2019.11.3 11:10-11:20
10:40~11:40 HCM and VT VF

VT ablation in Hypertrophic Cardiomyopahty

Asan Medical Center

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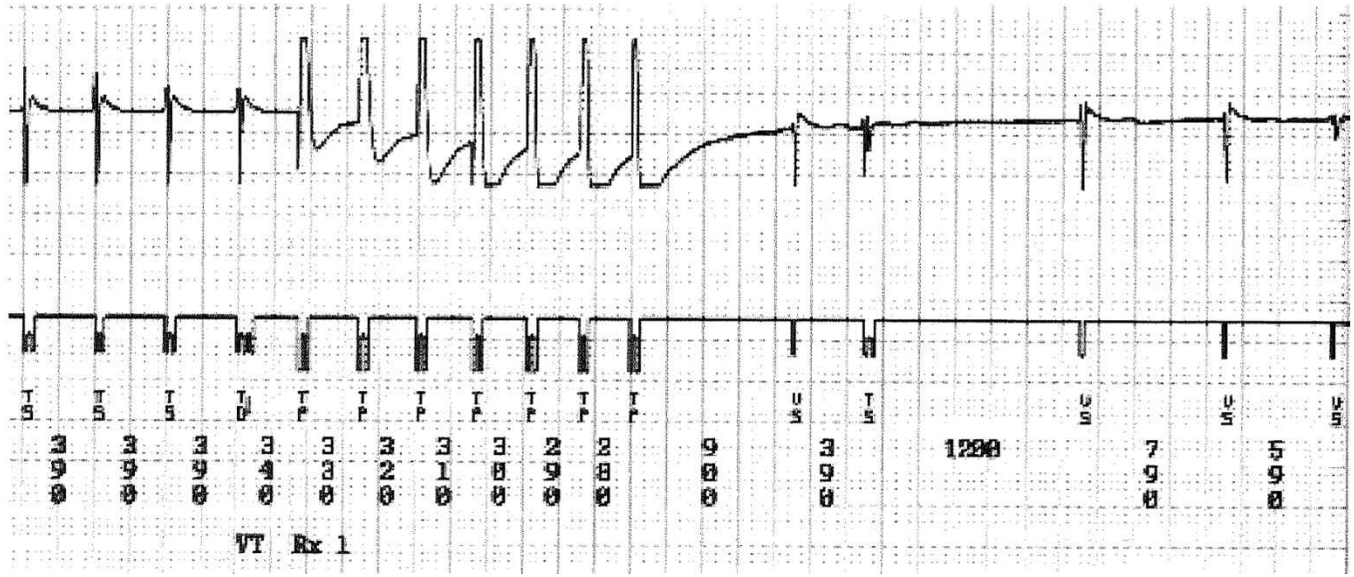
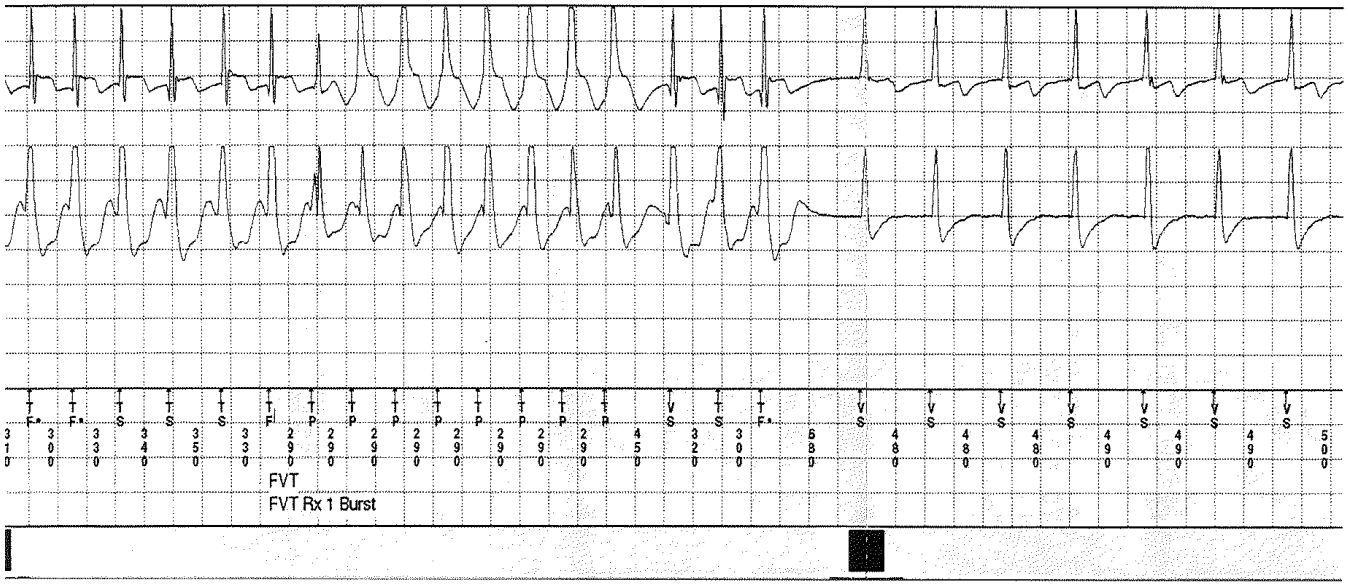
서울아산병원
Asan Medical Center

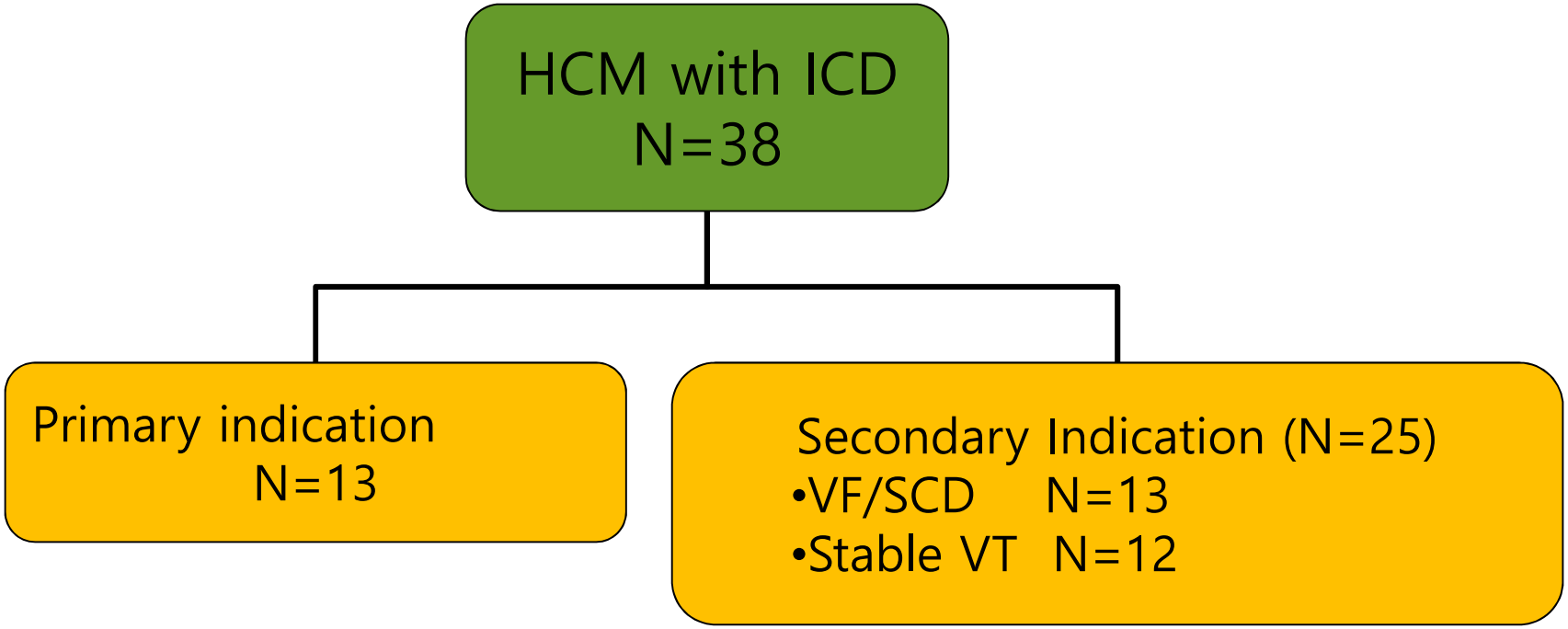




Changhoon Lee and GB Nam
2012 HRS

Methods

- From April 1996 to November 2010
- A total of 234 patients with ICDs in AMC



Changhoon Lee and GB Nam
2012 HRS

Ventricular tachyarrhythmia episodes and ICD discharge

	Shock and ATP (n=18)	Shock only (n=20)
VT	9	13
VF or SCD	9	7
ATP	155 (success in 148, fail in 6)	
Appropriate shock	6	53

