When to ablate a high PVC burden

Fall Update in Cardiology Symposium

Jordan Brewster, MD
Oklahoma Heart Institute
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~ No relevant disclosures ~

Getting started

★PVCs are common

- So what? Why do they matter?
- What potential harm do they impose?

★When we see a patient with PVCs, what do we do?

- Typical diagnostic work up and follow up

…When to ablate?

…What is a “high PVC burden?”

Case

★66 yo man with LVEF 20% and LVEDD 5.9 cm

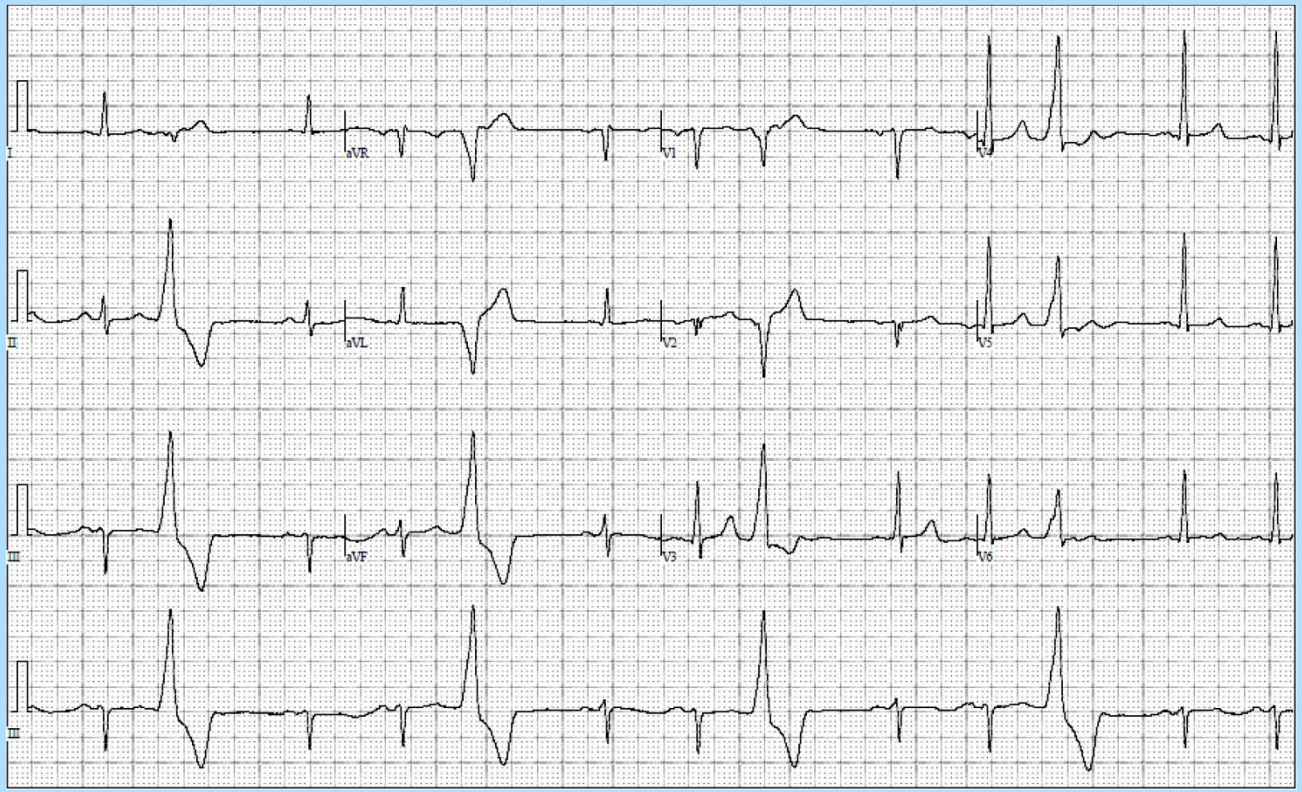
★Normal coronaries on catheterization

★Coreg and Entresto for > 3 months

★Referred for ICD

Case

- ★PVCs noted on EKG
  - ▶Holter 24.8% PVC burden
- ★PVC ablation of the LCC/AMC
  - ▶After 6 months:
    - ▸ Holter < 1% PVCs
    - ▸ ECHO: EF 20% to 45%
    - ▸ LVEDD 5.9 to 5.4 cm
    - ▸ No ICD recommended



Premature ventricular contractions

★Focal, premature myocardial depolarizations from the ventricle

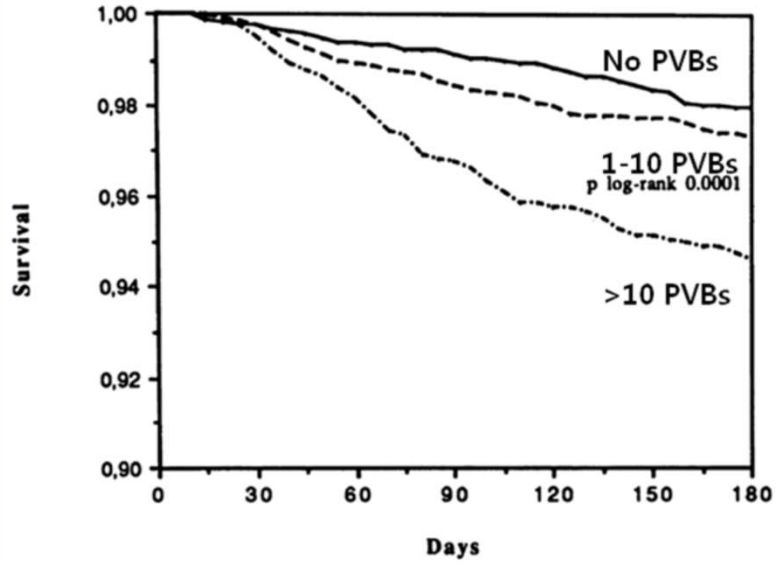
★In normal patients, detected on 1-5% of ECGs and 40-75% of Holter monitors

- ▶ More prevalent with age

★In structural heart disease (i.e. post MI), PVCs correlated with increased mortality

- ▶ *PVC suppression not shown to reduce mortality*

Increased mortality in post-MI patients with increasing PVC frequency



Miaggioni et al. GISSI-2 results. *Circulation* 1993;87:312-22.

