

.....

Papillary muscle PVC ablation

. ↓ ↓ . . .

Yoo Ri Kim

Dongguk University, School of Medicine, Korea









Korean Heart Rhythm Society COI Disclosure

Yoo Ri Kim:

The author is a consultant (receiving consulting fees) of InterMD company and an (unpaid) advisor of Digital Heathcare Partners (DHP)







Content

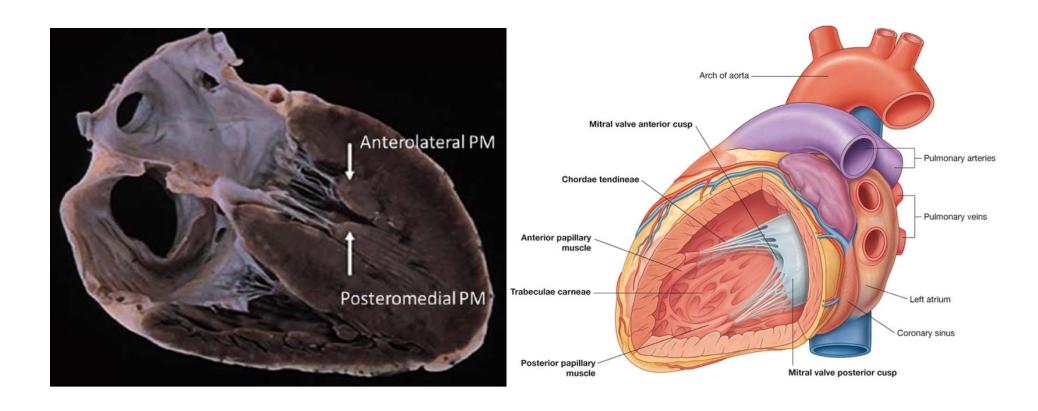
- 1. ECG Characteristics & anatomic consideration
- 2. Differential diagnosis & clinical characteristics
- 3. Treatment and prognosis
 - 1) Catheter ablation techniques
 - ① With 3D
 - ② With ICE
 - 3 With Cryo
 - 2) Practical aspect of ablation of PM VA
- 4. Summary