Changhoon Lee and GB Nam
2012 HRS

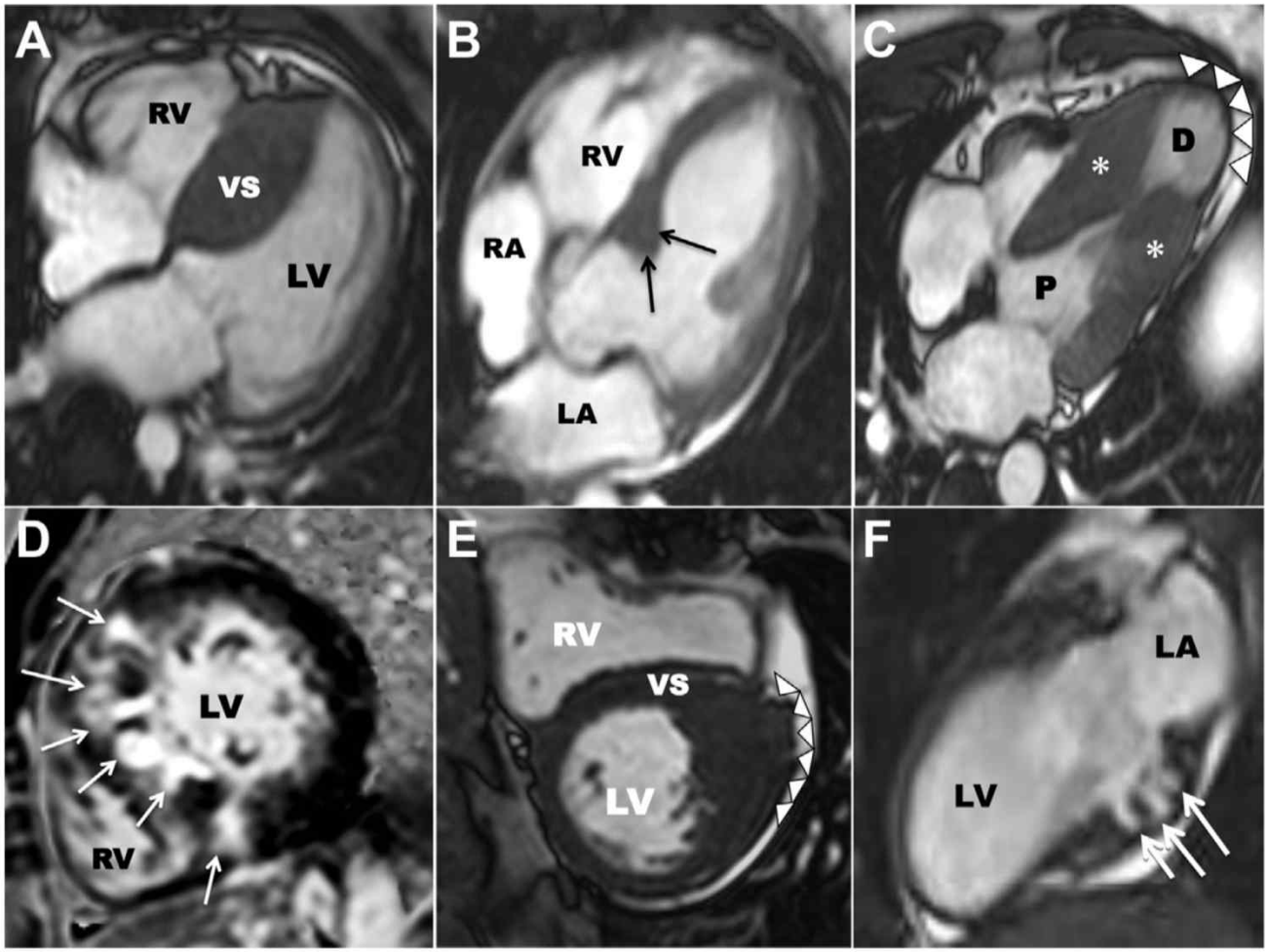
Conclusion

- **VT is the major mechanism of tachycardia in patients with HCM and SCD or in patients without previously documented VTs**
- **An empirical programming of ICDs for ATP therapy could successfully terminate most VT episodes and decrease the number of unnecessary ICD shocks**

Changhoon Lee and GB Nam
2012 HRS

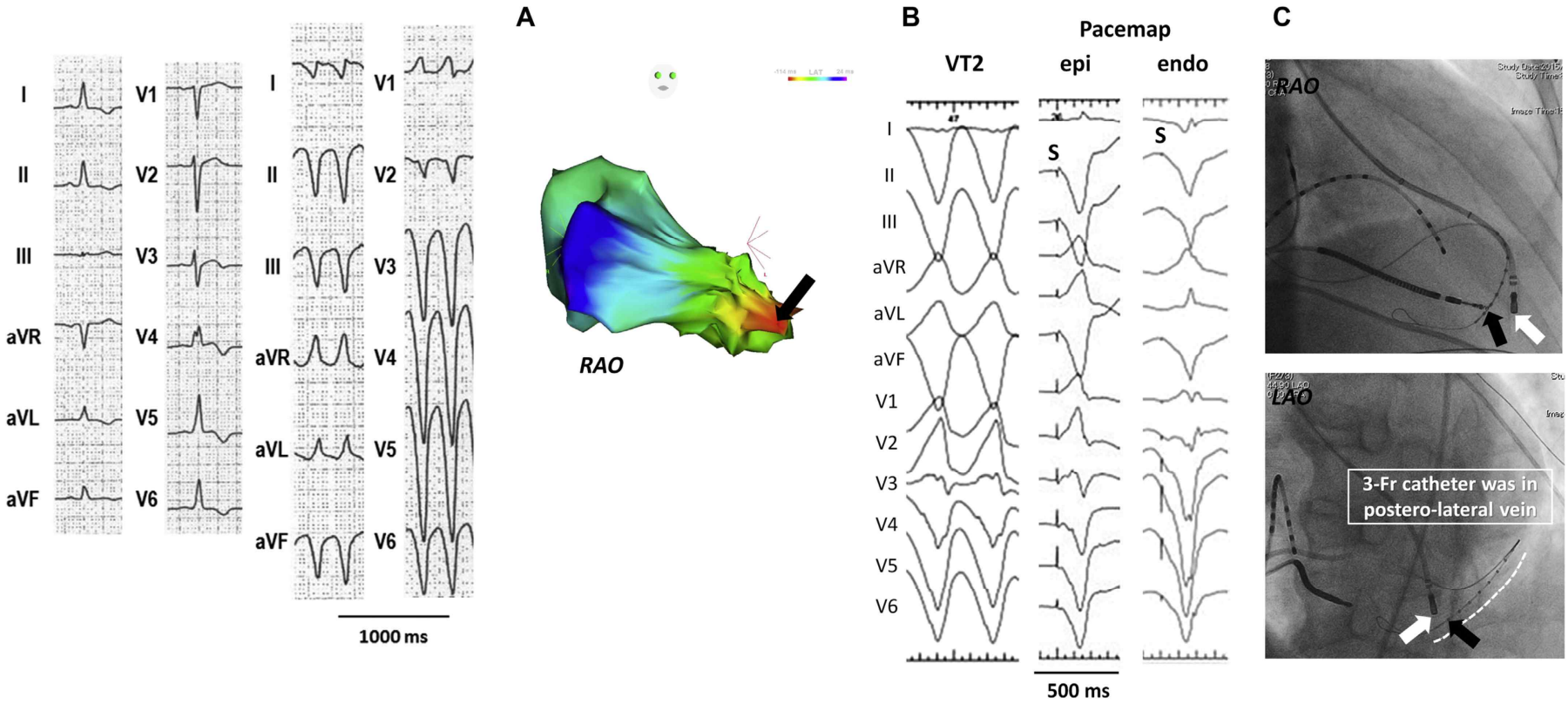
Cardiovascular MR Images Demonstrate Diversity of the HCM

Patchy scars and apical aneurysm
Diffuse cardiomyosite disarray, interstitial fibrosis