Cardiac Arrhythmia Suppression Trial (CAST)

Flecainide, encainide effectively suppressed PVCs post MI

- Resulted in increased cardiac mortality
- Class IC antiarrhythmics HARMFUL in ischemic disease

Abstract *Background and Methods.* In the Cardiac Arrhythmia Suppression Trial, designed to test the hypothesis that suppression of ventricular ectopy after a myocardial infarction reduces the incidence of sudden death, patients in whom ventricular ectopy could be suppressed with encainide, flecainide, or moricizine were randomly assigned to receive either active drug or placebo. The use of encainide and flecainide was discontinued because of excess mortality. We examined the mortality and morbidity after randomization to encainide or flecainide or their respective placebo.

Results. Of 1498 patients, 857 were assigned to receive encainide or its placebo (432 to active drug and 425 to placebo) and 641 were assigned to receive flecainide or its placebo (323 to active drug and 318 to placebo). After a mean follow-up of 10 months, 89 patients had died: 59 of arrhythmia (43 receiving drug vs. 16 receiving placebo; P = 0.0004), 22 of nonarrhythmic cardiac causes (17 receiving drug vs. 5 receiving placebo; P = 0.01), and 8 of noncardiac causes (3 receiving drug vs. 5 receiving placebo). Almost all cardiac deaths not due to arrhythmia were attributed to acute myocardial infarction with shock (11 patients receiving drug and 3 receiving placebo) or to chronic congestive heart failure (4 receiving drug and 2 receiving placebo). There were no differences between the patients receiving active drug and those receiving placebo in the incidence of nonlethal disqualifying ventricular tachycardia, proarrhythmia, syncope, need for a permanent pacemaker, congestive heart failure, recurrent myocardial infarction, angina, or need for coronary-artery bypass grafting or angioplasty.

Conclusions. There was an excess of deaths due to arrhythmia and deaths due to shock after acute recurrent myocardial infarction in patients treated with encainide or flecainide. Nonlethal events, however, were equally distributed between the active-drug and placebo groups. The mechanisms underlying the excess mortality during treatment with encainide or flecainide remain unknown. (N Engl J Med 1991; 324:781-8.)

eath and Cardiac Arrest (with Resuscitation) in the CAST, According to Treatment Group.

	Encainide Group		Flecainide Group		Both Groups		Total
	Active Drug	Placebo	Active Drug	Placebo	Active Drug	Placebo	
	number of patients						
	432	425	323	318	755	743	1498
arrests	44	19	19	7	63	26†	89
arrest	42	15	18	6	60	21‡	81
tation	5	1	2	0	7	1	8
	29	12	14	4	43	16§	59

Idiopathic premature ventricular contractions

★*No evidence of structural heart disease following investigation*

★60-70% arise from the outflow tract of the RV or LV

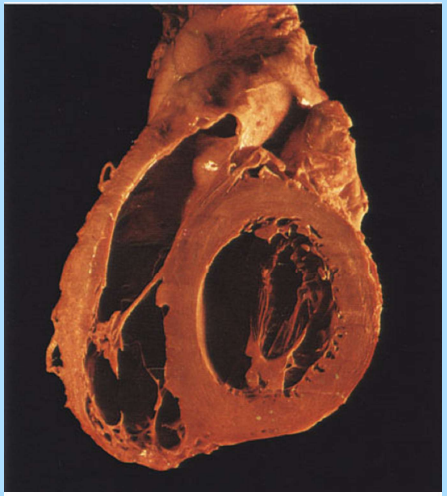
★PVCs at rest generally considered benign finding

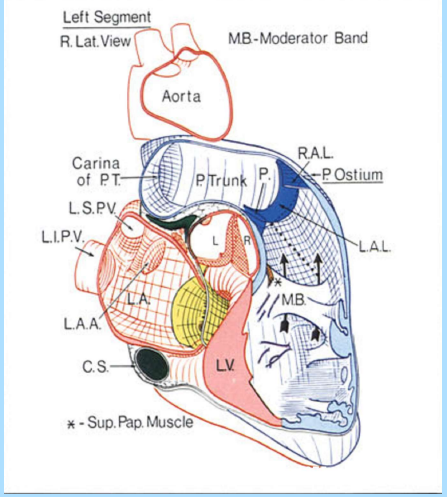
- *Idiopathic PVCs induced with exercise may impart negative prognosis*

