumeral muscular dystrophy
Erb's limb-girdle dystrophy
Myotonic dystrophy
Friedreich's ataxia

Familial cardiomyopathies

*Potentially reversible, either spontaneously or with treatment, according to case reports.

ARRHYTHMOGENESIS IN IDCM

- *Myocardial fibrosis,myocyte disarray,and membrane abnormalities*
- *Autopsy studies have shown substantial left ventricular subendocardial scarring in 33% of patients and patchy areas of replacement fibrosis in 57%, accompanied by increased perivascular fibrous tissue and perimyocytic fibrosis in the left ventricle*
- *Slow conduction through muscle bundles separated by interstitial fibrosis can cause a zigzag path and promote reentry*

Epidemiology of dilated cardiomyopathy

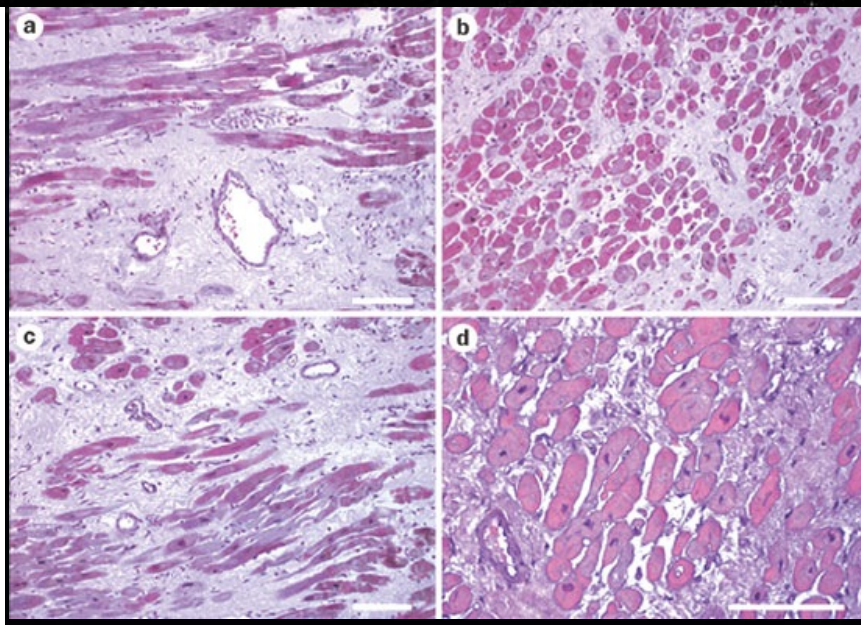
A prospective post-mortem study of 5252 necropsies

S. Rakar, G. Sinagra, A. Di Lenarda, A. Poletti, R. Bussani*, F. Silvestri*, F. Camerini and the Heart Muscle Disease Study Group

Table 2 Dilated cardiomyopathy: microscopic findings

Patients	Fibrosis (0–3; f/p)	Hypertrophy (0–3)	Thinned myocytes (f/w)	Interstitial oedema (0–3)	Fatty infiltration (0–3; f/w)	Inflammatory infiltrates (+/−)	Arteriolar involvement (0–3)	Myofibre disarray (+/−)	Endocardial thickening (0–3)
1	3/f	2	w	2	1/f	−	1	+	1
2	3/p	1	w	2	1/f	−	0	−	2
3	1/p	1	w	2	1/f	−	0	−	0
4	1/p	1	f	1	2/f	−	1	−	0
5	1/f	1	w	1	3/f	−	0	+	1
6	2/p	2	f	2	2/f	−	0	+	0
7	1/p	1	w	1	1/f	−	1	−	0
8	2/f	1	w	1	1/f	−	0	−	2
9	2/f	2	f	1	2/f	−	1	−	0
10	2/f	1	w	2	2/f	+	0	−	1
11	1/f	2	w	1	0	−	0	−	2
12	1/p	1	f	1	2/f	+	0	−	2
13	2/f	1	w	1	0	+	0	−	2
14	1/p	1	w	1	0	+	0	−	2
15	1/f	1	w	1	1/f	+	0	−	0
16	1/f	0	f	1	1/f	−	1	−	1
17	1/f	1	w	2	3/f	−	2	−	2
18	1/p	1	f	1	2/w	−	0	−	0
19	1/p	2	f	2	1/f	−	2	−	0
20	1/p	2	w	1	1/f	−	2	−	1
21	1/p	0	w	2	0	−	2	−	2
22	1/f	1	w	1	2/f	−	1	−	0
23	3/p	1	w	1	0	−	0	−	—
24	2/f	1	w	1	0	+	0	−	2

f=Focal; p=perivascular/pericellular; w=widespread.



Neilan, T. **CMR quantification of myocardial scar provides additive prognostic information in nonischemic cardiomyopat***hy. JACC Cardiovascular Imaging*, *6*(9), 944–954. *2013*

The LGE pattern was mid-myocardial in 52% of the patients, epicardial 26%, focal/insertion points in 20%, and diffuse in 2%.

The Characteristics and Distribution of the Scar Tissue Predict Ventricular Tachycardia in Patients with Advanced Heart Failure

MIKI YOKOKAWA, M.D.,* HIROSHI TADA, M.D.,* KEIKO KOYAMA, M.D.,† TOSHIHIKO INO, R.T.,† SHIGEKI HIRAMATSU, M.D.,* KENICHI KASENO, M.D.,* SHIGETO NAITO, M.D.,* SHIGERU OSHIMA, M.D.,* and KOICHI TANIGUCHI, M.D.*
From the *Division of Cardiology, and †Radiology, Gunma Prefectural Cardiovascular Center, Maebashi, Gunma Japan

Results: *In the DCM group, almost all hyperenhanced areas were nontransmural, and presented frequently in the midwall layer. The volume of the hyperenhanced areas and total number of hyperenhanced segments were greater in patients with sustained VT than in those without. On the other hand, in the ICM group, transmural or subendocardial hyperenhanced areas were detected in the territory of the coronary arteries. The volume of the hyperenhanced areas and total number of transmural hyperenhanced segments in patients with sustained VT were unexpectedly smaller than in those without. However, the percentage of nontransmural hyperenhanced segments was greater in patients with sustained VT than in those without.*

Compared with post-MI VT, the scar tends to be smaller and less confluent, the total number of the transmural scar segments is significantly smaller, and with less endocardial involvement in nonischemic CMP.

Table III.

Measurement Variables of CMR in Patients with Dilated Cardiomyopathy (DCM) and Ischemic Cardiomyopathy (ICM)

	DCM (n = 29)	ICM (n = 18)	P Value
Enhancement, patient n (%)	18 (62)	18 (100)	<0.005
The volume of the hyperenhanced areas, %	6 ± 9	36 ± 14	<0.0001
Total hyperenhanced segments, n	3 ± 4	12 ± 3	<0.0001
Transmural hyperenhanced segments, n	0 ± 0	3 ± 2	<0.0001
Nontransmural hyperenhanced segments, n	3 ± 4	8 ± 3	<0.0001
Nontransmural /total hyperenhanced areas, %	97 ± 6	67 ± 20	<0.0001

Delayed-enhancement MR imaging typically reveals nontransmural scar areas often distributed in the basal portion of the ventricular free wall or basal to midportion of the septum.

Table IV.

Measurement Variables of CMR in Patients with and Without Sustained VT in the Dilated Cardiomyopathy (DCM) and Ischemic Cardiomyopathy (ICM) Groups

	DCM (n = 29)			ICM (n = 18)		
	VT (+) (n = 12)	VT (−) (n = 17)	P Value	VT (+) (n = 8)	VT (−) (n = 10)	P Value
Enhancement, patient n (%)	12 (100)	6 (35)	<0.0001	8 (100)	10 (100)	1.0
The volume of the hyperenhanced areas, %	13 ± 11	2 ± 3	<0.01	25 ± 5	46 ± 11	<0.0001
Total hyperenhanced segments, n	6 ± 4	1 ± 2	<0.0001	11 ± 2	12 ± 3	0.8
Transmural hyperenhanced segments, n	0 ± 0	0 ± 0	0.9	2 ± 1	4 ± 2	<0.005
Nontransmural hyperenhanced segments, n	6 ± 4	1 ± 2	<0.0001	9 ± 2	7 ± 3	0.7
Nontransmural/total hyperenhanced areas, %	96 ± 7	100 ± 0	1.0	79 ± 11	57 ± 21	<0.05