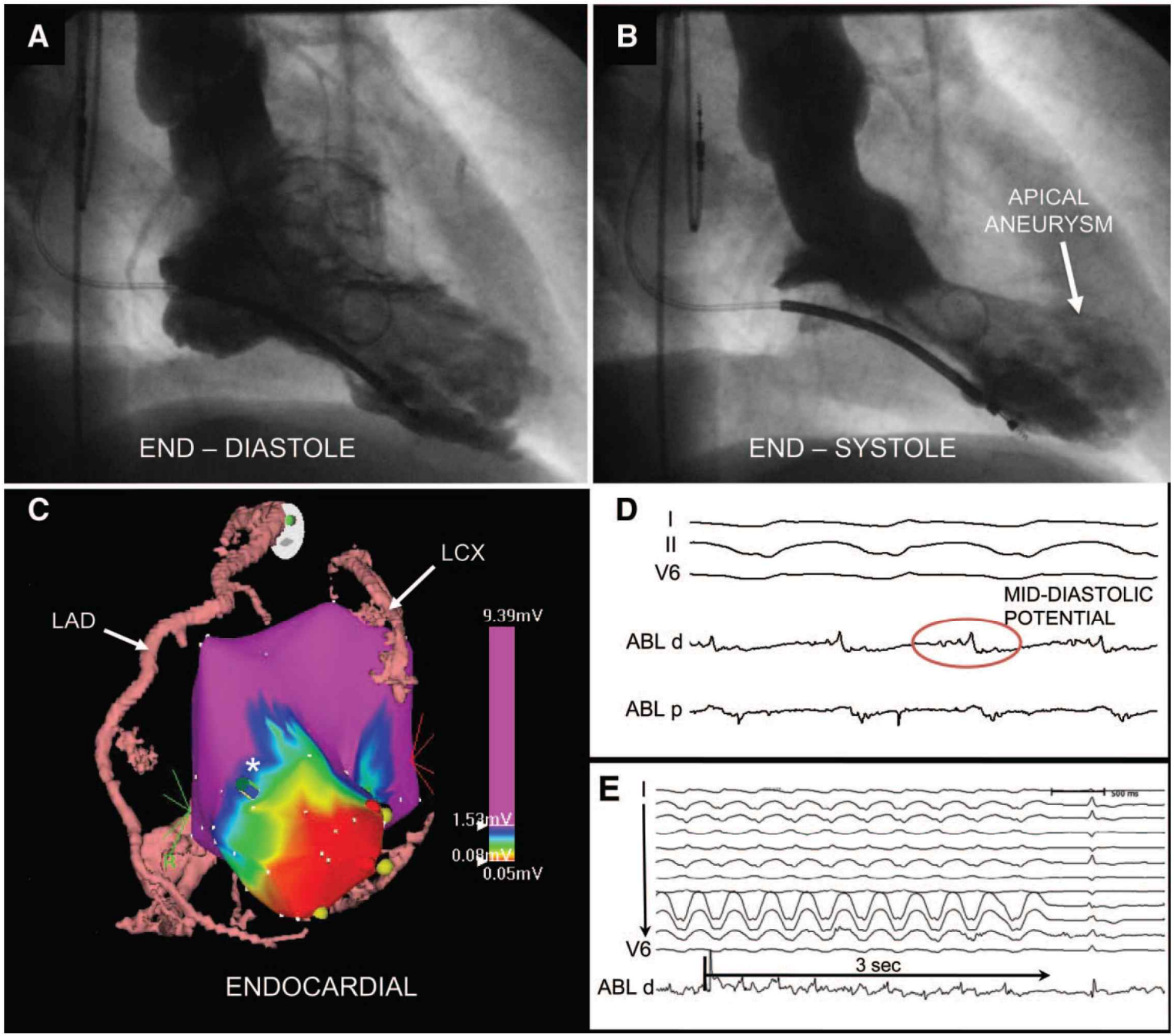
J Am Coll Cardiol 2014;64:83–99

RF Ablation of VT in Pts with HCM and Apical Aneurysm



CONCLUSIONS: In patients with HCM and AA, endocardial RFCA of AA effectively suppressed monomorphic VT which was related to AA and resulted in satisfactory outcomes.

J Am Coll Cardiol EP 2018;4:339–50

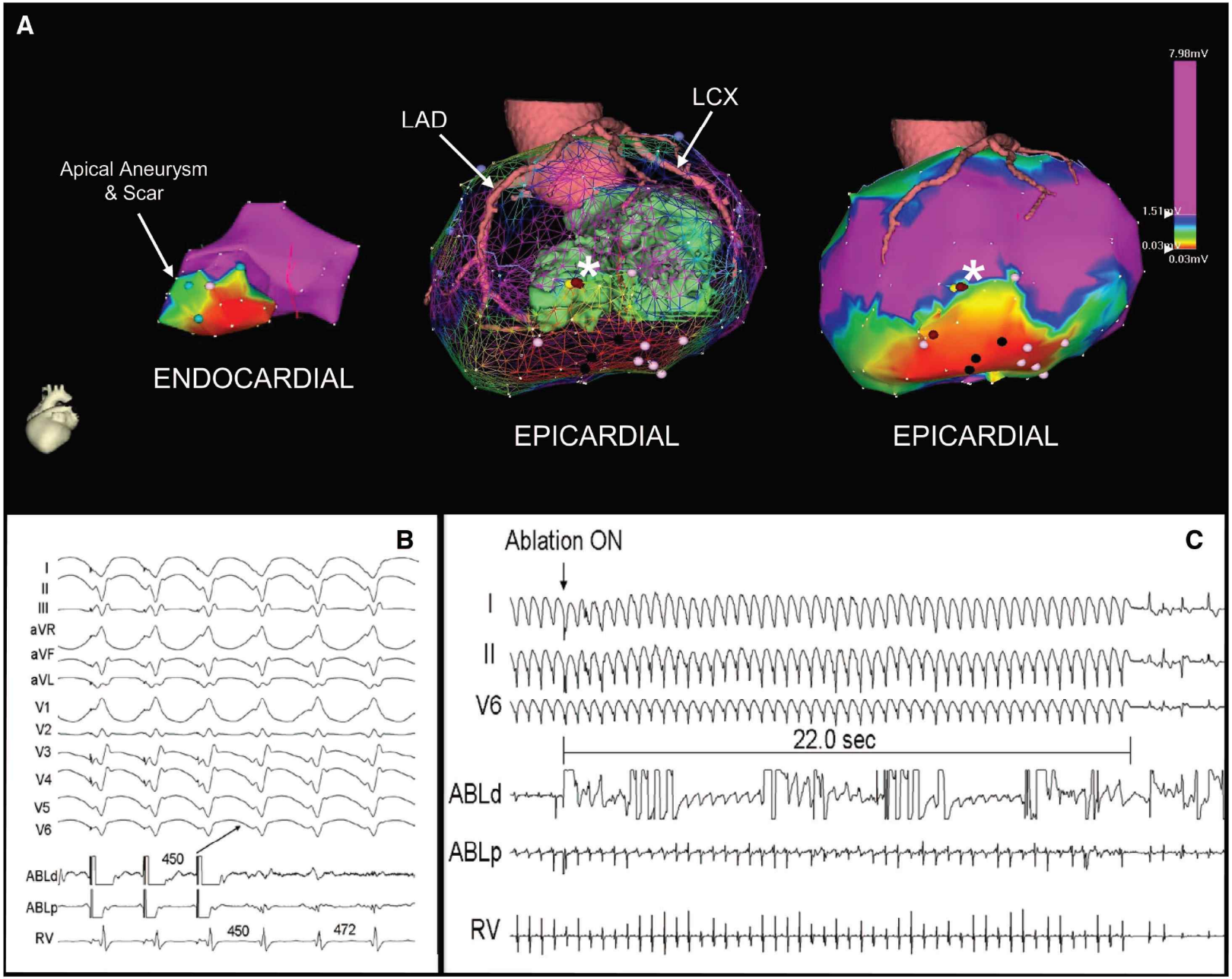

A
END – DIASTOLE
B
APICAL
ANEURYSM
END – SYSTOLE
C
LCX
LAD
9.39mV
1.53mV
0.08mV
0.05mV
ENDOCARDIAL
D
I
II
V6
MID-DIASTOLIC
POTENTIAL
ABL d
ABL p
E
I
500 ms
V6
3 sec
ABL d

Long-Term Outcomes of Combined Epi-Endocardial Ablation of Monomorphic VT Related to HCM

Table 2. Two-Dimensional Echocardiogram Findings of Patients With HCM-Related VT

Pt. No.	LVEF, %	LVEDD, mm	LVESD, mm	IVS, mm	PW, mm	Findings
1	55	52	31	11	11	Focal midventricular and apical hypertrophy; midcavitary obstruction with gradient=25 mm Hg; apical aneurysm
2	68	57	37	8	12	Apical hypertrophy; midcavitary systolic gradient=11 mm Hg
3	57	47	27	16	12	Asymmetric hypertrophy; midseptum=26 mm; mid-PW=25 mm; midcavitary obliteration with gradient of 45 mm Hg after Valsalva maneuver; apex akinetic/aneurysmal
4	63	51	31	14	11	Asymmetric LV hypertrophy without gradient; no wall motion abnormalities
5	40	55	41	15	17	Asymmetric LV hypertrophy; lateral wall hypokinesis
6	83	42	22	24	12	Asymmetric septal hypertrophy; midcavitary obliteration with gradient=23 mm Hg
7	48	48	33	12	12	Apical akinesis/aneurysmal dilatation; organized apical thrombus; midcavitary obliteration
8	43	56	43	16	8	Asymmetric septal hypertrophy and LV dilatation; inferior wall and inferior/midseptal hypokinesis and akinesis from LV base to apex
9	45	45	33	26	15	Asymmetric septal hypertrophy without obstruction; apical and anteroapical hypokinesis
10	65	31	20	24	17	Severe asymmetric septal hypertrophy; no LV outflow tract obstruction

Circ Arrhythm Electrophysiol. 2011;4:185-194


A
Apical Aneurysm
& Scar
ENDOCARDIAL
LAD
LCX
EPICARDIAL
7.98mV
1.51mV
0.03mV
0.03mV
EPICARDIAL
B
I
II
III
aVR
aVF
aVL
V1
V2
V3
V4
V5
V6
450
ABLd
ABLp
RV
450
472
C
Ablation ON
I
II
V6
22.0 sec
ABLd
ABLp
RV