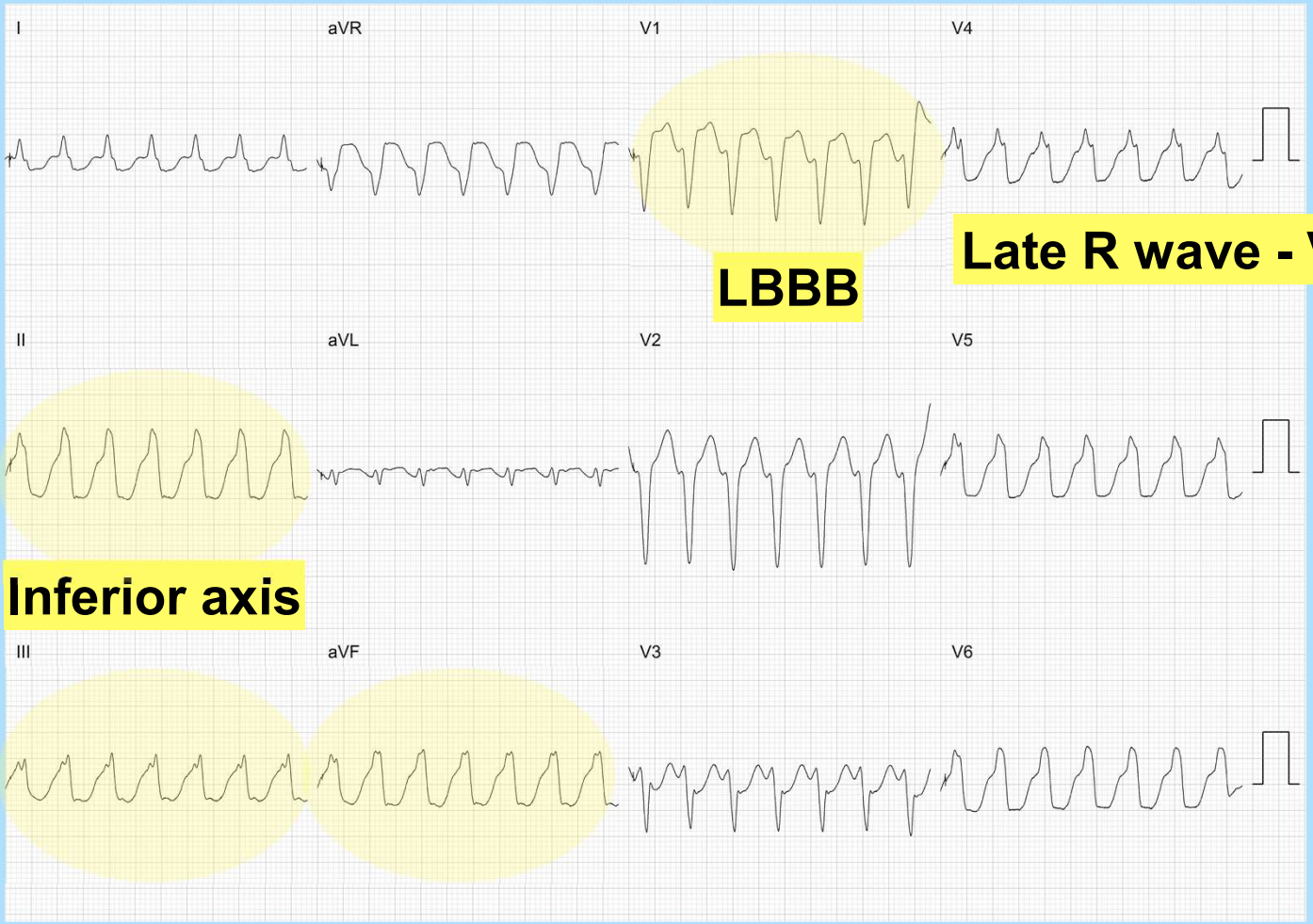
★**Circumstances requiring treatment**

- Symptoms
- Cardiomyopathy
- *PVCs triggering VF*
- *Suboptimal CRT pacing*

RVOT VT/PVC

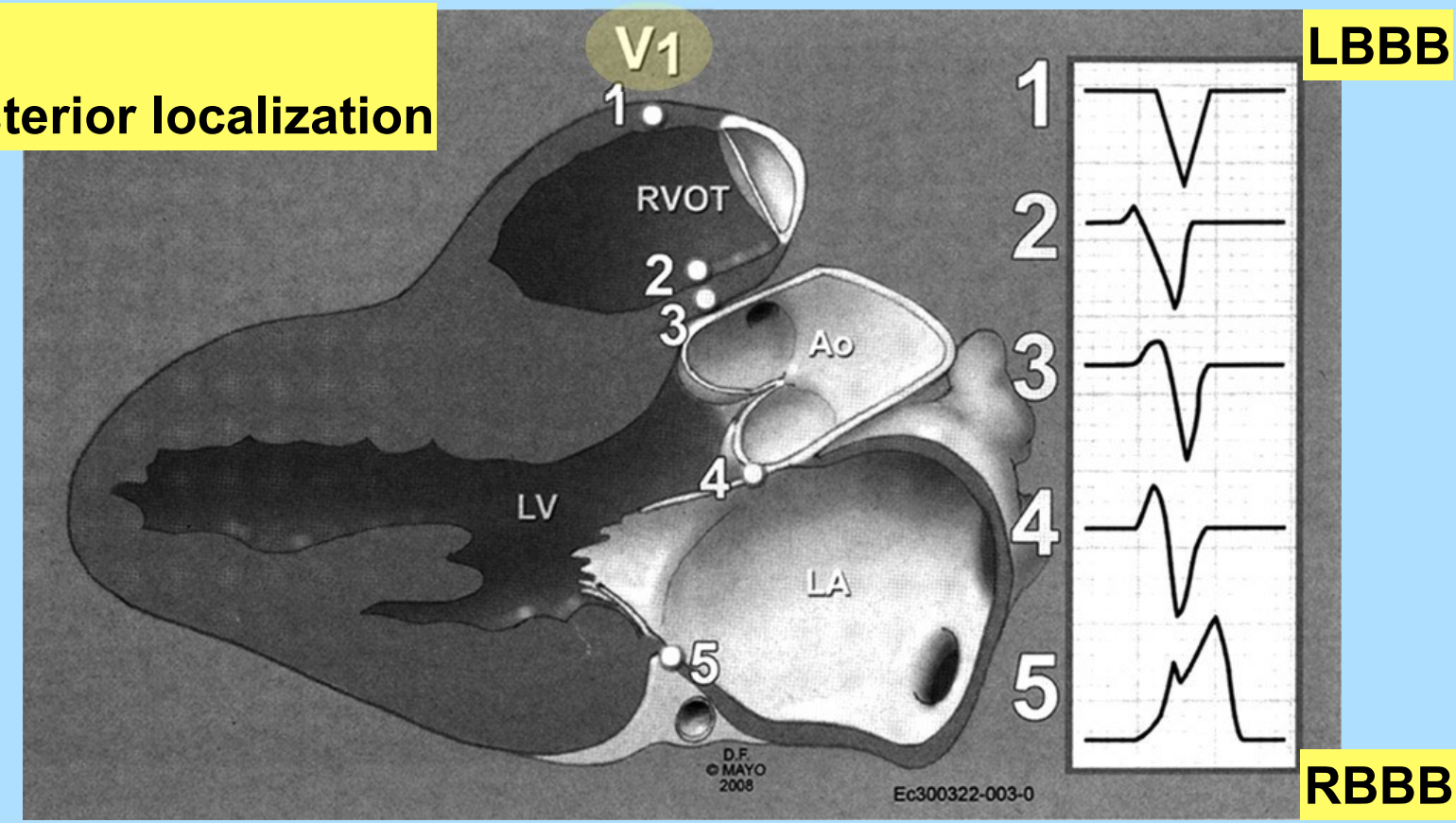



Left Segment
R. Lat. View
MB-Moderator Band
Aorta
Carina of P.T.
P. Trunk
P.
R.A.L.
P. Ostium
L.S.P.V.
L.I.P.V.
L
R
L.A.L.
L.A.
MB
L.A.A.
C.S.
LV
* - Sup. Pap. Muscle


I
aVR
V1
V4
LBBB