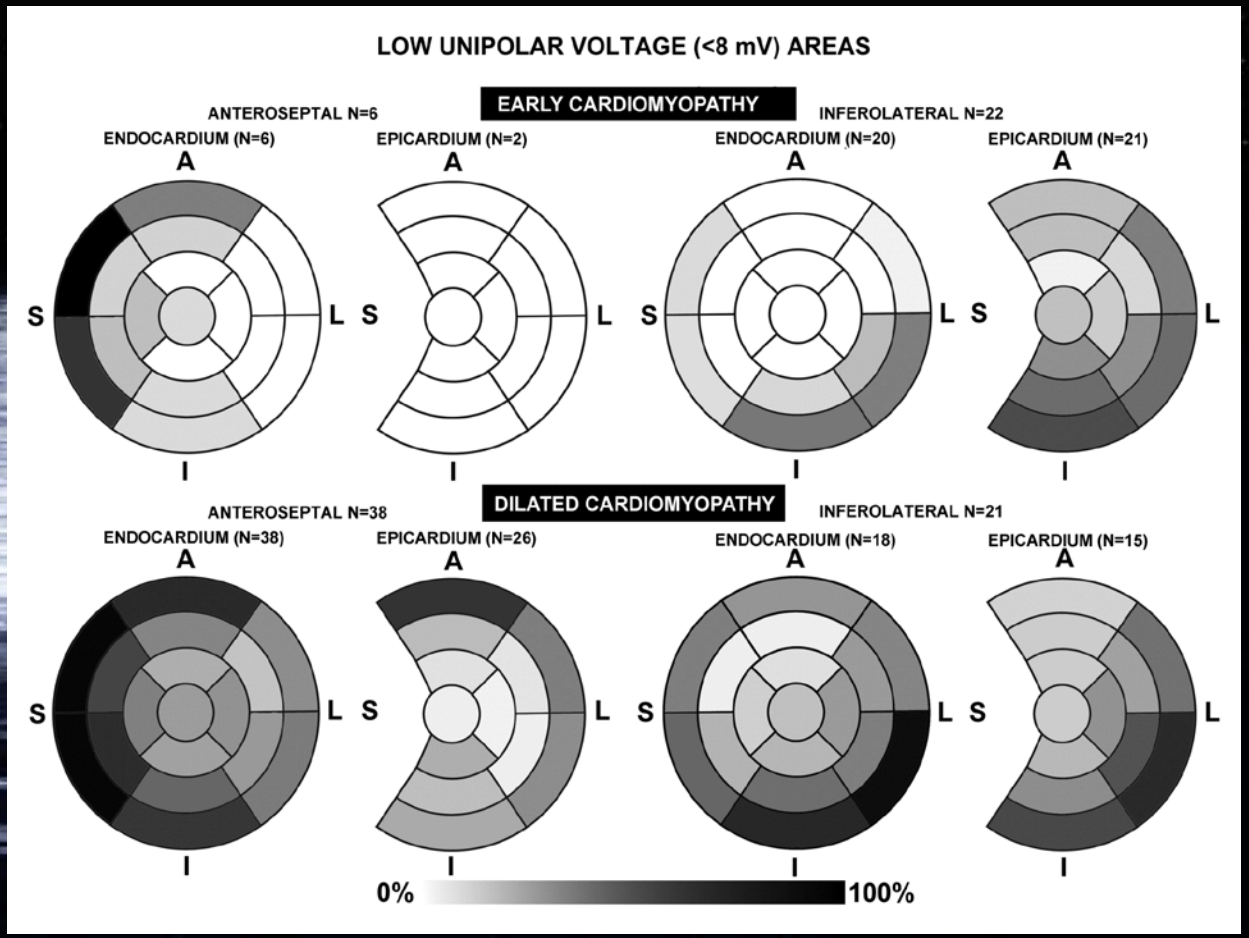
The values are the mean ± SD.
CMR = contrast-enhanced magnetic resonance imaging; n = number; VT = ventricular tachycardia.

Oloriz, T., Silberbauer, J., Maccabelli, G et al. **Catheter Ablation of Ventricular Arrhythmia in Nonischemic Cardiomyopathy: Anteroseptal Versus Inferolateral Scar Sub-Types**. Circulation: Arrhythmia and Electrophysiology, 7(3), 2014

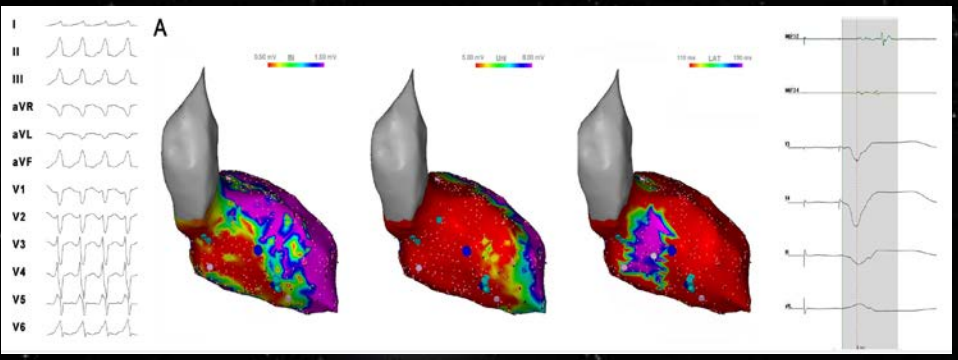
STUDY POPULATION
87 patients (CARTO)

28 Early-Cardiomyopathy: LV volume: normal or mildly dilated, EF ≥45%
59 Dilated-Cardiomyopathy, LV volume: moderate or severe dilatation, EF ≤45%

Patients were categorized as predominant Anteroseptal or Inferolateral scar type based on the majority percentage of endocardial segments (eight segments each) displaying a unipolar scar voltage less than 8mV, excluding the apex.



ANTEROSEPTAL SCAR PATTERN



INFEROLATERAL SCAR PATTERN

VT morphology:

Left inferior axis
(positive predictive value, 100%)

Right superior axis
(positive predictive value, 89%)

Late potentials

Infrequent
(11% versus 74%; P<0.001)

Common
(81% versus 4%; P<0.001)

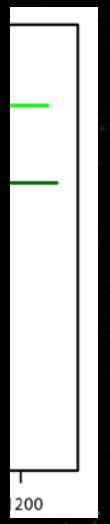
ABLATION SITE

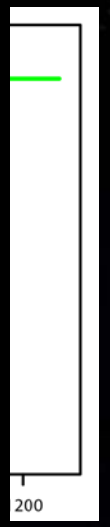
OUTCOME

ADDITIONAL CLUES

The higher proportion of anteroseptal patients fulfilling diagnostic criteria for DCM in our data may related to more extensive endocardial scarring and common intraventricular conduction delay, frequently as a left bundle branch block pattern, causing LV dyssynchrony and dysfunction, leading to a worse prognosis

AS-E
AS-D





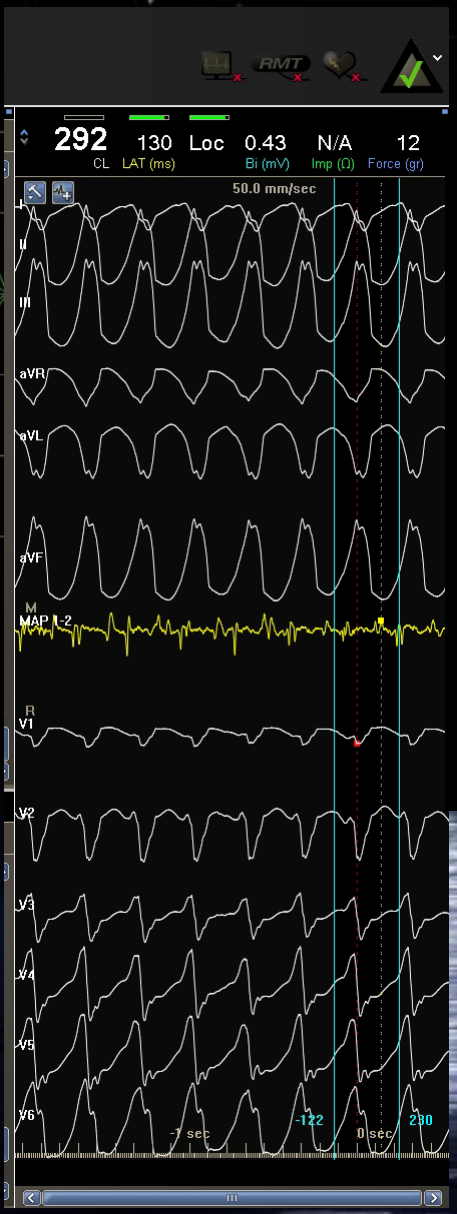
A high proportion of inferolateral patients with or without LV dysfunction had a clinically suspected myocarditis supporting the possible role of viral infection in this subgroup