Long-Term Outcomes of Combined Epi-Endocardial Ablation of Monomorphic VT Related to HCM

Pt. No.	Arrhythmia Induced	No. MMVTs	VT Cycle Length, ms	Scar Location	Entrainment	Activation Mapping	Termination During Ablation	Termination Site	Termination Time, s	Ablation Sites	Irrigated Catheter	Postablation VT Induced
1	…	0	…	Endo (LV apex) Epi (LV apex, lateral)	No	No	…	…	…	Endo Epi	No	No
2	PMVT	0	…	Endo (LV apex) Epi (LV apex)	No	No	…	…	…	Endo Epi	No	No
3	MMVT	1	472	Endo (LV apex) Epi (LV lateral, apex)	Yes	Yes	Yes	Epi	22	Endo Epi	Yes	No
4	MMVT	1	270	Epi (LV lateral base)	Yes	Yes	Yes	Epi	3	Epi	No	No
5	MMVT	2	VT1-370, VT2-280 (RVOT)	Epi (LV anterolateral base)	No	Yes	Yes–VT1	Epi	30	Epi	No	Yes
6	…	0	…	None	…	…	…	…	…	…	…	…
7	MMVT	3	VT1-330, VT2-310, VT3-300	Endo (LV apex) Yes Epi (LV apex)	Yes	Yes–VT1	Endo-reinducible	Endo-12 Epi	Endo Epi-3	Yes Epi	No	
8	MMVT	5	VT1-305, VT2-305, VT3-310, VT4-330, VT5-350	Endo (LV anteroapex)	Yes	No	Yes-VT1	Endo	7	Endo	Yes	No
9	MMVT, nonsustained	1	480	Endo (LV anterolateral, apex) Epi (LV anterolateral, apex)	No	No	…	…	…	Endo Epi	Yes	No
10	MMVT	1	280	Epi (LV apex, basal inferior)	No	No	…	…	…	Epi	Yes	No

Endo indicates endocardial; Epi, epicardial; MMVT, monomorphic VT; NSVT, nonsustained VT; PMVT, polymorphic VT.

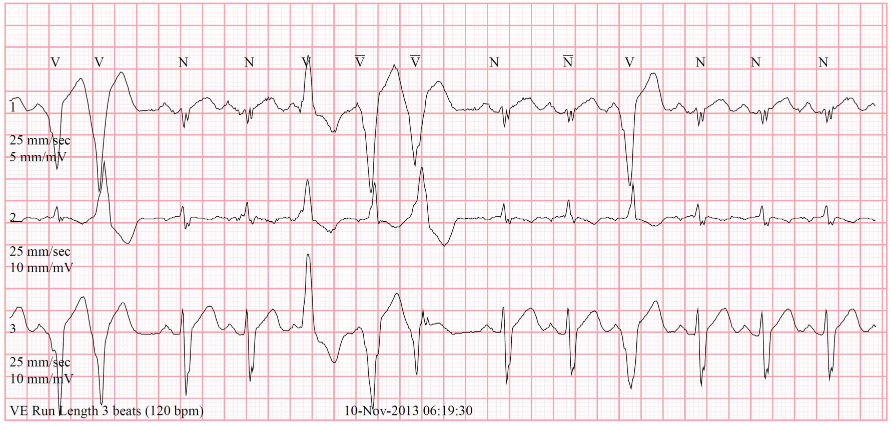
Electrophysiological-identified epicardial scar was present in 8 (80%) patients, endocardial scar in 6 (60%), and no scar in 1 (10%).

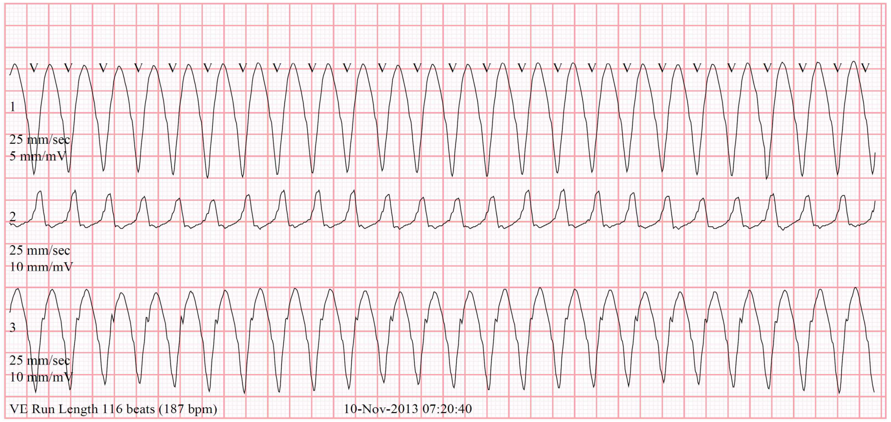
Circ Arrhythm Electrophysiol. 2011;4:185-194

M/42

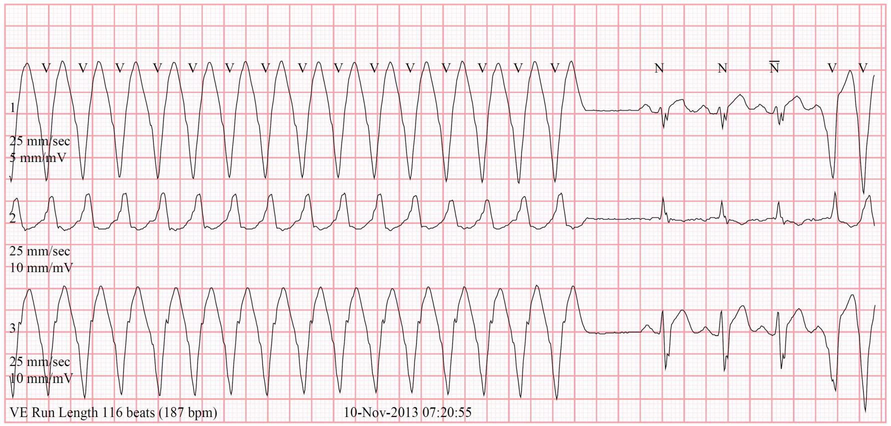
2009. Aug Dx with HCM at outside hospital
2013.10 palpitation
Holter – nonsustained, sustained VT (for 35sec)

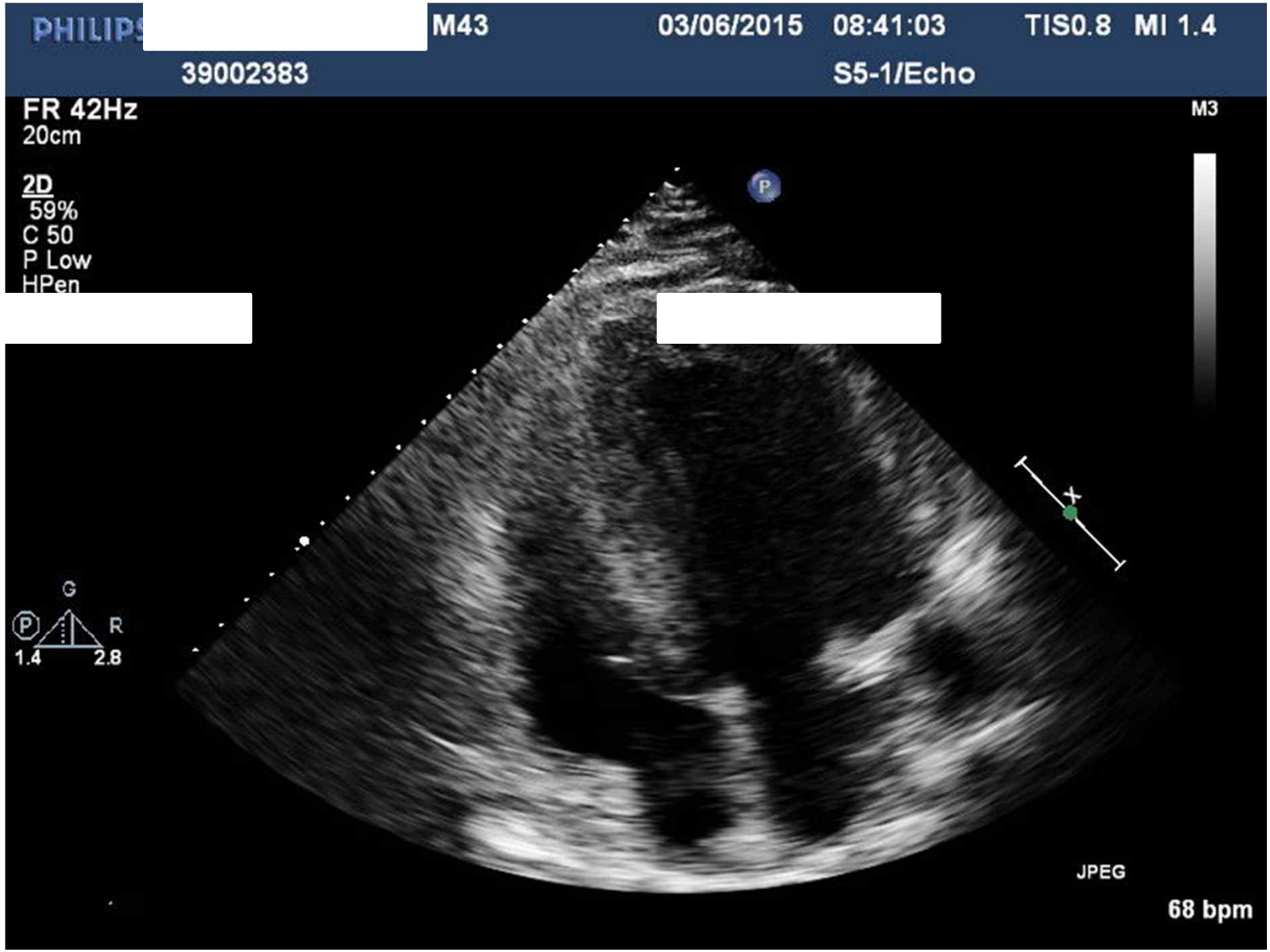
MRI
1. Hypertrophy of myocardium,
particularly in interventricular septum resulting in LVOTO with SAM.
--> HCMP, likely.
2. Multifocal patchy enhancement in apical anterior, apical inferior, mid-to-basal anterior septal, mid-to-basal inferior septal walls, suggestive of myocardial fibrosis.
3. MRI 상 moderate to severe Mitral regurgitation (regurgitation fraction 30% ~ 35%)