Late R wave - V4
II
aVL
V2
V5
Inferior axis
III
aVF
V3
V6

Outflow tract PVCs

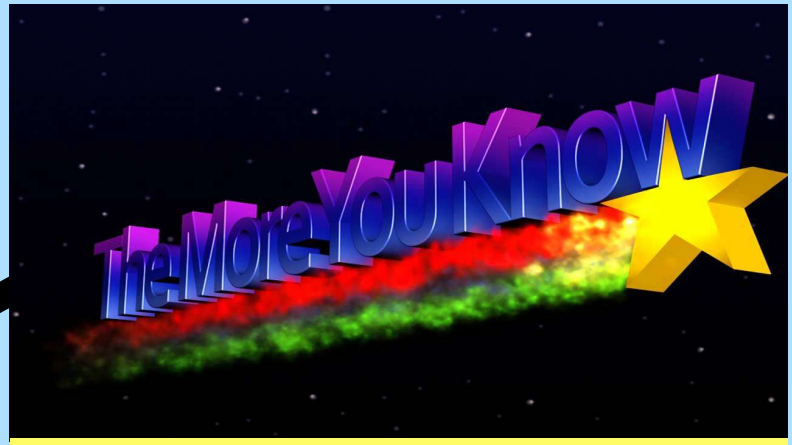
Lead V1:
Anterior-posterior localization



Prystowski, et al. *JACC* 2012;59(20):1733-44

Common PVC related symptoms

- ★Palpitations
  - ▶Chest “pounding”
  - ▶Heart “stops”
- ★Fatigue
- ★Exercise intolerance
- ★Dizziness/lightheadedness
- ★**Pseudo-bradycardia (non-perfused PVCs)**