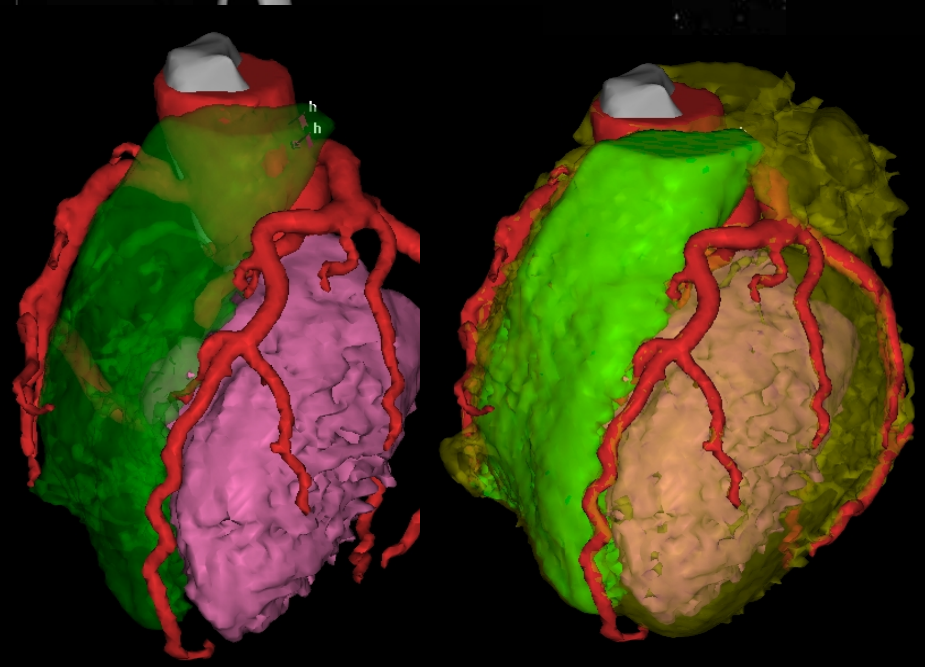
STRAT

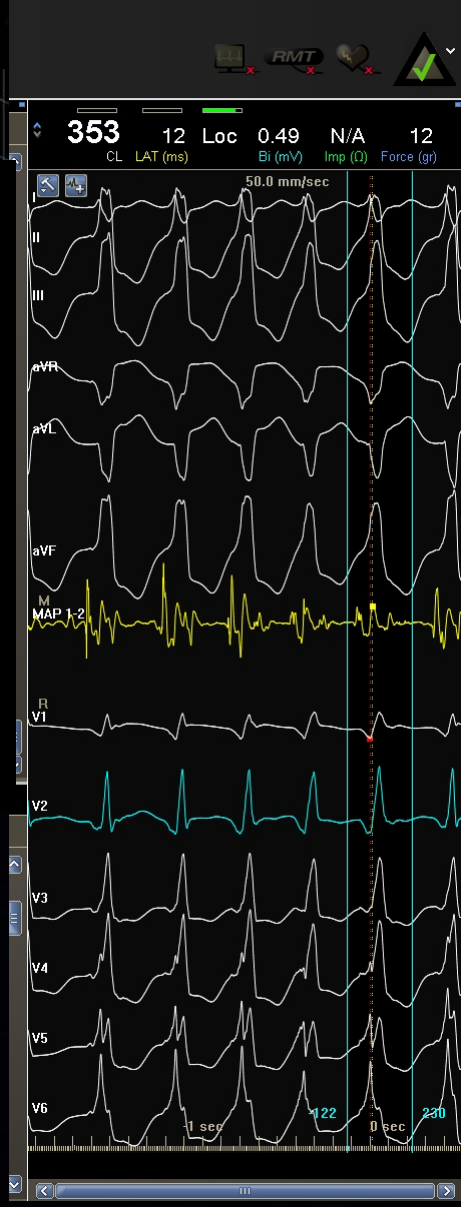
LEFT INFERIOR VT AXIS – AS PATTERN – ENDOCARDIAL ABLATION

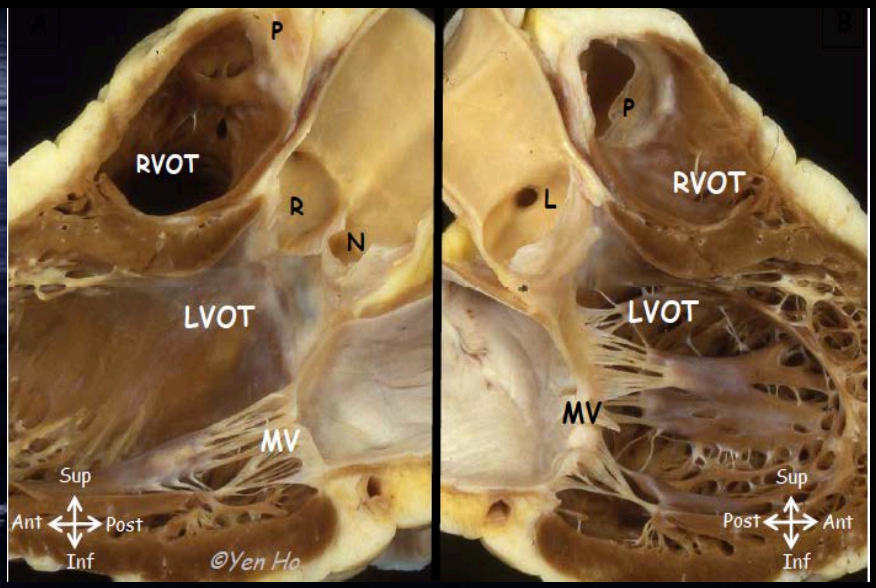
RIGHT SUPERIOR VT AXIS – IL PATTERN – ENDO-EPICARDIAL ABLATION

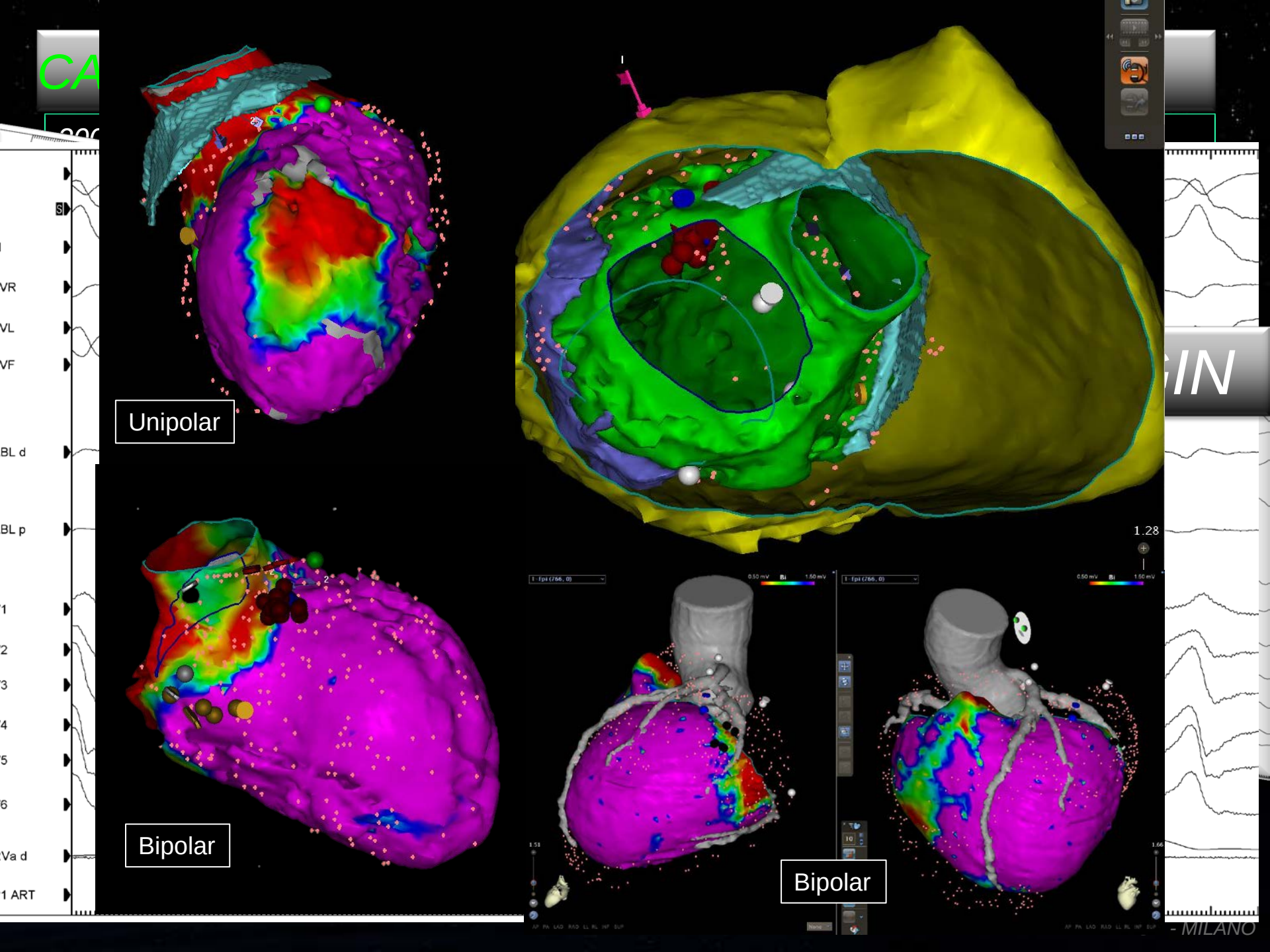
Frequent pattern in IDCM



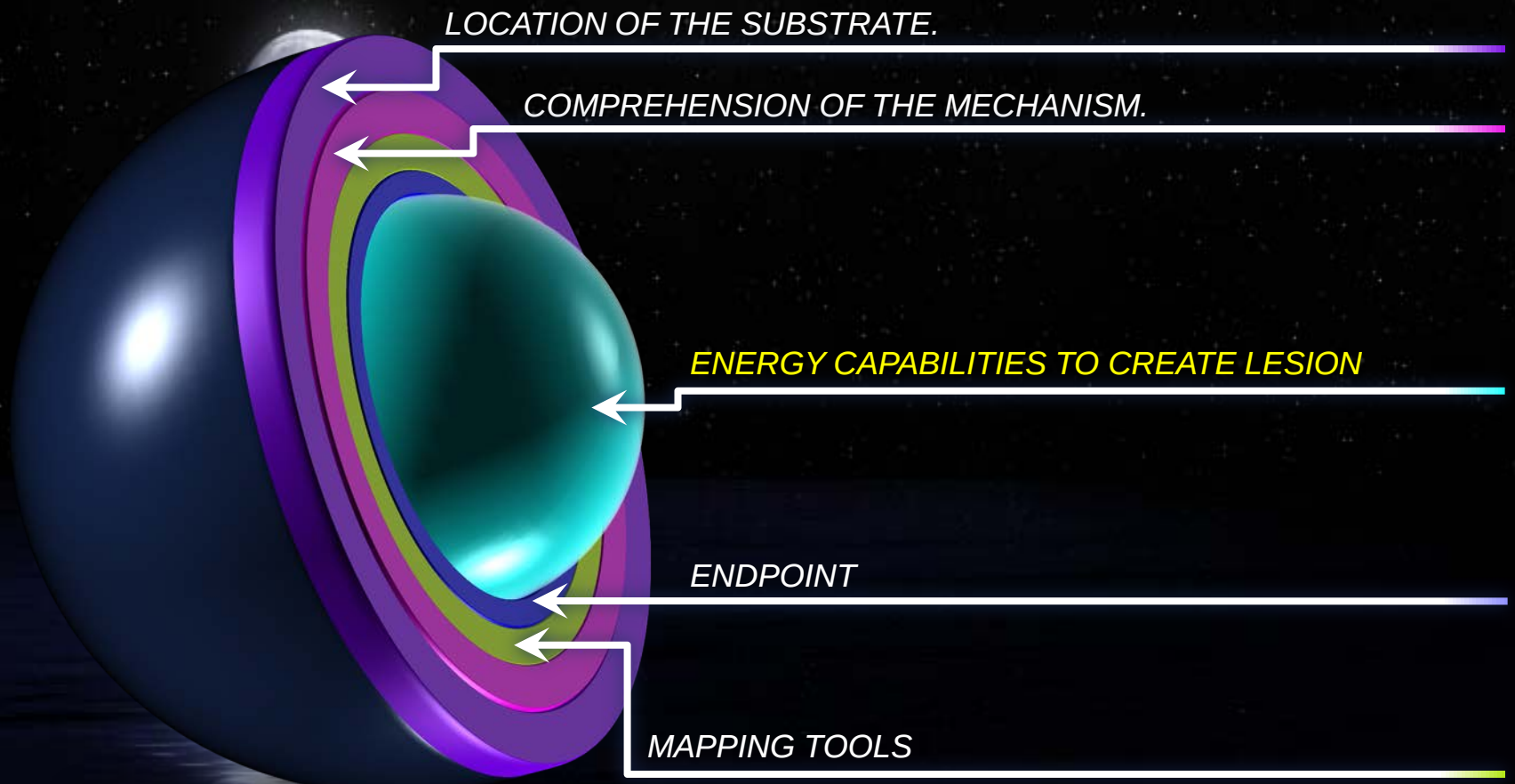







Unipolar
Bipolar
Bipolar
1.28

Building to the Core of a successful VT ablation

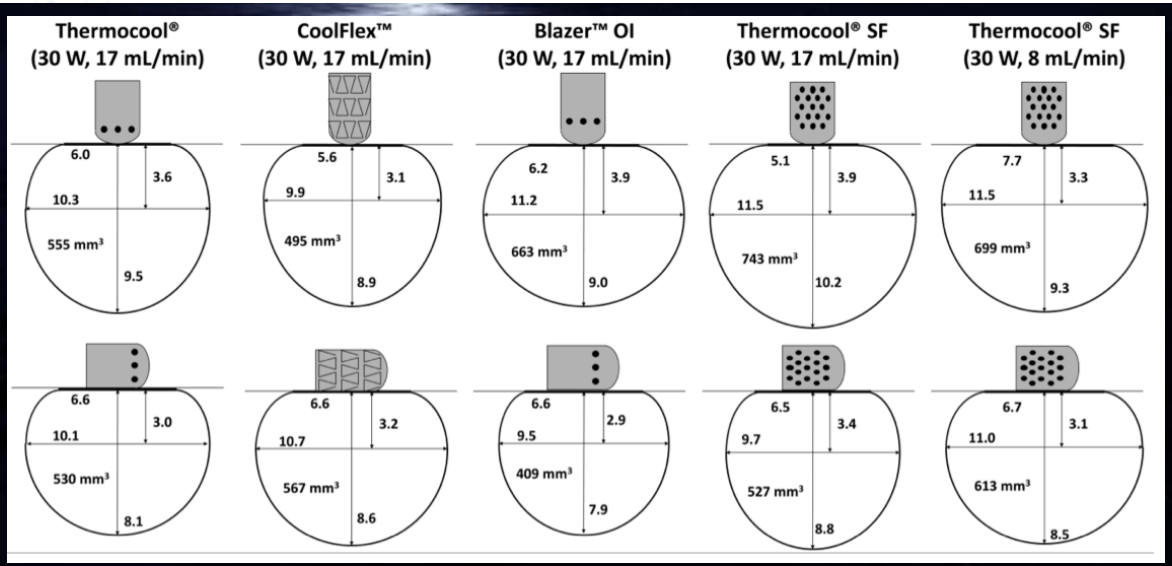


ENERGY CAPABILITIES TO CREATE LESION: THE STUMBLING BLOCK

RADIOFREQUENCY ENERGY

	Cooled-Tip RF Ablation (n=65)			Standard RF Ablation (n=33)	
	Normal Tissue (n=37)	Normal Tissue on Fat (2.6±1.2 mm) (n=7)	Infarcted Tissue (n=21)	Normal Tissue (n=22)	Normal Tissue on Fat (3.1±1.2 mm) (n=11)
Power, W	44.8±6.8	45±4.4	35.6±7.1*	25±16.8*	16±14
Temperature, °C	39.7±3.2	40.2±2	41.4±2.2	68±6.2*	69±4.1
Initial impedance, Ω	146±24	146±16	110±12*	148±13	145±11
Impedance drop, Ω	27.8±10.3	27.6±10.1	19.7±4.4*	23.8±4*	17.1±2
Lesion characteristics					
Long axis, mm	15.9±3.5	15.6±4.2	14.6±2.7	14.9±2.7	11±4.7
Short axis, mm	13.7±3.5*	12.3±4.3	11.8±2.9	11±2.7	9±2.4
Depth, mm†	6.7±1.7* (4–9)	4.1±2 (1–7)	5.5±1.2* (4–8)	3.7±1.3 (2–6)	None

D'Avila et al Circulation, 2004



Moreno, J. JCE in press

Biophysical Parameters During Radiofrequency Catheter Ablation of Scar-Mediated Ventricular Tachycardia: Epicardial and Endocardial Applications via Manual and Magnetic Navigation