Anatomic consideration

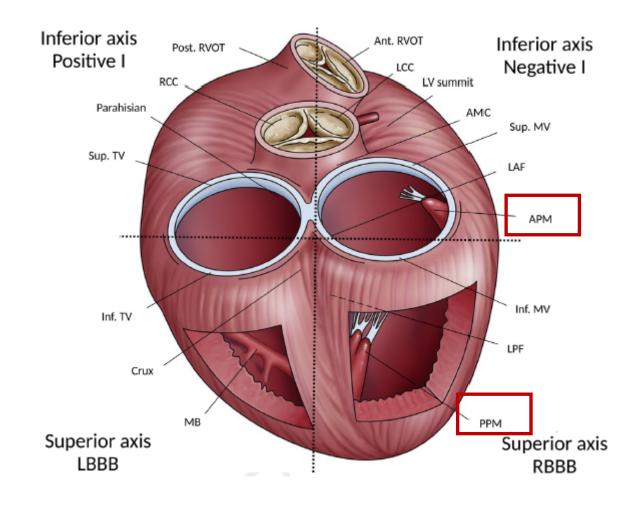








ECG characteristics & Anatomy

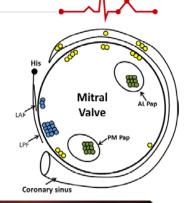


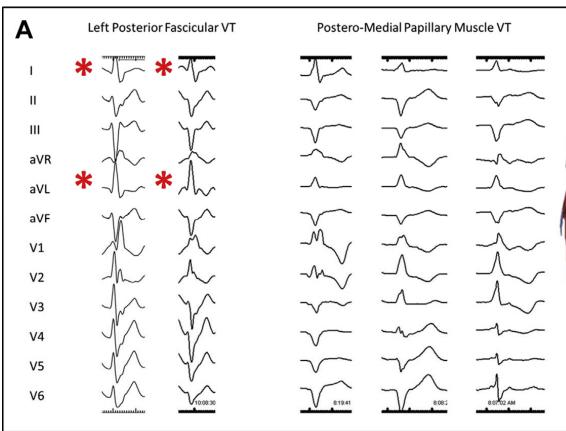


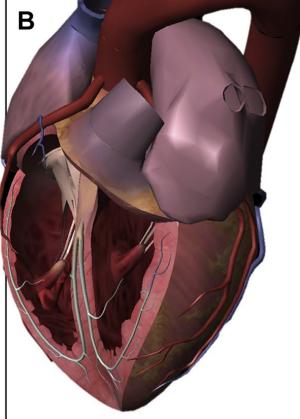


Fascicular vs. Papillary m

- no lateral q wave -





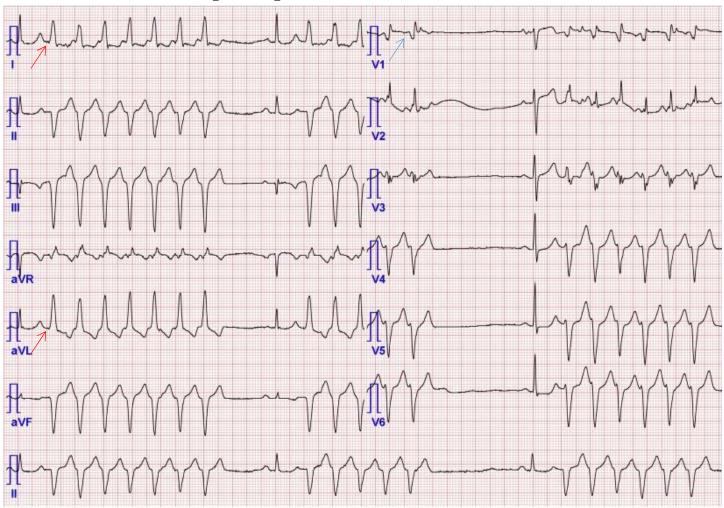








Case 1. 34/M palpitation



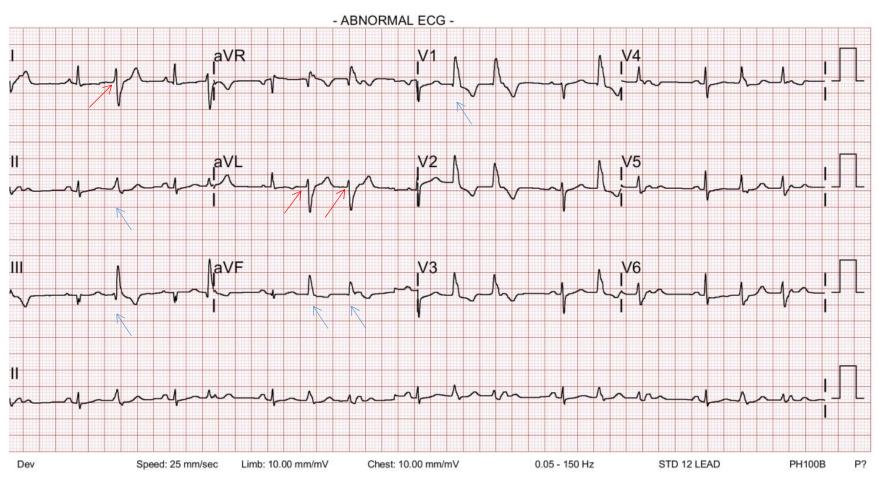
RBBB, superior axis, wider QRS, V1 qR wave, no q in lateral leads







Case2. 54/F neck pounding, presyncope



V1 qR pattern, inferior axis, deep S in V6, lateral no q wave





Clinical characteristics of PM VA

- Inducibility with exertion of epinephrine, isoproterenol
- PVCs rather than VT
- Lack of inducibility with PES (V or A stim)
- Refractoriness to verapamil and Na⁺ channel blocker
- Earliest V activation on the PM
- Challenging to ablate

Ventricular Fibrillation Triggered by PVCs from Papillary Muscles: Clinical Features and Ablation

FRANCESCO SANTORO, M.D.,*,† LUIGI DI BIASE, M.D., PH.D., F.H.R.S.,*,†,‡,§
PATRICK HRANITZKY, M.D.,†,¶ JAVIER E. SANCHEZ, M.D.,† PASQUALE SANTANGELI,
M.D.,*,† ALESSANDRO PAOLETTI PERINI, M.D.,# JOHN DAVID BURKHARDT, M.D.,
F.H.R.S.,‡ and ANDREA NATALE, M.D., F.H.R.S.†,‡,|,**,††