A pacemaker will <u>not</u> treat PVCs

PVC-induced cardiomyopathy

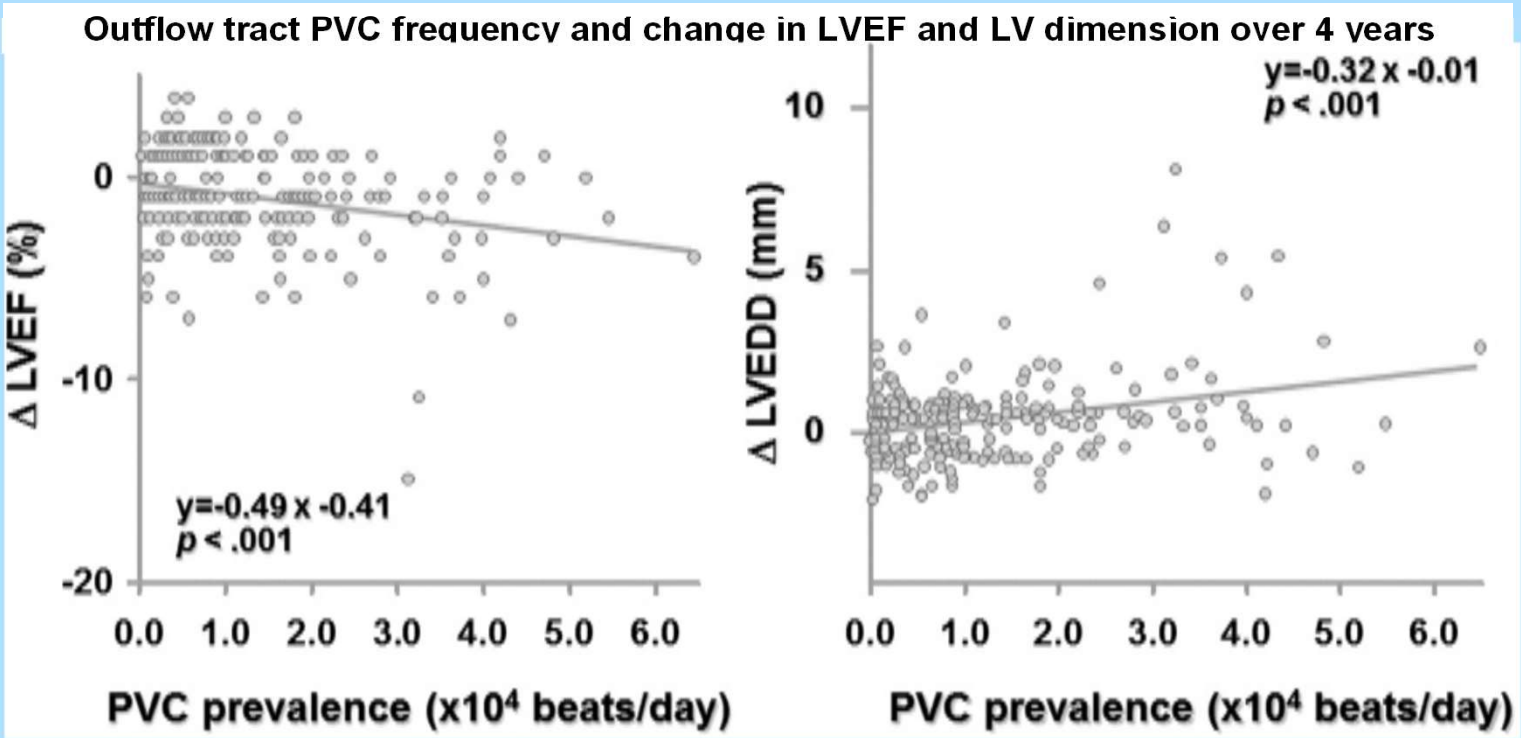
★**Duffy et al. 1998**

★**<u>Reversible</u> CMP associated with PVCs**

★Retrospective case study, 14 patients with >20k PVCs Holter, EF <40%

- 7 patients received medical therapy for PVCs (4 with amiodarone)
  - 5 of these patients experienced >75% reduction in PVC burden on therapy
  - 4/5 experienced increased LVEF (27% ± 10% to 49 ± 17%)

LV dysfunction and PVC burden



Niwano et al. *Heart* 2009;95:1230-7

PVC frequency and PVC-CMP

★Broad range of PVC percentages associated with risk

- Kanei et al. > 10k PVCs/24 hours
- Niwano et al. > 20k PVCs/24 hours
- Yarlagadda et al. > 5% burden
- Hasdemir et al. >10% burden
- **Baman et al. >24% (sens./spec. of 79/78%)**
- MOST trial (RV pacing) >40%

★Most patients with *frequent* PVCs have *preserved* LVEF

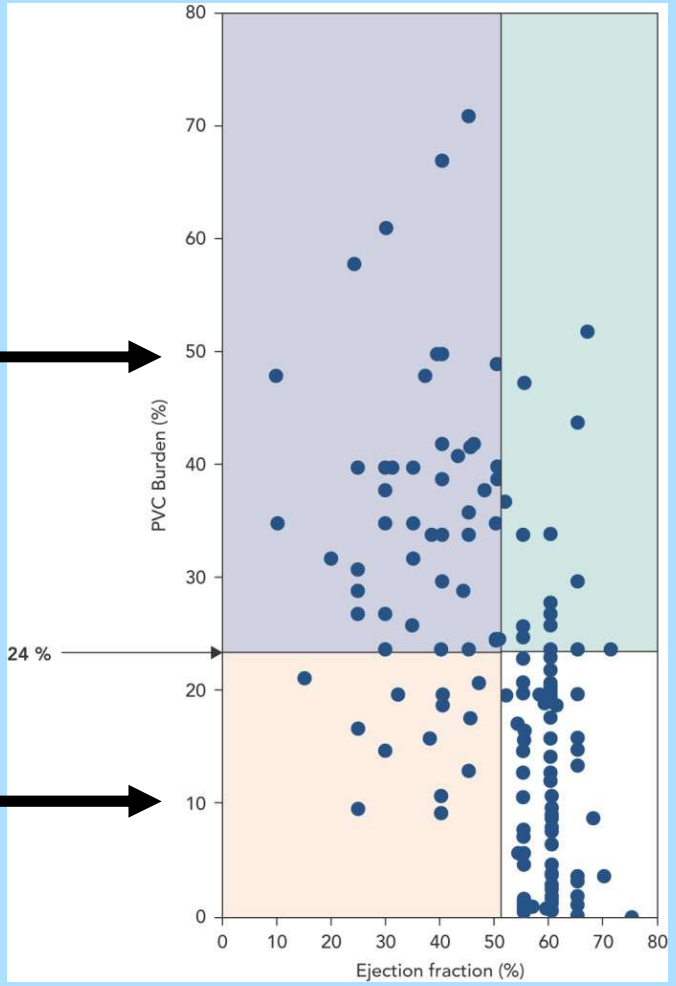
PVC burden and CMP

174 consecutive patients with idiopathic PVCs
-24 Holter
-ECHO

PVC burden (%)	Sensitivity (%)	Specificity (%)
10	100	46
16	90	58
21	80	75
27	70	88
30	60	91
34	50	91
35	40	94
39	30	95
41	20	96
50	10	99

Most specific

Most sensitive



Baman et al. *Heart Rhythm* 2010;7:865-9

Other risk factors for PVC-CMP

★PVC origin: RV origin > LV origin

★PVC QRS duration >140 ms

★PVC coupling interval ≤450 ms (“short-coupled” PVCs)