TARA BOURKE, M.D., ERIC BUCH, M.D., M.S., NILESH MATHURIA, M.D., YOAV MICHOWITZ, M.D., RICKY YU, B.S., RAVI MANDAPATI, M.D., KALYANAM SHIVKUMAR, M.D., PH.D., and RODERICK TUNG, M.D.

From the UCLA Cardiac Arrhythmia Center, UCLA Health System, David Geffen School of Medicine at UCLA, Los Angeles, California, USA

Ablation Effectiveness and Biophysical Parameters. *Background:* There is a paucity of data on biophysical parameters during radiofrequency ablation of scar-mediated ventricular tachycardia (VT).

Methods and Results: Data were collected from consecutive patients undergoing VT ablation with open-irrigation. Complete data were available for 372 lesions in 21 patients. The frequency of biophysical parameter changes were: >10Ω reduction (80%), bipolar EGM reduction (69%), while loss of capture was uncommon (32%). Unipolar injury current was seen in 72% of radiofrequency applications. Both EGM reduction and impedance drop were seen in 57% and a change in all 3 parameters was seen in only 20% of lesions. Late potentials were eliminated in 33%, reduced/modified in 56%, and remained after ablation in 11%. Epicardial lesions exhibited an impedance drop (90% vs. 76%, $P = 0.002$) and loss of capture (46% vs. 27%, $P < 0.001$) more frequently than endocardial lesions. Lesions delivered manually exhibited a >10Ω impedance drop (83% vs. 71%, $P = 0.02$) and an EGM reduction (71% vs. 40%, $P < 0.001$) more frequently than lesions applied using magnetic navigation, although loss of capture, elimination of LPs, and a change in all 3 parameters were similarly observed.