From the "Department of Cardiology, University of Foggia, Foggia, Italy; †Texas Cardiac Arrhythmia Institute, St. David's Medical Center, Austin, Texas, USA; ‡Department of Biomedical Engineering, University of Texas, Austin, Texas, USA; §Albert Einstein College of Medicine, Montefiore Hospital, New York, USA; ¶Division of Cardiac Electrophysiology, Department of Medicine, Duke University Medical Center, Durham, North Carolina, USA; #Department of Heart and Vessel, University of Florence, Florence, Italy; ||EP Services, California Pacific Medical Center, San Francisco, California, USA; "Interventional Electrophysiology, Scripps Clinic, San Diego, California, USA; and ††Case Western Reserve University, Cleveland, Ohio, USA

VF from Papillary Muscle. Background: Animal studies showed that papillary muscles can be sources of ventricular fibrillation (VF) in both the left and right ventricle, but this occurrence in humans has been described only in patients with ischemic heart disease.

Objective: To investigate the role of papillary muscle premature ventricular contractions (PVCs) as triggers for VF and the safety and feasibility of catheter ablation in these patients.

Methods: Six patients (2 male; age, 40 ± 11 years; 5 with a normal structural heart and 1 with nonischemic cardiomyopathy) with history of VF resulting in repetitive implantable cardioverter defibrillator shocks, despite antiarrhythmic drug therapy, and a papillary muscle focus of PVCs triggering VF were included and underwent mapping and ablation of PVCs.

Results: PVCs were observed to trigger VF and localized by mapping the earliest activation point that matched pace mapping of the same area. In 2 patients, PVCs originated from the left ventricle at the posteromedial papillary muscle; in 4 patients, PVCs originated from the right ventricle, at the posterolateral papillary muscle. Elimination of the triggering PVC was obtained in these areas after 19 ± 12 minutes by radiofrequency application. During a follow-up of 58 ± 11 months using ambulatory monitoring and defibrillator memory interrogation, no patients had recurrence of symptomatic ventricular arrhythmias.

Conclusion: Papillary muscles from both ventricles represent an anatomic structure potentially involved in the onset of VF, also in normal structural heart. PVCs arising from this area can be successfully eliminated by radiofrequency ablation, resulting in freedom from recurrent VF at long-term follow-up. (J Cardiovasc Electrophysiol, Vol. 25, pp. 1158-1164, November 2014)





Case2. 42% PVC, no MVP, LVEF 49%

VPC Bigeminy

Location: Unknown

HOLTER REPORT

Room ID: IM03

147259 QRS complexes

61637 Ventricular beats (42%)

Supraventricular beats (< 1%)
 % of total time classified as noise

104 Minimum at 05:06:23 15-Apr

101 Average
141 Maximum at 22:27:27 14-Apr
3601 Beats in tachycardia (>100 bpm), 2% total
0 Beats in bradycardia (<60 bpm), 0% total
1.16 Seconds Max R-R at 06:08:42 15-Apr

Supraventriculars (S, J, A)

- 0 Isolated

O Couplets
D Bigeminal cycles
Runs totaling 0 beats

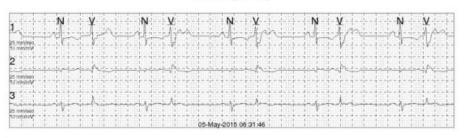
Ventriculars (V, F, E, I)

46945 Isolated

5955 Couplets 39690 Bigeminal cycles

884 Runs totaling 2782 beats
6 Beats longest run 140 bpm 22:18:11 14-Apr
3 Beats fastest run 185 bpm 09:00:30 15-Apr





Non-sustained VT (5beats)



Non-sustained VT (5 beats)









Medication

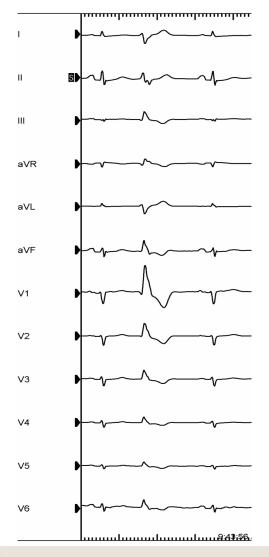
- 4.23, Holter PVC 42%, NSVT 6 beats, presyncope
 - → verapamil 40mg tid
- 5.6, Holter PVC 30%, NSVT 5 beats, presyncope
 - > verapamil 40mg tid,
 - → add propafenone 150mg bid
- 6.2, Holter PVC 31%, LVEF 50% with palpitation consider amiodarone or catheter ablation