1
25 mm/sec
5 mm/mV
2
25 mm/sec
10 mm/mV
3
25 mm/sec
10 mm/mV
VE Run Length 3 beats (120 bpm)
10-Nov-2013 06:19:30

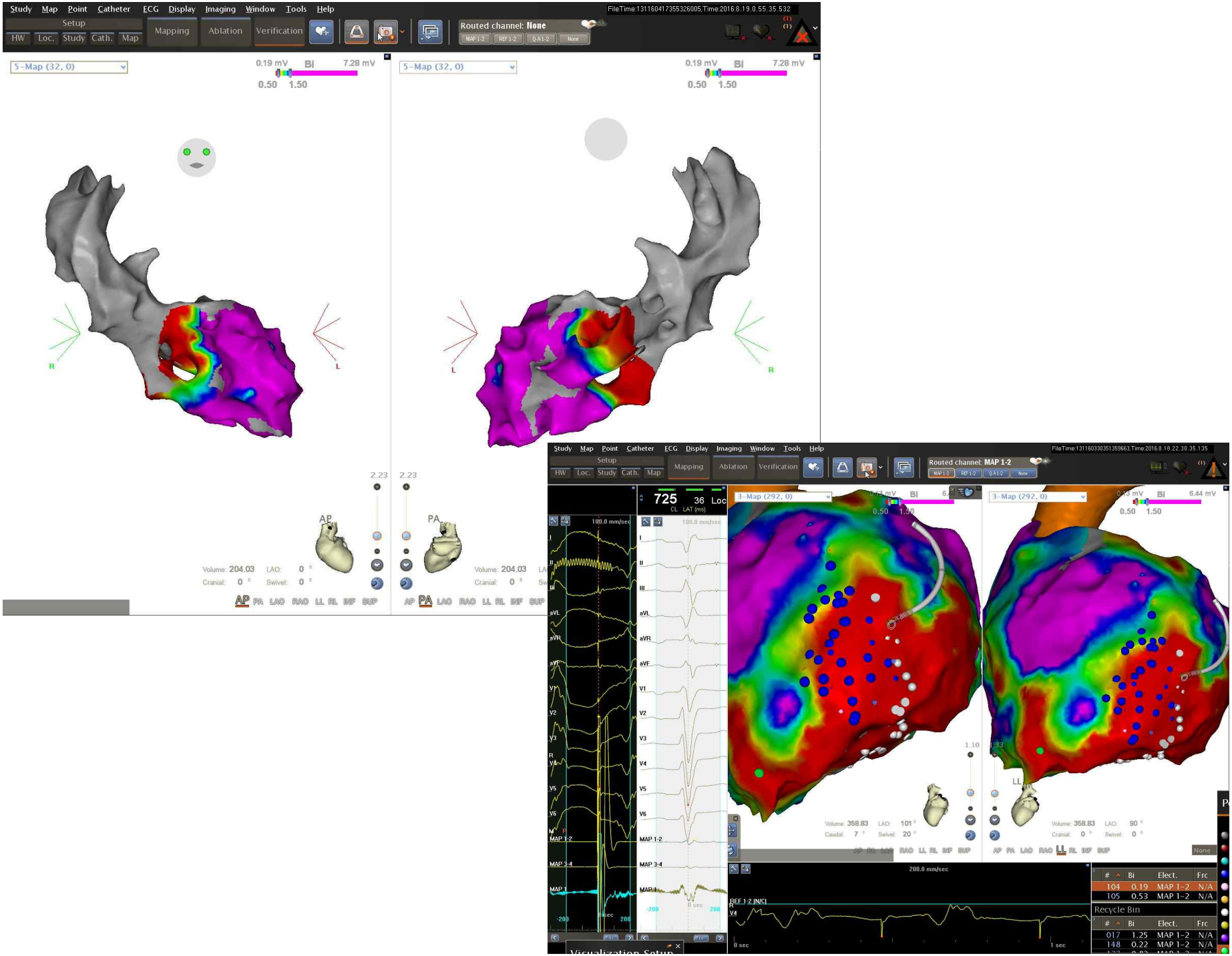

1
25 mm/sec
5 mm/mV
2
25 mm/sec
10 mm/mV
3
25 mm/sec
10 mm/mV
VE Run Length 116 beats (187 bpm)
10-Nov-2013 07:20:40


1
25 mm/sec
5 mm/mV
2
25 mm/sec
10 mm/mV
3
25 mm/sec
10 mm/mV
VE Run Length 116 beats (187 bpm)
10-Nov-2013 07:20:20

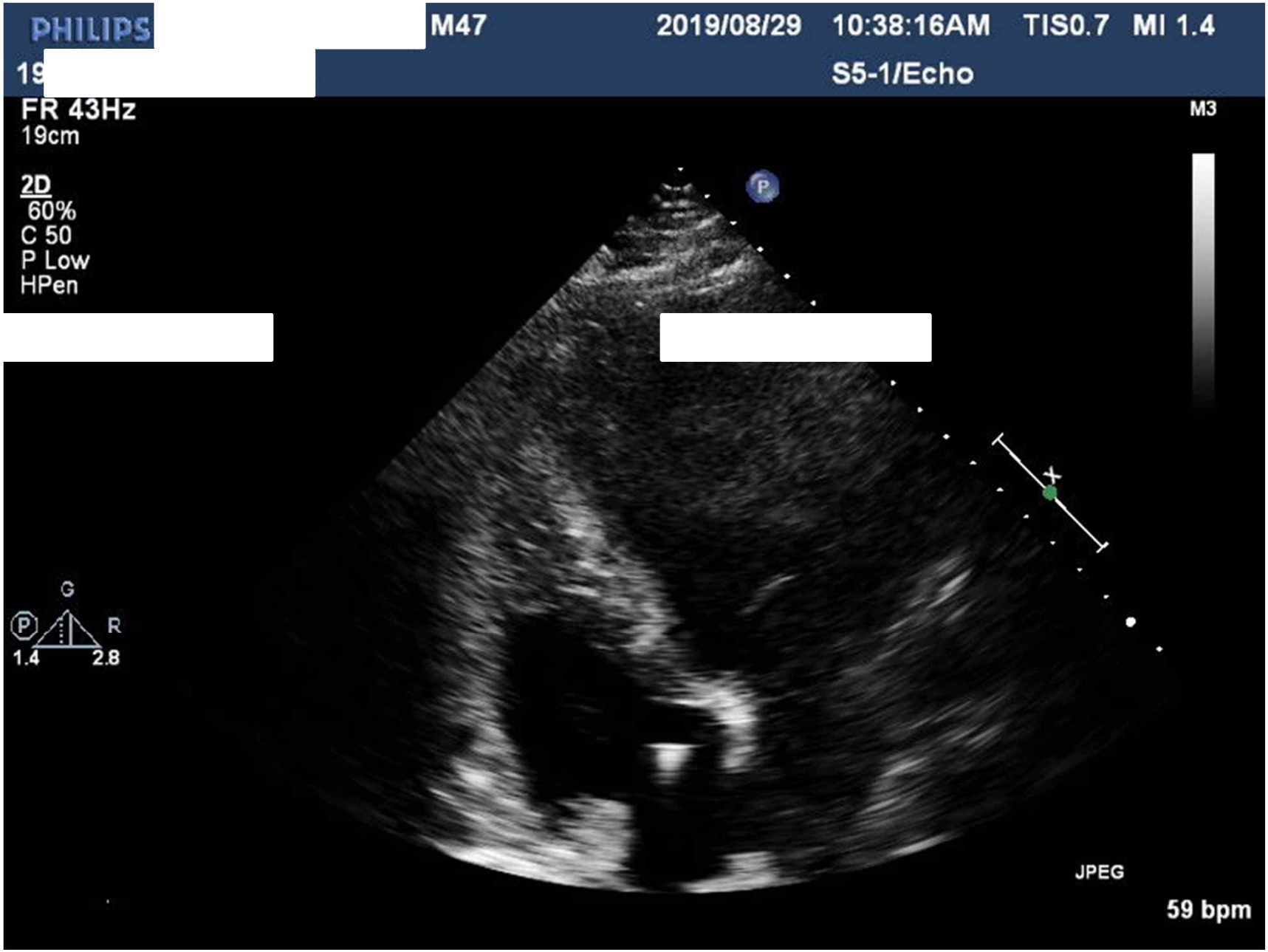

1
25 mm/sec
5 mm/mV
2
25 mm/sec
10 mm/mV
3
25 mm/sec
10 mm/mV
VE Run Length 116 beats (187 bpm)
10-Nov-2013 07:20:55