Conclusions: VT ablation is inefficient as the majority of radiofrequency lesions do not achieve more than one targeted biophysical parameter. Only one-third of RF applications targeted at LPs result in complete elimination. Epicardial ablation within scar may be more effective than endocardial lesions, and lesions applied manually may be more effective than lesions applied using magnetic navigation. New technologies directed at identifying and optimizing ablation effectiveness in scar are clinically warranted. *(J Cardiovasc Electrophysiol, Vol. pp. 1-9)*

HINT

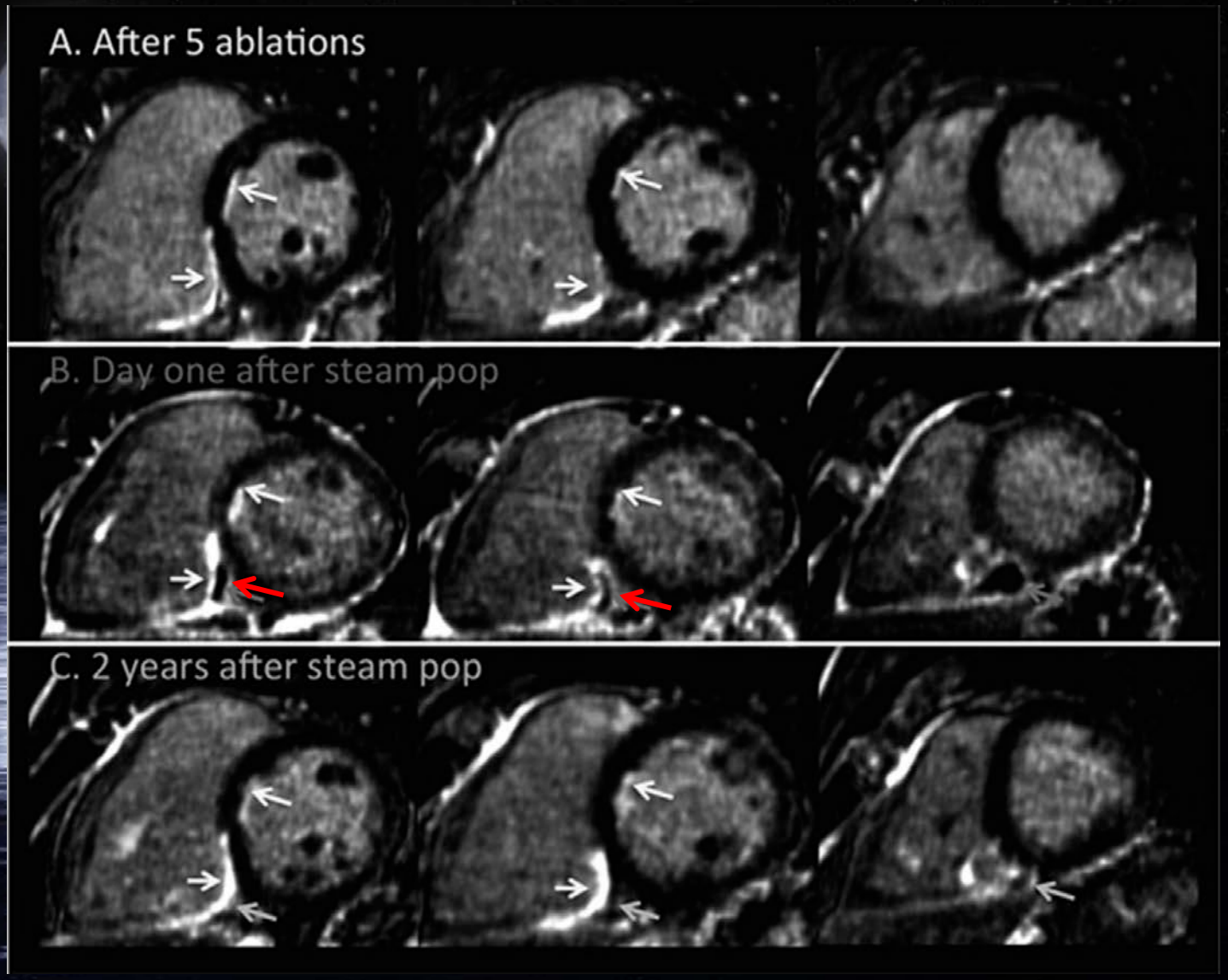
INCREASE THE ENERGY ??

	50W, 30 mL/min				50W, 15 mL/min	Global
	BW ThermoCool®	SJM CoolFlex™	BSc Blazer™ OI	BW SF-30	BW SF-15	p value
# Lesions	12	12	12	12	12	
Voltage (V)	60.7±1.8	60.6±1.6	59.6±1.8	59.8±2.5	60.8±1.6	n.s.
Current (mA)	805±17	834±12	845±20	835±22	826±18	n.s.
Power (W)	49.8±0.2	49.9±0.1	49.8±0.4	49.8±0.2	49.9±0.2	n.s.
Impedance (Ω)						
- Initial	121±14	110±16	115±16	117±15	123±16	n.s.
- Final	93±12	96±18	92±17	88±15	92±11	n.s.
- Minimum	85±11	84±14	82±13	83±15	87±13	n.s.
Tip Temperature (°C)						
- Mean	38.4±3.2 * •	41.2±6.5 ₸‡¶	34.8±2.5 ‡	33±1.4 *₸	33.4±0.8 •¶	<0.001
- Maximum	43.9±4.8	48.2±14.6*•	42±11.8	37.9±1.8•	36.8±1.2*	0.008
Pop before 30s	1 (8%)	8 (67%)	0 (0%)	2 (17%)	3 (25%)	
Pop before 60s	3 (25%)	12 (100%)	4 (33%)	2 (17%)	6 (50%)	
Pop before 90s	9 (75%)	12 (100%)	9 (75%)	8 (67%)	8 (67%)	
Pop before 180s	12 (100%)	12 (100%)	12 (100%)	9 (75%)	11 (91%)	

Perpendicular Article

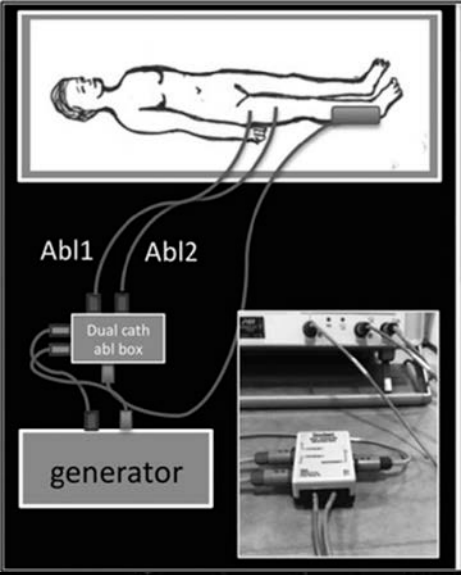
Moreno, J. JCE in press

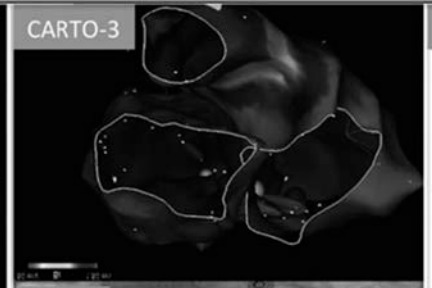
MRI data before and after sixth ablation procedure.