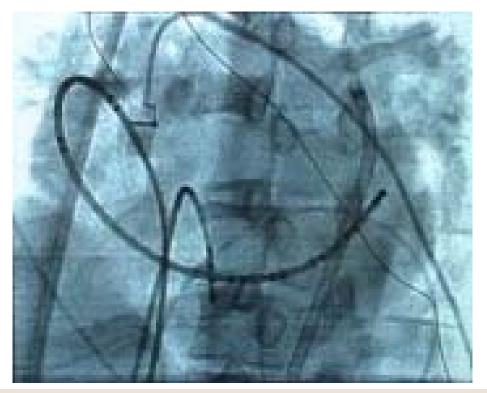


Baseline PVC ECG



Catheter position

- Duodeca (HRA- distalCS)
- Deca (His-RV)
- Ablation (irrigation DF curve) retrograde aortic approach

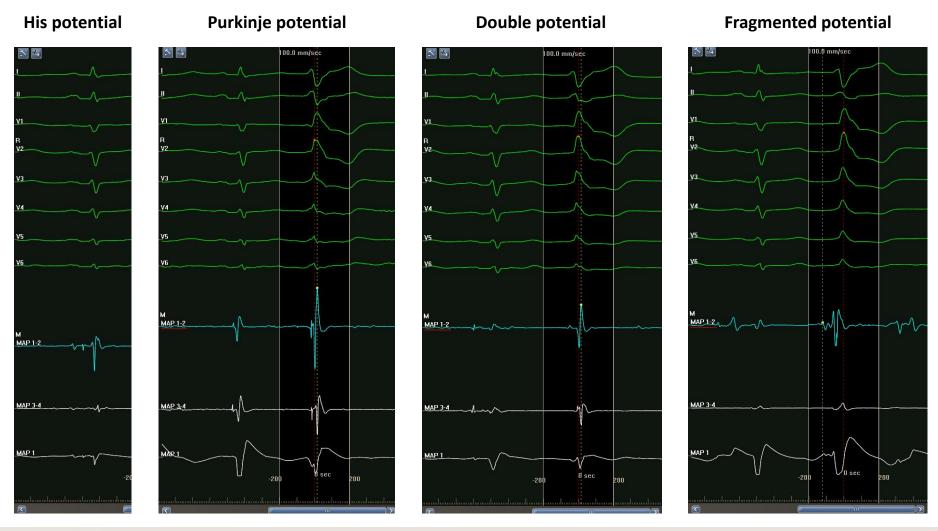








EGM at 3D mapping system









ICE

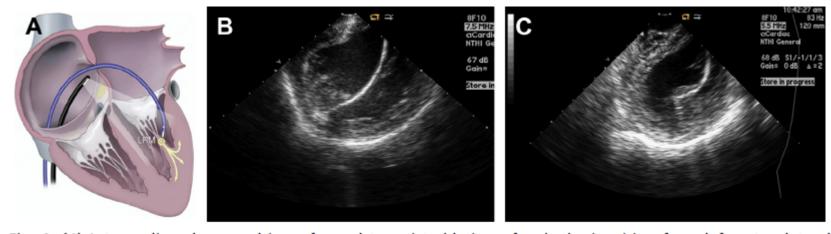
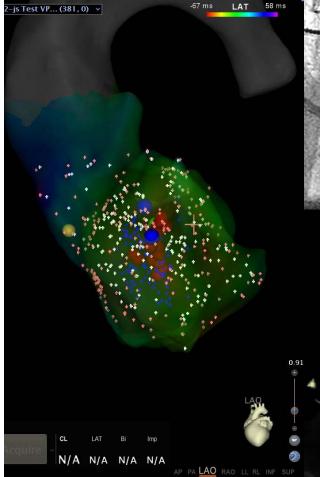


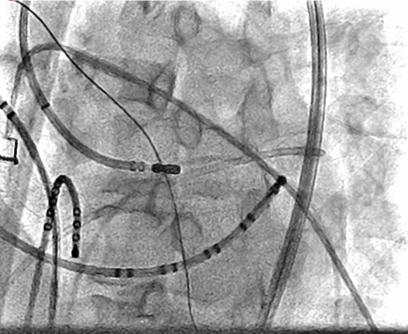
Fig. 4. (A) Intracardiac ultrasound is performed to assist ablation of arrhythmias rising from left anterolateral papillary muscle. (B, C) It allows clear visualization and confirmation of contact with the anterolateral and posteromedial muscles. ([A] From Liu XK, Barrett R, Packer DL, et al. Successful management of recurrent ventricular tachycardia by electrical isolation of anterolateral papillary muscle. Heart Rhythm 2008;5(3):481; with permission.)