★Interpolated PVCs

★Male gender

★Lack of symptoms

★Epicardial origin

Data are conflicting

Catheter ablation of PVCs

★Yarlagadda et al. 2005

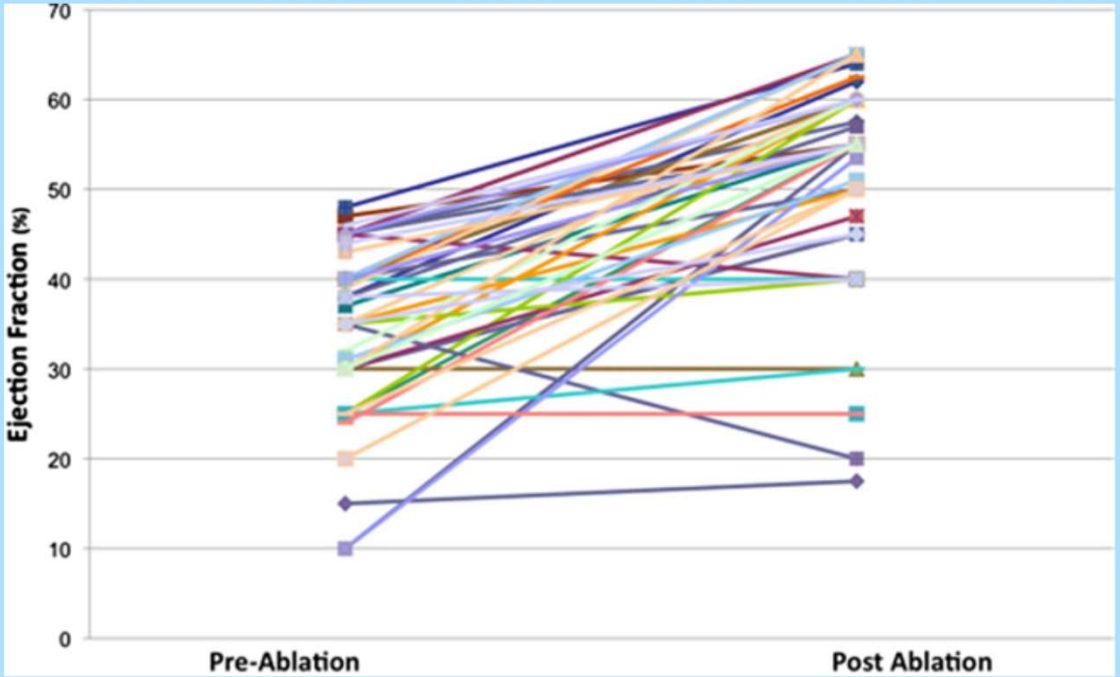
★27 patients with RVOT PVCs presented for catheter ablation

- 8 patients with LVEF <45%
  - 7/8 had successful ablation and resultant **increase in LVEF** (39 ± 6% to 62 ± 6%)
  - Mean PVC burden >17,000/24 hr
  - 2/8 with cardiomyopathy had < 6,000/24 hr