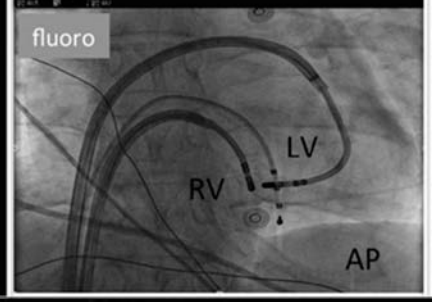


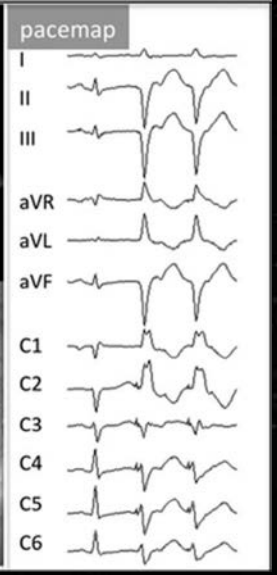
Berte, Circulation 2014

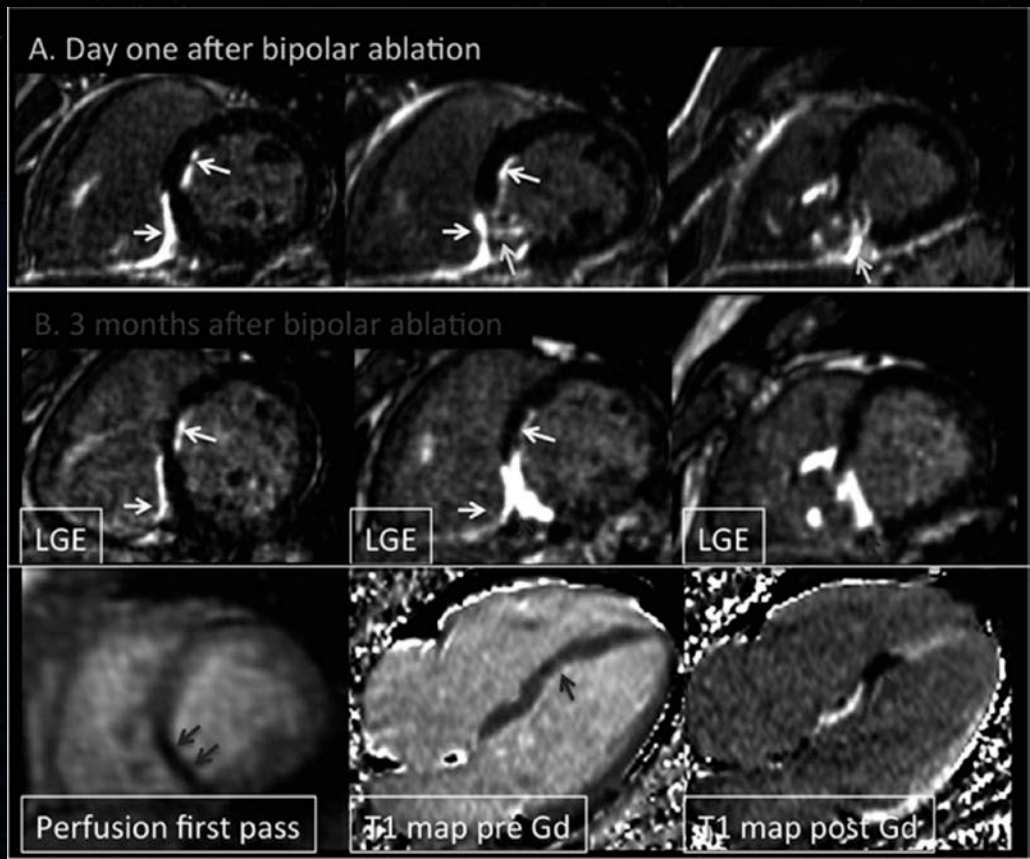
Bipolar ablation





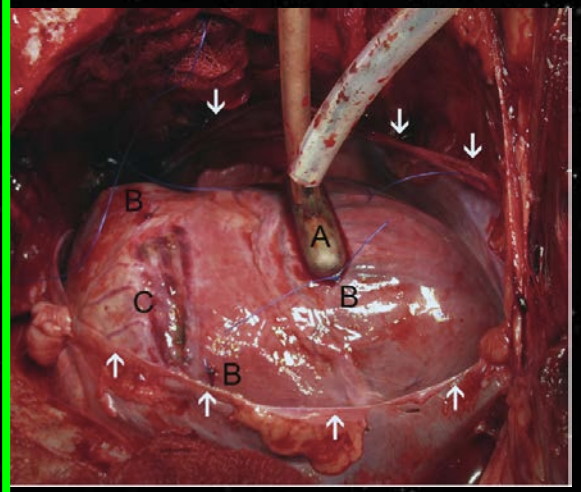






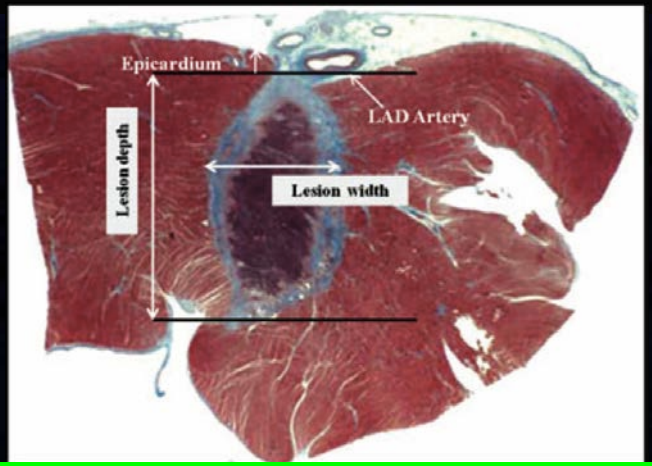
Alternative energy delivery modalities

Sapp JL, Beeckler C, Pike C, Parkash R, Gray CJ, Zeppenfeld K, Kuriachan V, Stevenson WG. ***Initial human feasibility of infusion needle catheter ablation for refractory ventricular tachycardia.*** *Circulation. 2013;128:2289-2295.*

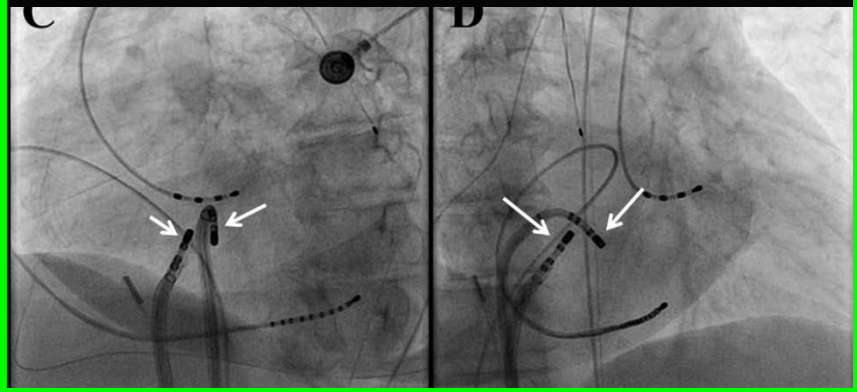


Neven, K., van Driel, V., van Wessel, H., van Es, R., DOEVENDANS, P. A., & Wittkampf, F (2014). Epicardial linear electroporation ablation and lesion size. Heart Rhythm 11(8), 1465-1470.

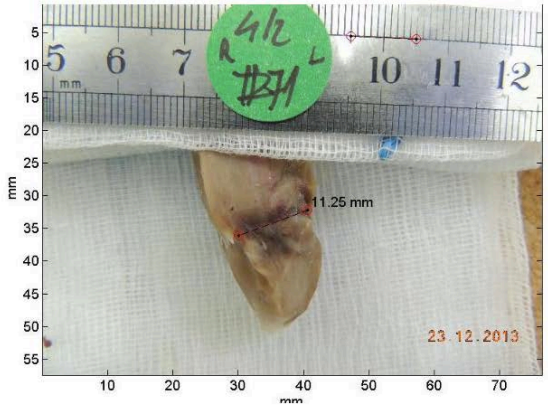
Koruth JS, Dukkipati S, Carrillo RG, Coffey J, Teng J, Eby TB, Reddy VY, D'avila A. ***Safety and efficacy of High-Intensity focused ultrasound atop coronary arteries during epicardial catheter ablation.*** *Journal of Cardiovascular Electrophysiology. 2011;22:1274-1280*



Koruth JS, Dukkipati S, Miller MA, Neuzil P, D'Avila A, Reddy VY. ***Bipolar irrigated radiofrequency ablation: a therapeutic option for refractory intramural atrial and ventricular tachycardia circuits.*** *Heart Rhythm. 2012;9:1932–1941.*



Bipolar ablation lesion assessment



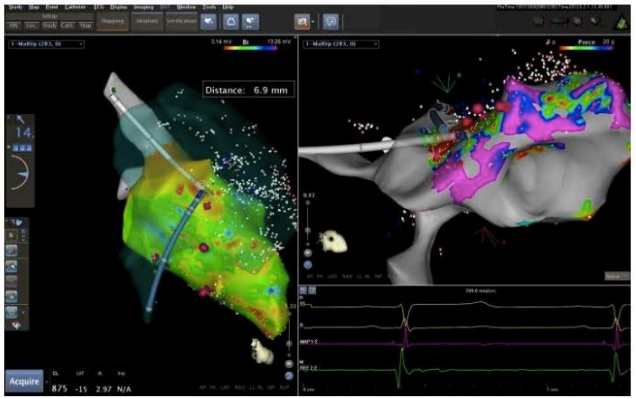
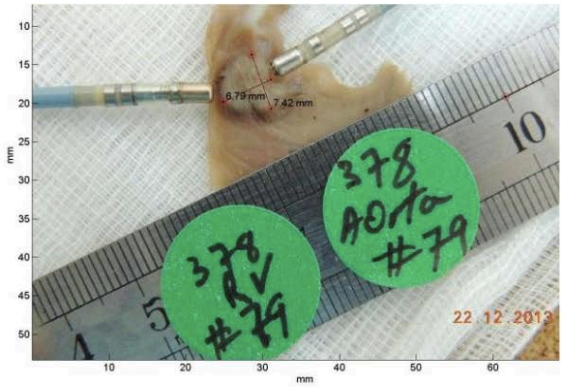


Figure 4- Animal #71 Lesion #4 (point # 412)



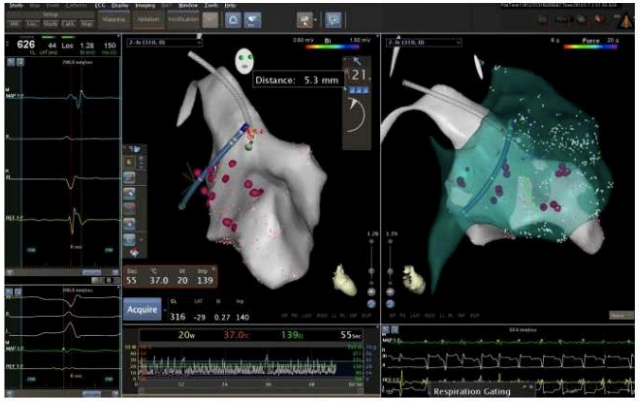


Figure 5 - Animal # 79 lesion # 6 (point #378)