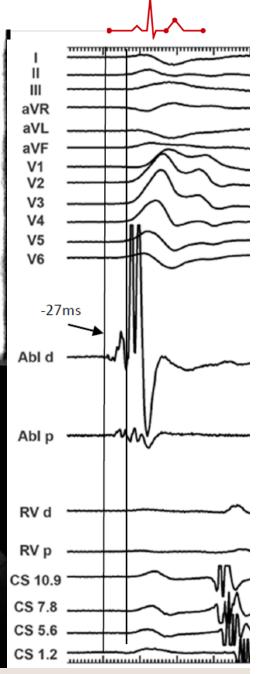


RF ablation







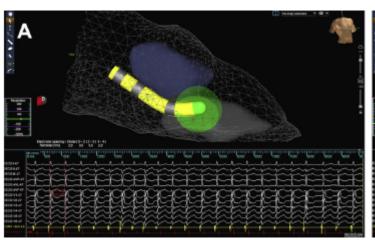








Cryoablation vs. RF ablation



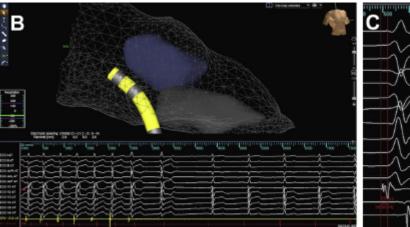


Table 1. Baseline Characteristics

Characteristics	RF (n=9)	CRYO (n=12)	<i>P</i> Value
Age, mean	40.0±10.6	41.4±14.3	0.9
Male	4 (44.4%)	6 (50%)	0.8
LVEF	59.9±9.5	58.1±5.1	0.08
SHD	3 (33.3%)	4 (33.3%)	1.0
VA			
VT	2 (22.2%)	3 (25.0%)	0.04
NSVT	1 (11.1%)	7 (58.3%)	
PVC	6 (66.7%)	2 (16.7%)	
AADs Pre	1 (11.1%)	3 (25%)	0.4
AADs Post	1 (11.1%)	0	0.4

Table 2. Procedural Characteristics

Characteristics	RF (n=9)	CRYO (n=12)	P value		
P Success	7 (77.8%)	12 (100.0%)	0.08		
Recurrence	4 (44%)	0	0.03		
RF Time	11.3±4.2	11.0±3.0	0.9		
Energy time	425.3±86.1	700.0±216.1	0.002		
TP Time	131.7±9.0	126.5±25.5	0.4		
Complications	0	0			
ProA	8 (88.9%)	0	0.001		
Cath Stab	2 (22.2%)	12 (100.0%)	0.001		
Days F-UP	87 (IQR, 65-148)	360 (IQR, 116-365)			

Cath stab indicates catheter stability during energy delivery; CRYO,







Contact force sensing catheter

LV PMs (N = 59)	Non-CFS RF/ CTII (N = 23)	CFS RF/ICE3D (N = 18)	CRYO/CTII (N = 18)	Р
Catheter stability	6 (26%)	9 (50%)	18 (100%)	<0.0001
Pro-arrhythmia	18 (78%)	14 (78%)	O (O%)	<0.0001
VEGM-QRS	$32.4 \pm 5.6 \text{ms}$	$33.2 \pm 4.7 \text{ ms}$	30.2 ± 12.4 ms	1
PMAP score	22 (IQR 22-24)	22 (IQR 22-24)	22 (IQR 22-24)	0.7
Effective lesion locatio	n			
I- PM Apex	2 (9%)	1 (6%)	2 (11%)	0.1
II- PM Body	2 (9%)	8 (44%)	5 (28%)	0.1
III- PM Base	19 (82%)	9 (50%)	11 (61%)	0.1
CRYO dose (seg.)	N/A	N/A	766.7 ± 321.8	N/A
RF Dose (seg.)	361.6 ± 182.3	915 ± 653.1	N/A	N/A
Success	19 (83%)	18 (100%)	18 (100%)	0.03
Patients (N = 53)	Non-CFS RF CTII (N = 23)	CFS RF/ICE3D (N = 14)	CRYO/CTII (N = 16)	Р
Procedure duration	139 ± 39.7 min	164.5 ± 58 min	131.3 ± 26.2 min	0.3
Fluoroscopy time	14.2 ± 4.6 min	6.2 ± 1.5 min	10.7 ± 4.2 min	<0.0001
Transeptal access	10 (44%)	8 (57%)	16 (100%)	0.001
Minor complications	0	2 (12%)	0	0.1
Major Complications	1 (4%)	0	0	8.0
Prior ablation	0	2 (14%)	0	0.06
Circumferential	0	3 (21%)	2 (13%)	0.08
Focal	23 (100%)	12 (86%)	14 (88%)	0.2
Recurrence	11 (48%)	1 (7%)	3 (19%)	0.0172
FUP time (Mo)	12 ± 10	15 ± 18	13 ± 7.5	0.5307