PHILIPS
M43
03/06/2015
08:41:03
TIS0.8
MI 1.4
39002383
S5-1/Echo
FR 42Hz
20cm
M3
2D
59%
C 50
P Low
HPen
G
R
1.4
2.8
JPEG
68 bpm

		EchoCG	shock	ATP
2013.12	**ICD implantation**			
2014.1.3	VT 1 VF 3	LV36/54, EF 54%	2	1
2014.7.11	Fast VT 2			2
2014.08.07	VT 2 VF 2		1	3
2015.03.02	Fast VT 3		2	1
2015.03.26	Fast VT 8		6	2
2015.04.10	VT 3		2	
2015.09.07	-			
2015.12.23	-	LV 45/60, EF 37% Mild (Gr 1) MR		
2016.06.17	VT 18		18	
2016.08.14	VT 5		5	

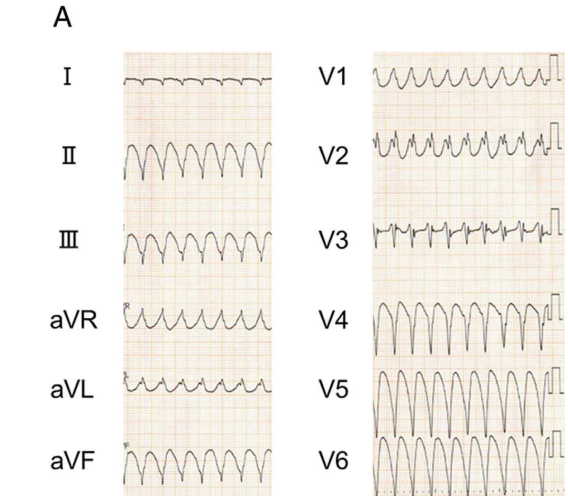

Study Map Point Catheter ECG Display Imaging Window Tools Help
Setup
HW Loc. Study Cath. Map
Mapping
Ablation
Verification
Routed channel: None
5-Map (32, 0)
0.19 mV BI 7.28 mV
0.50 1.50
AP
PA
Volume: 204.03
LAO: 0
Cranial: 0
Swivel: 0
AP PA LAO RAO LL RL INF SUP
Routed channel: MAP 1-2
3-Map (292, 0)
0.13 mV BI 6.44 mV
725
36 Loc
CL LAT (ms)
Volume: 358.83
LAO: 101
Caudal: 7
Swivel: 20
Volume: 358.83
LAO: 90
Cranial: 0
Swivel: 0
200.0 mm/sec
REF 1-2 (NC)
0 sec
1 sec
Recycle Bin
Bi Elect. Frc
104 0.19 MAP 1-2 N/A
105 0.53 MAP 1-2 N/A
017 1.25 MAP 1-2 N/A
148 0.22 MAP 1-2 N/A

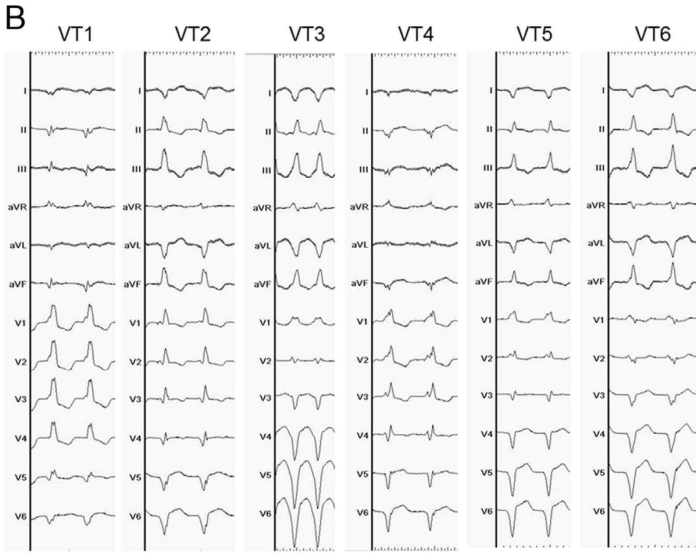
		EchoCG	shock	ATP
2013.12	**ICD implantation**			
2014.1.3	VT 1 VF 3	LV36/54, EF 54%	2	1
2014.7.11	Fast VT 2			2
2014.08.07	VT 2 VF 2		1	3
2015.03.02	Fast VT 3		2	1
2015.03.26	Fast VT 8		6	2
2015.04.10	VT 3		2	
2015.09.07	-			
2015.12.23	-	LV 45/60, EF 37% Mild (Gr 1) MR		
2016.06.17	VT 18		18	
2016.08.14	VT 5		5	
2016.08.18	**RFCA**			
2017.04.25	-			
2017.07.11	-			
2017.12.01	-			
2018.03.13	-			
2018.06.19	-	LV 49/62, EF 37% moderate MR		
2018.10.26	-			
2019.01.22	VT 2		1	
2019.8. 29	Generator, replaced	LV 56/65, EF 33% Prolapse of P3, moderate MR		

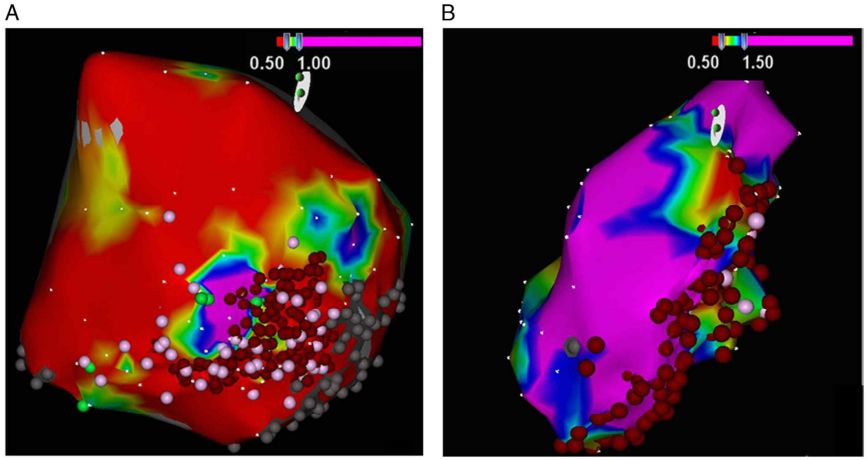

PHILIPS
M47
2019/08/29 10:38:16AM TIS0.7 MI 1.4
S5-1/Echo
FR 43Hz
19cm
2D
60%
C 50
P Low
HPen
M3
G
R
1.4
2.8
JPEG
59 bpm

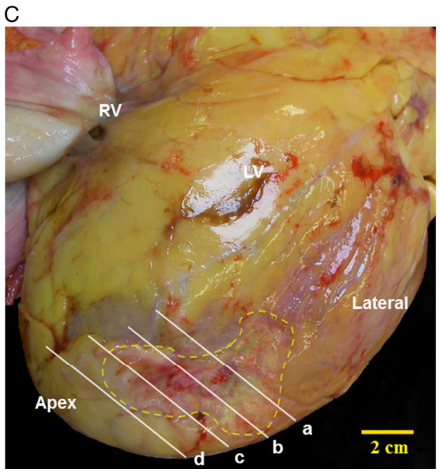
Pathology after combined epi, endocardial ablation for VT in a postmortem heart with HCM

The VT was clinically controlled by antiarrhythmic drugs (amiodarone, nifekalant, and lidocaine). The patient, however, died from deterioration of heart failure 49 days after the procedure.