		Ablation parameters				(mm)		(mm)		Comment
Animal No.	79									
# point on C3	Abl. Session	Map cath. location	RF power (W)	Time (sec)	Average force (g)	Inner depth	Outer depth	Inner width	Outer width	
#368	1	LV	20	60	19	4.4	6.85	6.55	9.83	RV lesion was not found
#370	2	LV	20	60	10	11.36		RV 8.22	LV 10.3	TM lesion
#380	3	LV	25	60	15	8.54	9.67	9.3	13.01	
#380	3	RV	25	60	15	7.51	8.34	9.51	13.68	
#374	4	LV	25	60	15	5.77	7.75	8.63	11.64	
#374	4	RV	25	60	15	5.87	6.74	8	11.96	
#377	5	LV	25	46	5	14.68		RV 5.58	LV 9.49	TM lesion
#378	6	RV-RVOT	20	60	10	8.63		RV8.69	LV 10.19	TM lesion
#380 (8)	7	LV-FW	25	60	15	1.87	6	4.17	6.56	RV lesion not found

Thanks for your attention

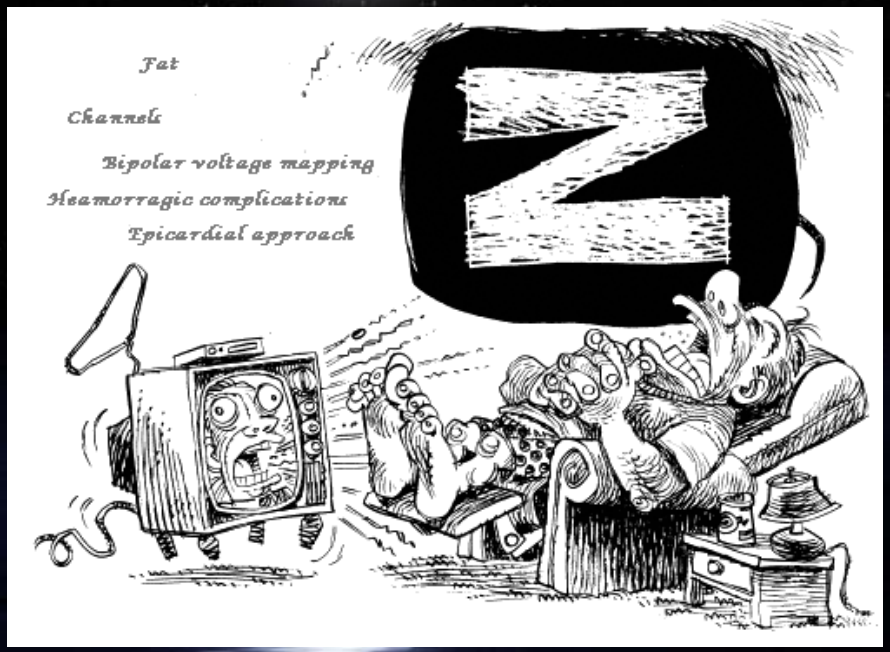
Gracias por su atención

Danke für Ihre Aufmerksamkeit

Merci pour votre attention

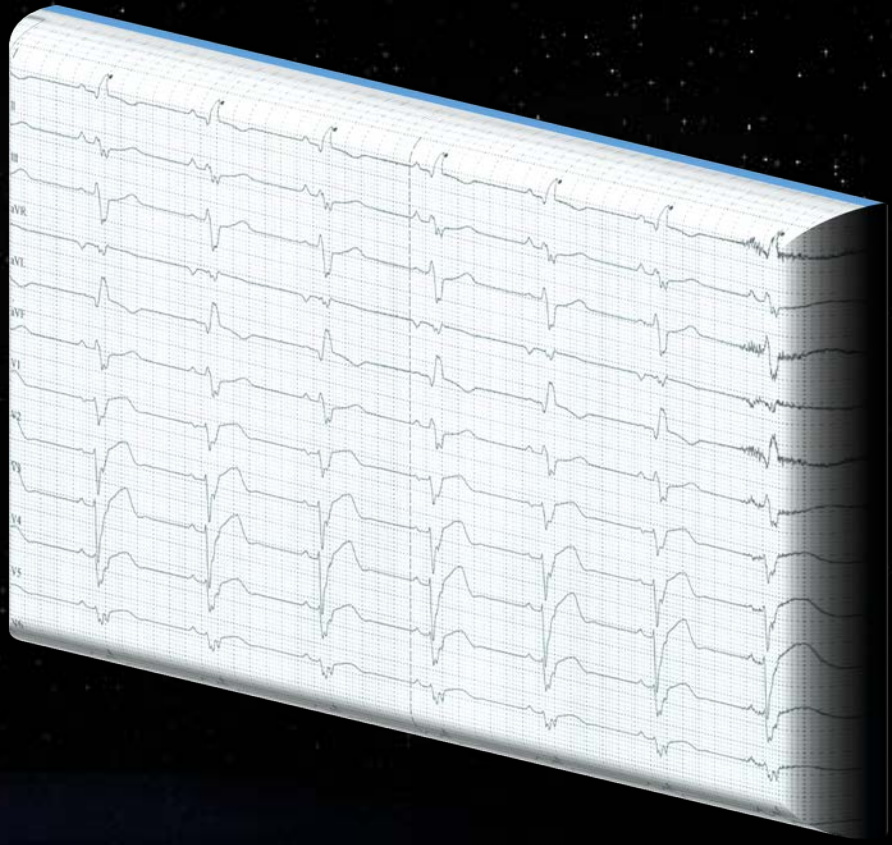
당신의 주의를 위한 감사합니다

спасибо для вашего внимания

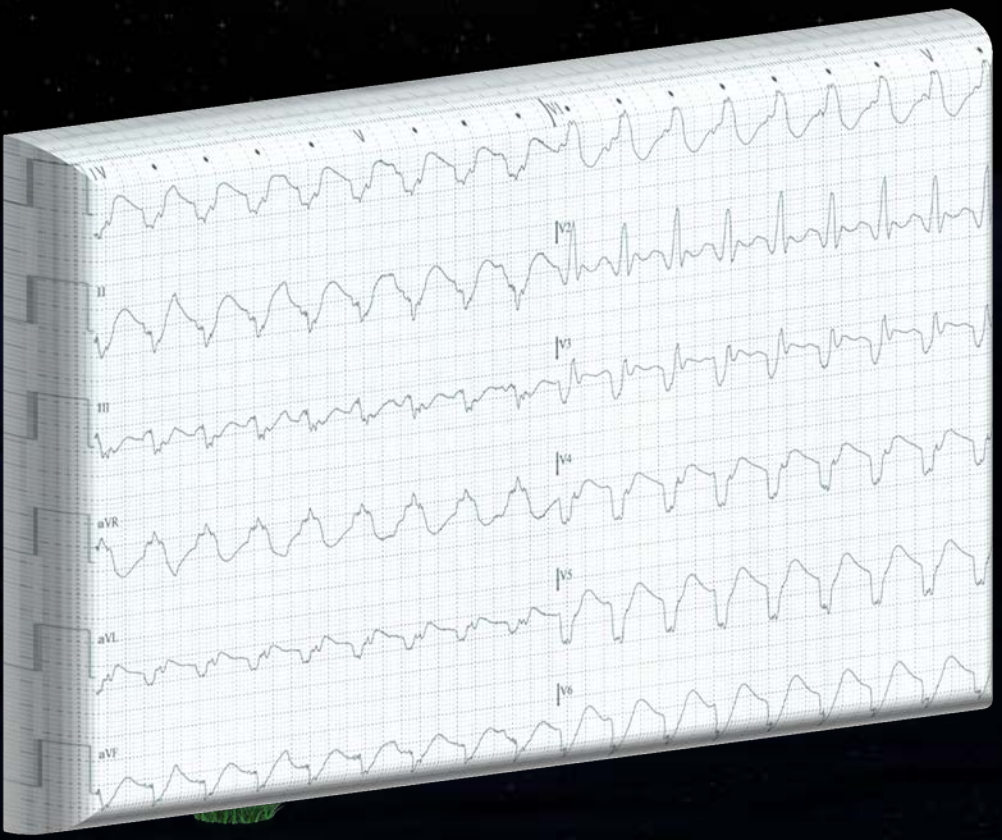


謝您的注意

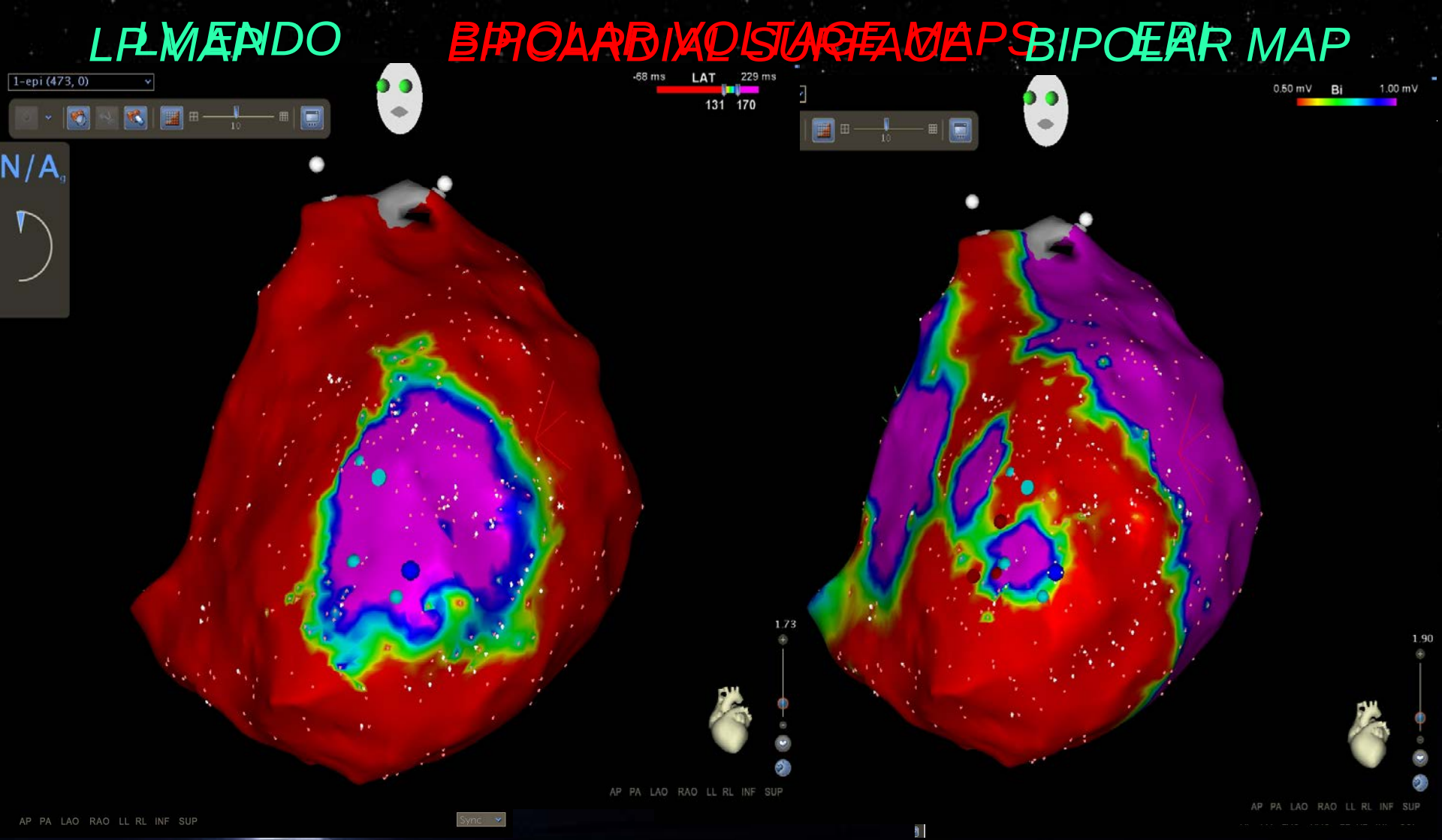