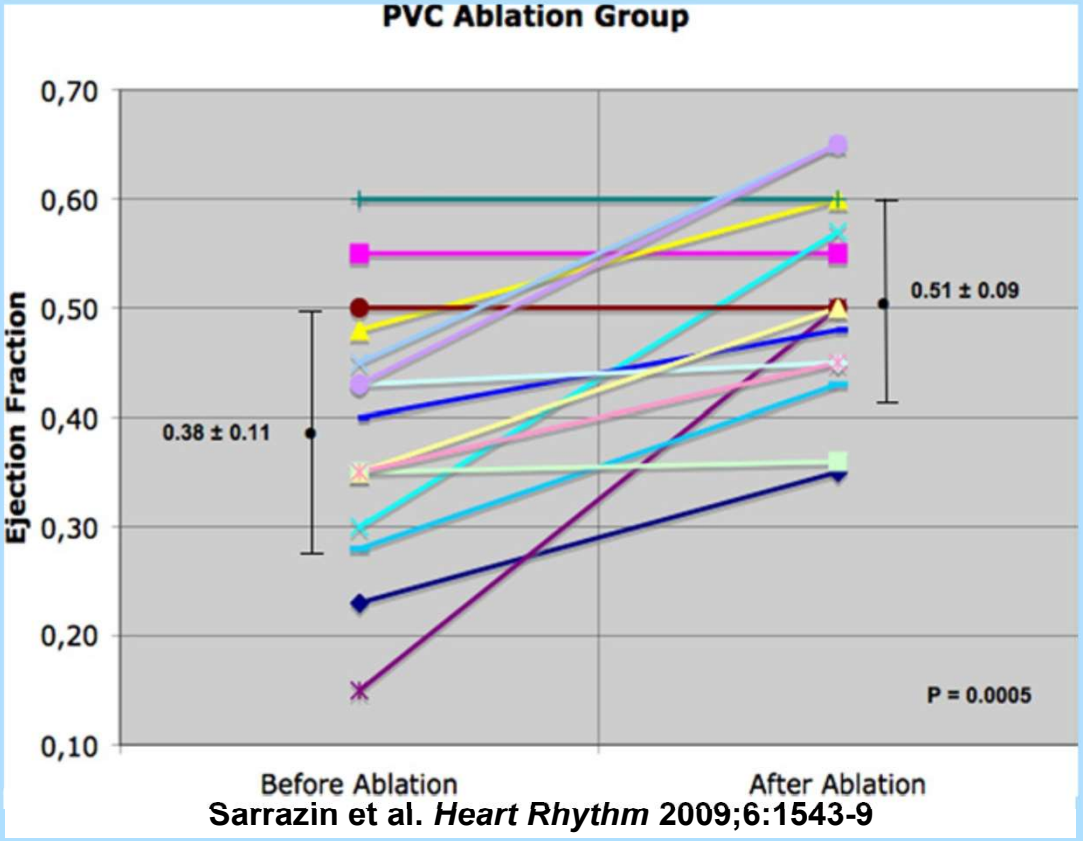
Baman et al. 2010

★146/174 patients (84%) with successful idiopathic PVC ablation

★Mean LVEF increased from 35 ± 9% to 54 ± 10%

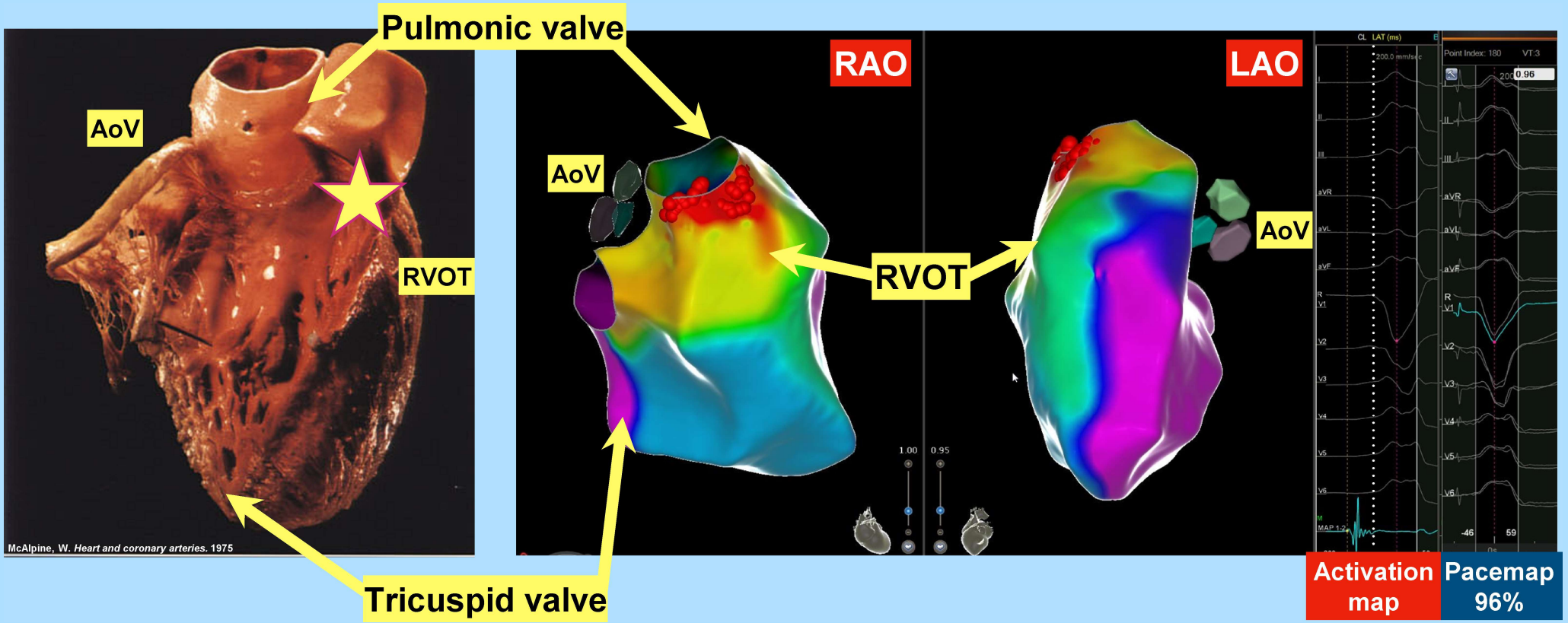


Ischemic PVC ablation can improve EF

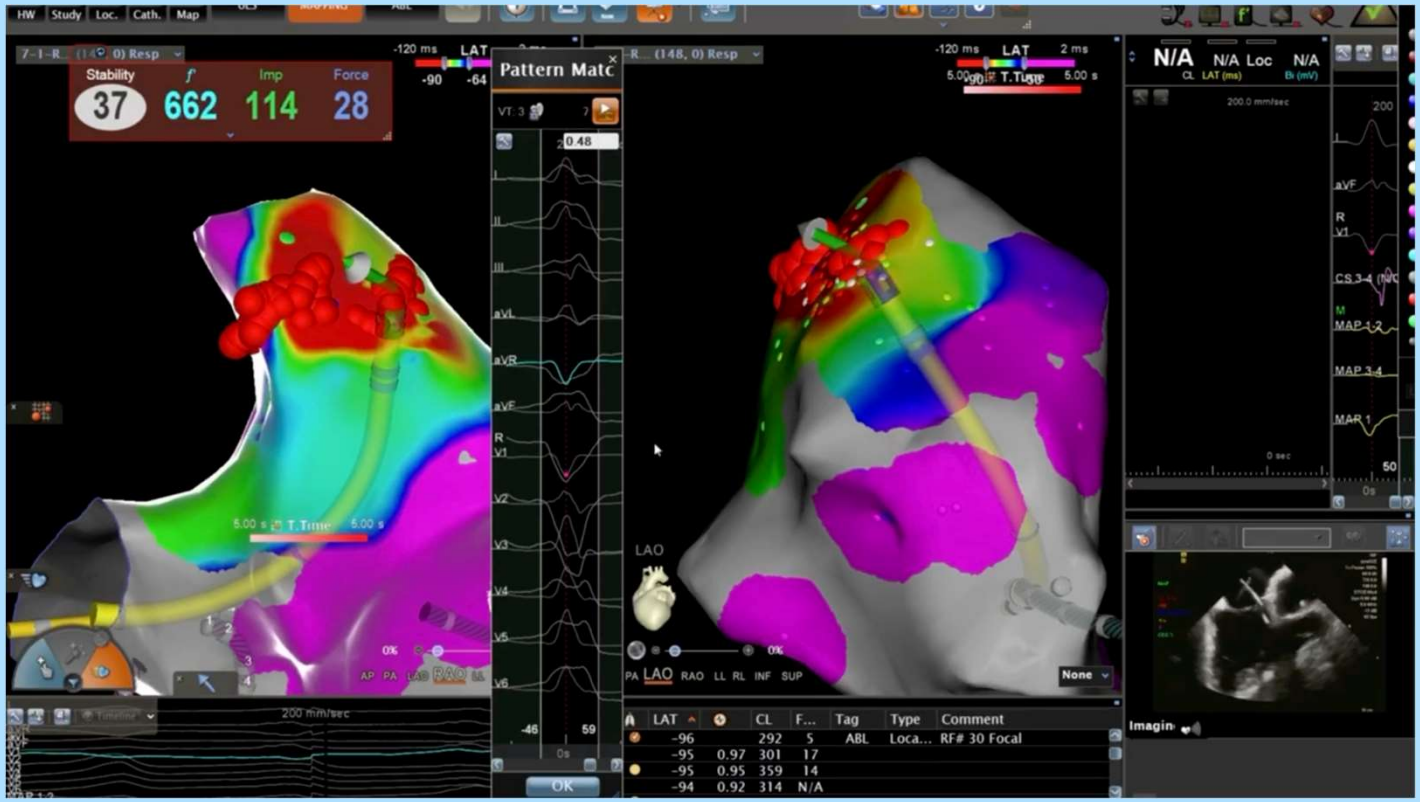


Sarrazin et al. *Heart Rhythm* 2009;6:1543-9

RVOT VT/PVC


Pulmonic valve
AoV
RVOT
McAlpine, W. Heart and coronary arteries. 1975
Tricuspid valve
RAO
LAO
AoV
AoV
RVOT
1.00
0.95
CL LAT (ms)
Point Index: 180
VT:3
0.96
-46
59
Activation map
Pacemap 96%

RVOT VT/PVC


Stability
37
f
662
Imp
114
Force
28
Pattern Matc