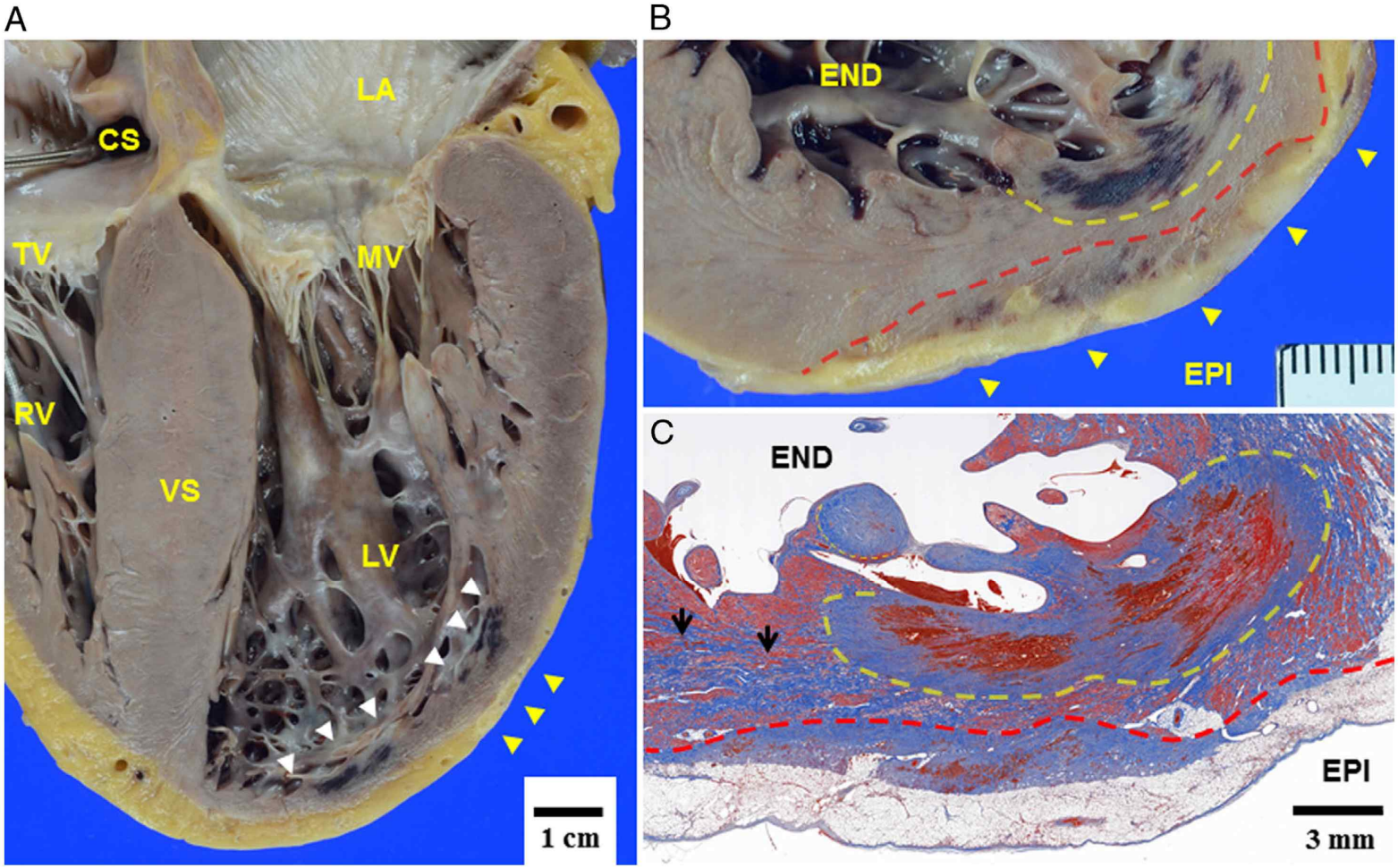




M/73

(Heart RhythmCaseReports2015;1:310–314)

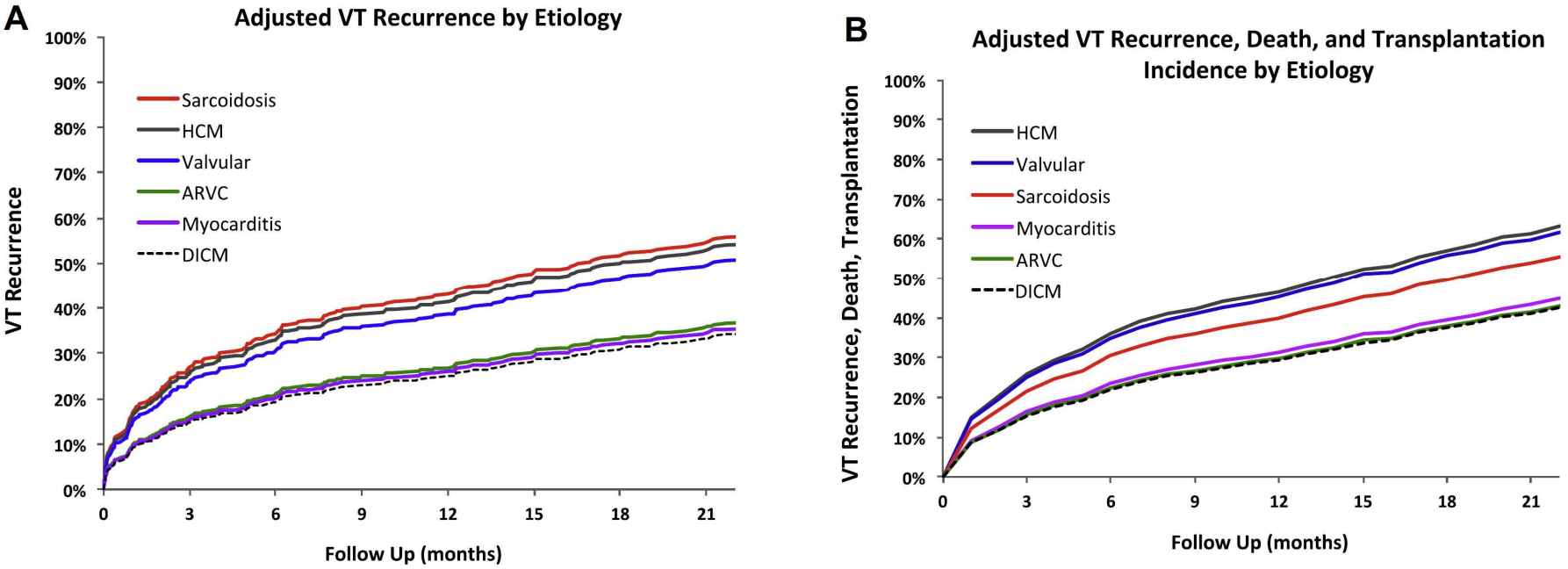
Yellow arrowheads indicate ablated lesions in epicardialfat,whichisapproximately2–4 mm thick. The yellow dotted line represents an ablation lesion from the endocardium. The red line represents an ablation lesion from the epicardium, and the ablation scar reached a maximum depth of 6 mm.



This pathologic report showed the limitation of the epicardial RFCA to deliver sufficient RF energy to the myocardium beyond the epicardial adipose tissue, which resulted in residual arrhythmogenic substrate even after combined epicardial and endocardial RFCA.

(Heart RhythmCaseReports2015;1:310–314)

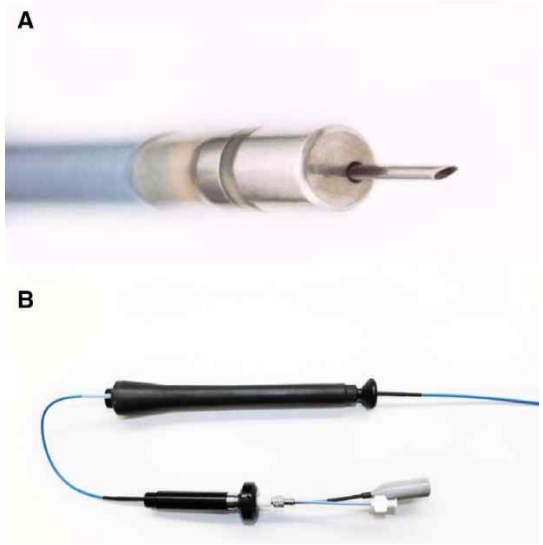
Outcomes of Catheter Ablation of VT based on Etiology in Nonischemic Heart Disease