BASAL ECG



Echo: LVEF 20%, extensive scar of the inferior IV septum and inferior wall, hypokinesis of the other walls,

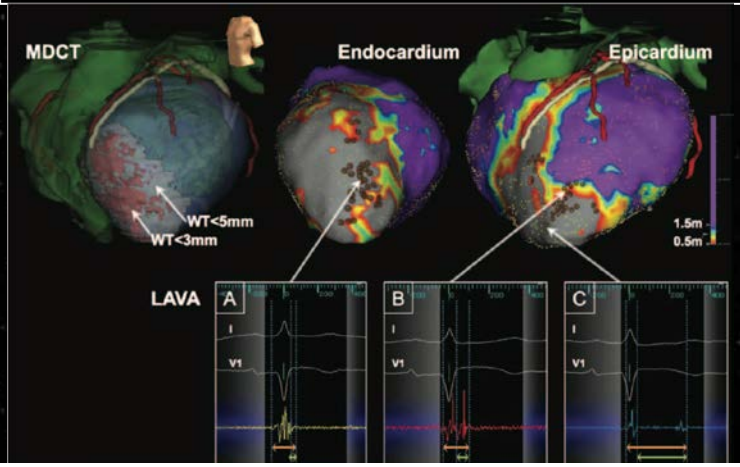


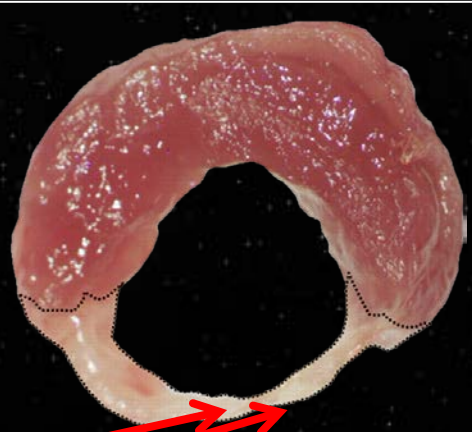
CLINICAL VT


LAT MAP
BIPOLAR VOLTAGE MAPS
EPICARDIAL SURFACE
BIPOLAR MAP
1-epi (473, 0)
-68 ms
LAT
229 ms
131
170
0.50 mV
Bi
1.00 mV
N/A
1.73
1.90
AP PA LAO RAO LL RL INF SUP
Sync


I
II
III
aVR
aVL
aVF
ABL d
ABL p
V1
V2
V3
V4
V5
V6
RVa d
P1 ART
R
F
...
...
...
1000 ms
10.59.54
10.59.56
10.59.58
11.00.00
11.00.02
ISR - MILANO

Komatsu, Y.,. Regional Myocardial Wall Thinning at Multidetector Computed Tomography Correlates to Arrhythmogenic Substrate in Postinfarction Ventricular Tachycardia: Assessment of Structural and Electrical Substrate. Circulation: Arrhythmia and Electrophysiology 2013





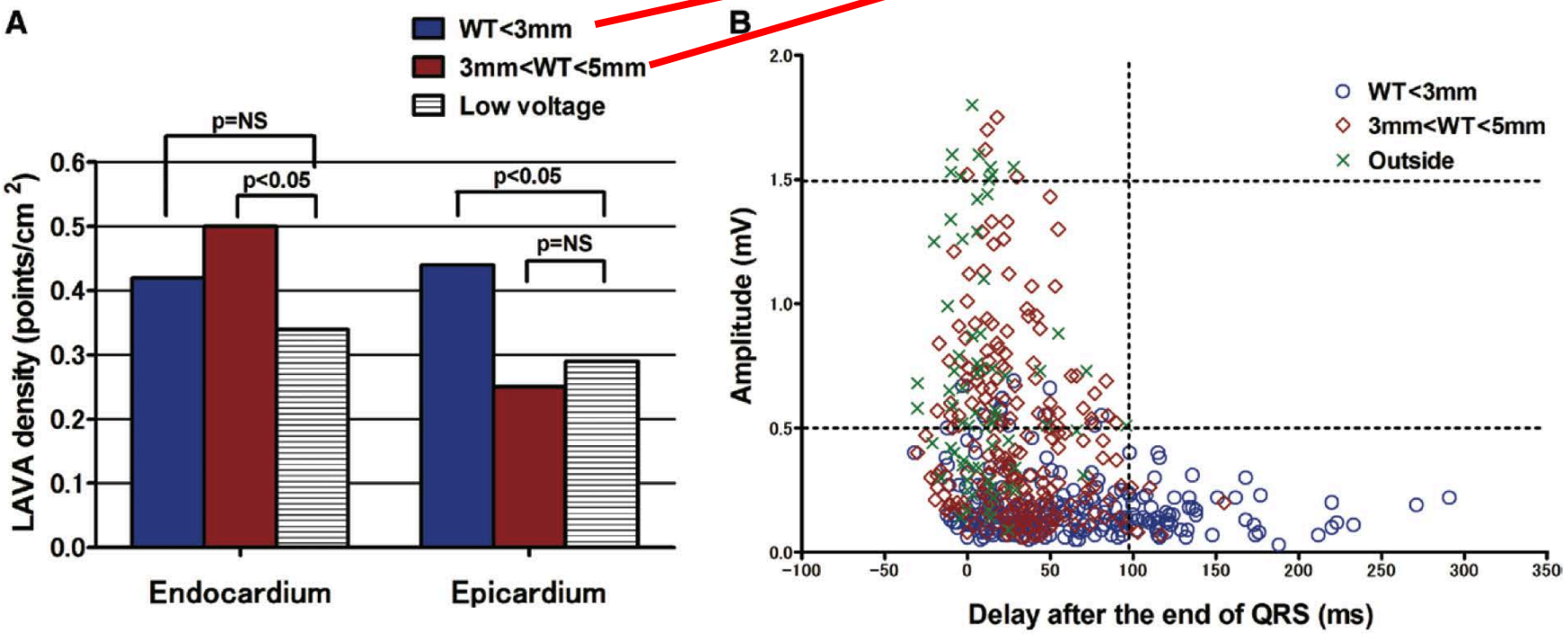


Figure 5. Distribution and characteristics of local abnormal ventricular activities (LAVA). The LAVA density in the low-voltage area was