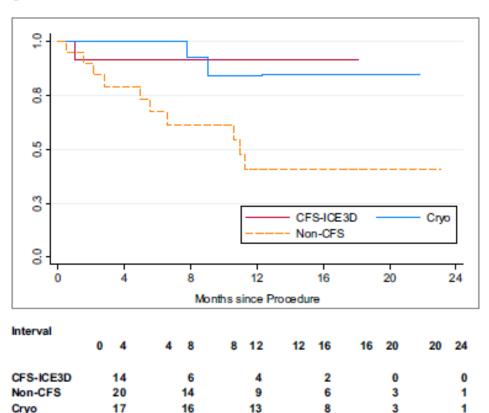


FIGURE 4 Patients free from ventricular arrhythmias after catheter ablation

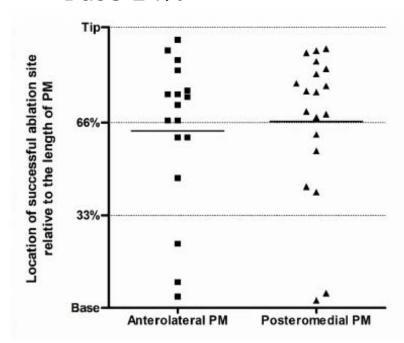


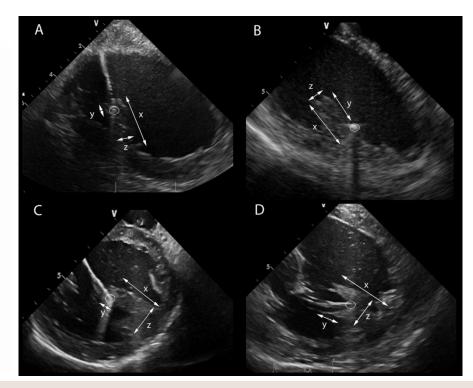




Tip or Base?

- Successful ablation site was located on
 - Tip 67%
 - Mid 19%
 - Base 14%



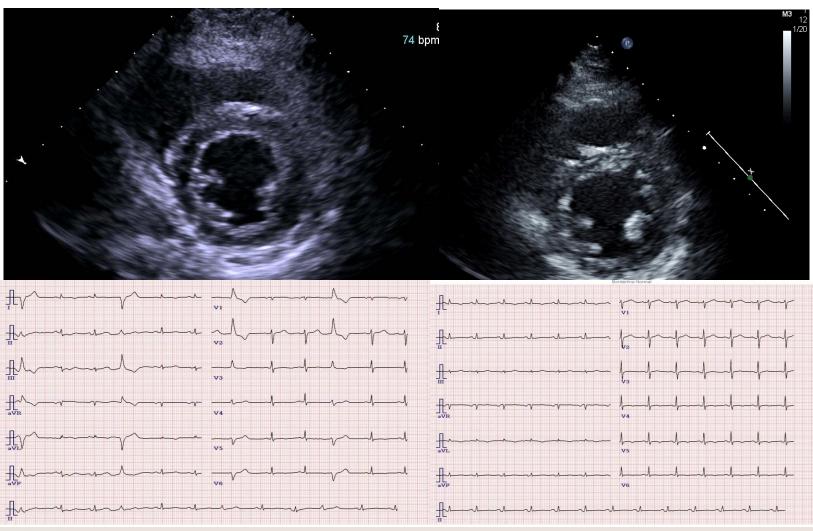








Post procedure TTE & ablation efficacy









Practical aspect of ablation for PM PVC

- ICE is necessary!
- Consideration anatomy
- If unsuccessful, change the access route
 - Retrograde vs. transseptal
- High power may be required without good contact
 - 30 to 70W, impedence drops upto 8 to 10 Ω
 - Be careful at the base
 - Consider rapid pacing to suppress cardiac motion
- Cryoablation to improve stability can help.