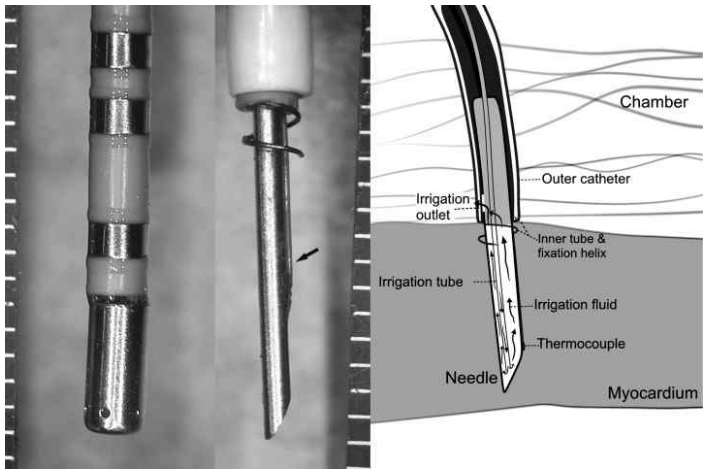


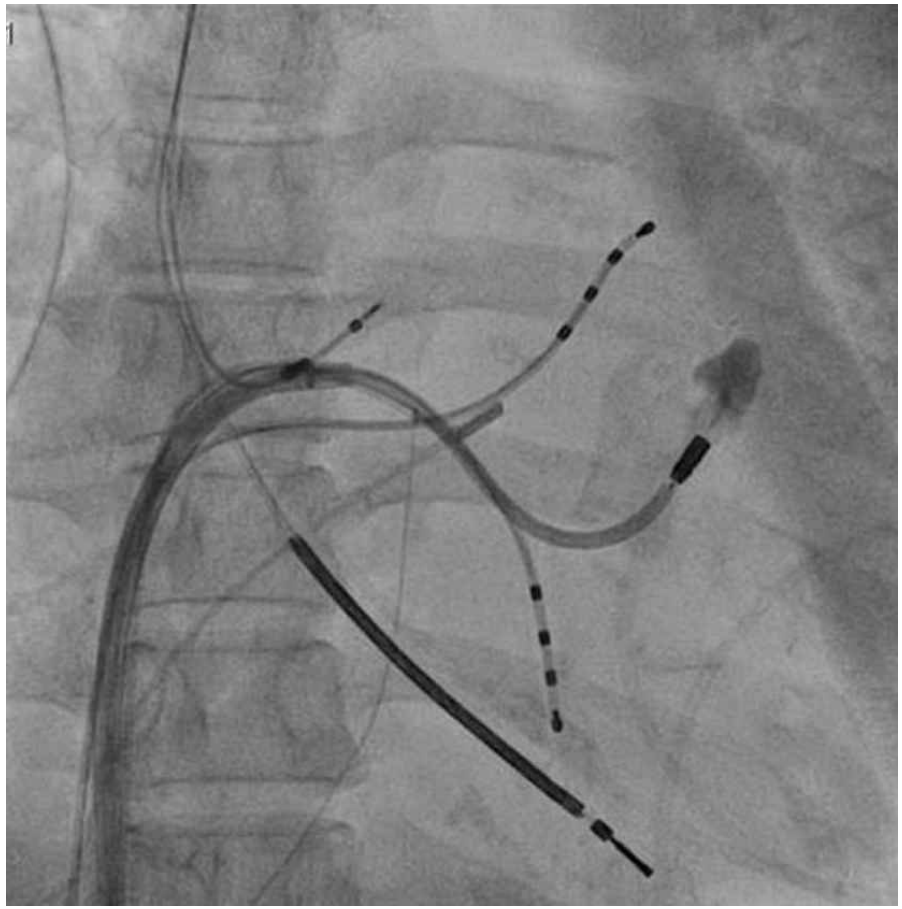
CONCLUSIONS Catheter ablation of VT in NICM is effective. Etiology of NICM is a significant predictor of outcomes, with ARVC, myocarditis, and DICM having similar but superior outcomes to hypertrophic cardiomyopathy, valvular cardiomyopathy, and sarcoidosis, after adjusting for potential covariates.

(J Am Coll Cardiol EP 2018;4:1141–50)

Infusion Needle Catheter Ablation

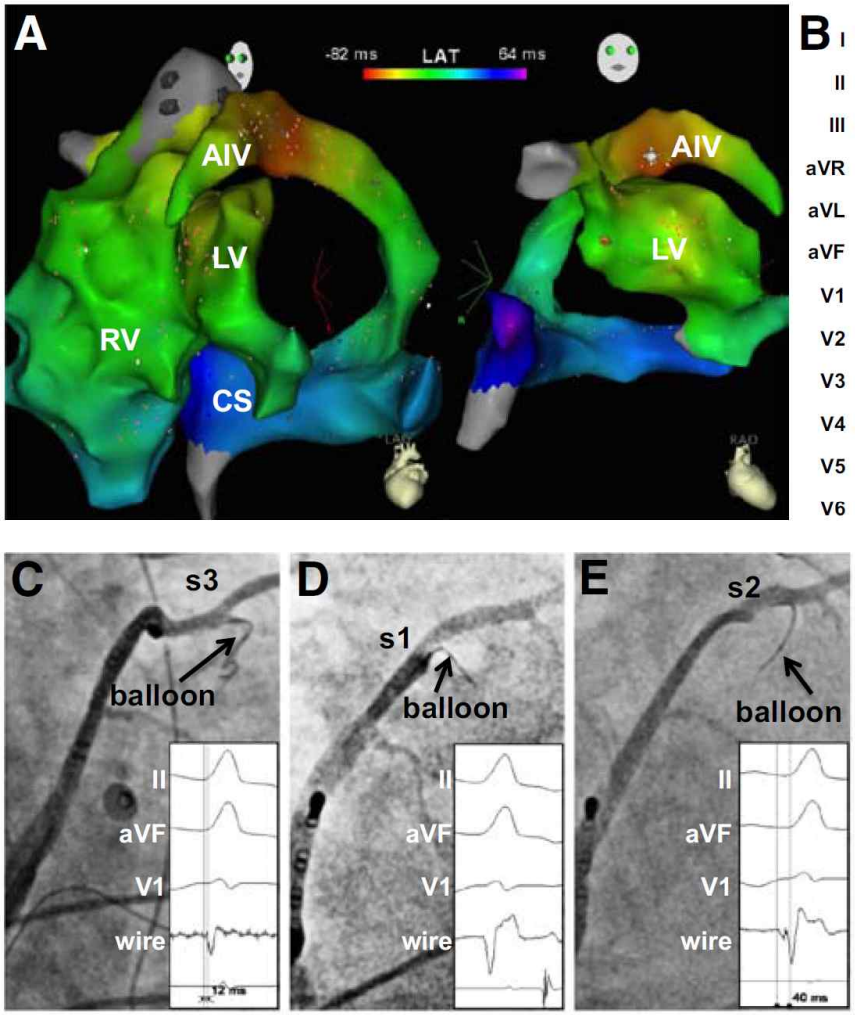






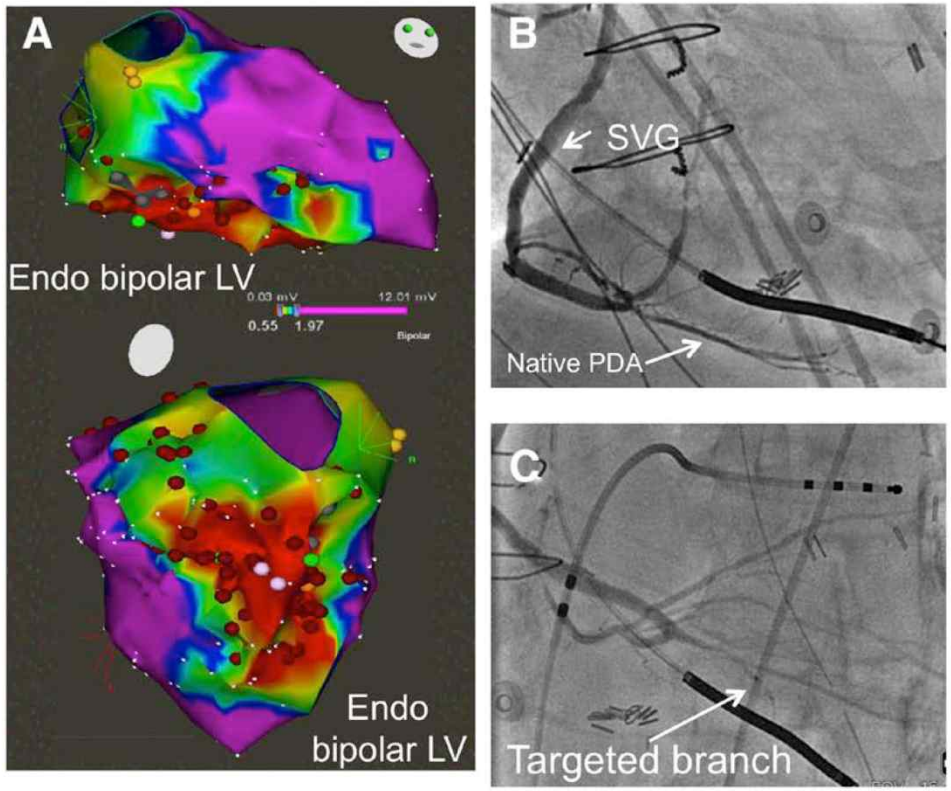
Circulation. 2013;128:2289–2295

Retrograde Coronary Venous Ethanol Infusion for Refractory VT



Circ Arrhythm Electrophysiol. 2016;9:e004352

Trans-coronary ethanol ablation



Circ Arrhythm Electrophysiol. 2017;10:e003676

Conclusion

1. VT is an important cause of frequent ICD shocks even in patients who presented with VF.
2. Patchy scars and apical aneurysm may provide electrophysiolgic melieu or arrhythmogenic mechanism similar to that of ischemic VT. Endocardial ablation may effectively suppress monomorphic VT in pts w HCM and LV apical aneurysm. Target of ablation, at the junction of the aneurysm rim and LV myocardium.
3. However, VT ablation in patients with HCM is challenging d/t anatomic limitations (mid-myocardial septal, diffuse nature of cardiomyosite disarray, interstitial fibrosis), and role of RFCA awaits further study.
4. Newer devices/techiques (ethanol ablation, needle, half saline, surgical cryoablation, SBRT)