Summary

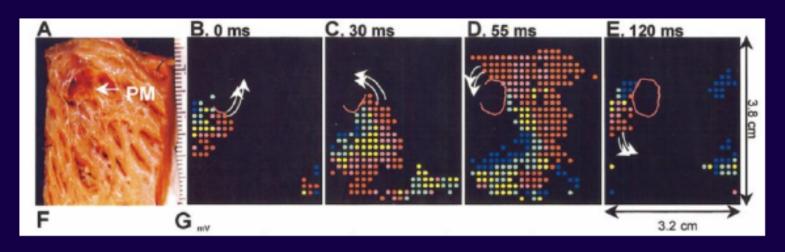
- PVC from papillary m have specific characteristics.
- Ablation may be challenging
- The recurrence risk is higher than for other forms of idiopathic VT
- Minimize sedation till PVCs are seen and mapped
- ICE is very helpful for defining anatomy and monitoring catheter-tissue contact
- Cryo-ablation may prove better than RF energy, but larger studies are need for comparison

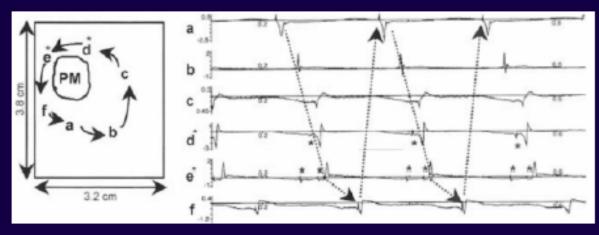




Role of Papillary Muscle in the Generation and Maintenance of Reentry During Ventricular Tachycardia and Fibrillation in Isolated Swine Right Ventricle

Young-Hoon Kim, MD; Fagen Xie, PhD; Masaaki Yashima, MD; Tsu-Juey Wu, MD; Miguel Valderrábano, MD; Moon-Hyoung Lee, MD; Toshihiko Ohara, MD; Olga Voroshilovsky, MS; Rahul N. Doshi, MD; Michael C. Fishbein, MD; Zhilin Qu, PhD; Alan Garfinkel, PhD; James N. Weiss, MD; Hrayr S. Karagueuzian, PhD; Peng-Sheng Chen, MD





Ventricular Fibrillation from PM

Ventricular Fibrillation Triggered by PVCs from Papillary

DECLIETE . FCC

RESULTS: PLPs potentials

PAT

nonischer

In 2 patie

muscle: ir

papillary I

postero PM rev bundle Six patien block (I morpho (positiv transiti V5 and superic

PVCs fr

PVCs wer

posteron Purkinje-like potentials (PLPs) preceding the PVC were found in 4 out of 6 patients.

PVCs occ phenylep

remainin PLPs were noted to precede the PVC potential arising from the RV posterolateral PM, in 3 out of 4 patients

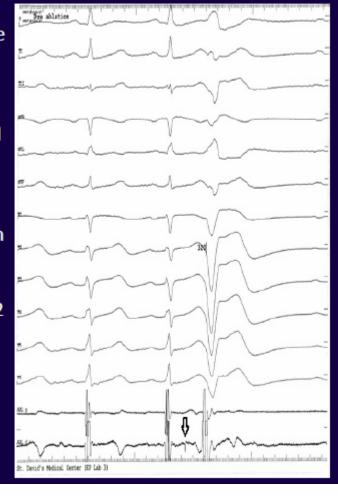
Earliest a the ablat intracard was 42 ±

PLPs were present in the septal and mitral annular endocardial scar of the patient with NICM.

Pace mar match wi all patien

An ICE gu was perfe activation mapping.

The septal scar extended to the medial border of the PM in this patient and these 2 areas were targeted for ablation with consolidating lesions.







PM VTs and mitral valve prolaps

- 7% of patients with SCD had MVP
- PVCs from PM may act as trigger VF in MVP
- Possible mechanism
 - Mechanical stretch, elongated chordae → fibrosis → conduction delay and micro-reentry
 - MRI-LGE inferobasal wall fibrosis
- MVP not seem to impair successful PVC ablation