LAT
LAO
N/A
N/A Loc
N/A
OK
Imagin

Challenges in catheter ablation

★Low PVC burden - limits mapping efforts

★Multiple PVC morphologies

★Patient comorbidities

★Non-outflow tract or epicardial origin

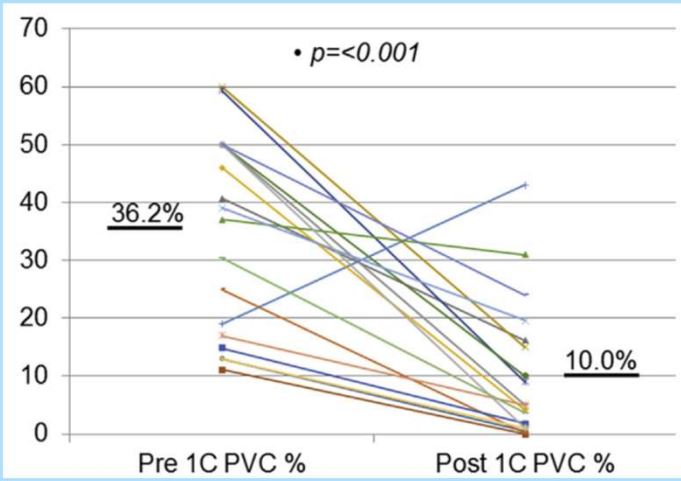
Medical therapy can be useful

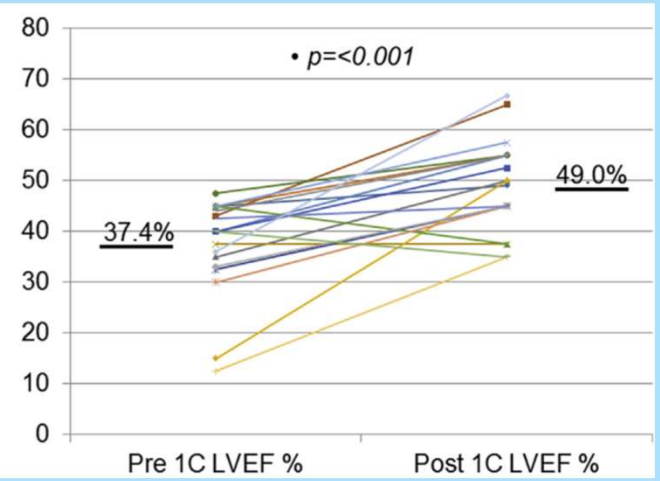
- **beta-blockers/CCBs**
- **flecainide**
- **sotalol**
- **amiodarone**

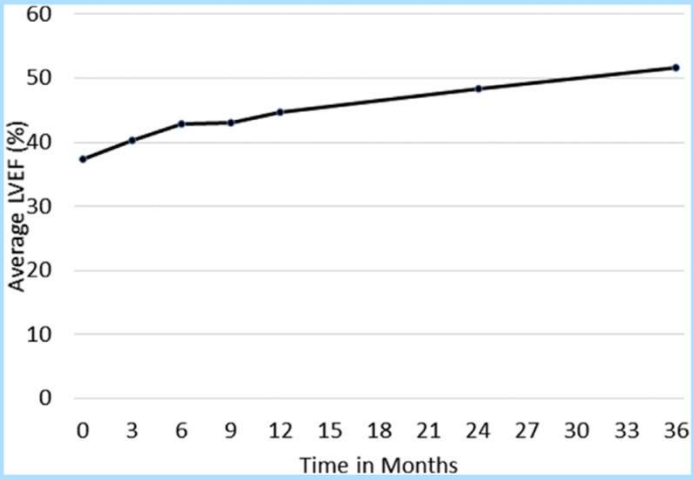
Flecainide for idiopathic PVC-CMP

★Flecainide decreased PVC burden and improved LVEF in **idiopathic** PVC-CMP

★No SCD or sustained ventricular arrhythmias reported







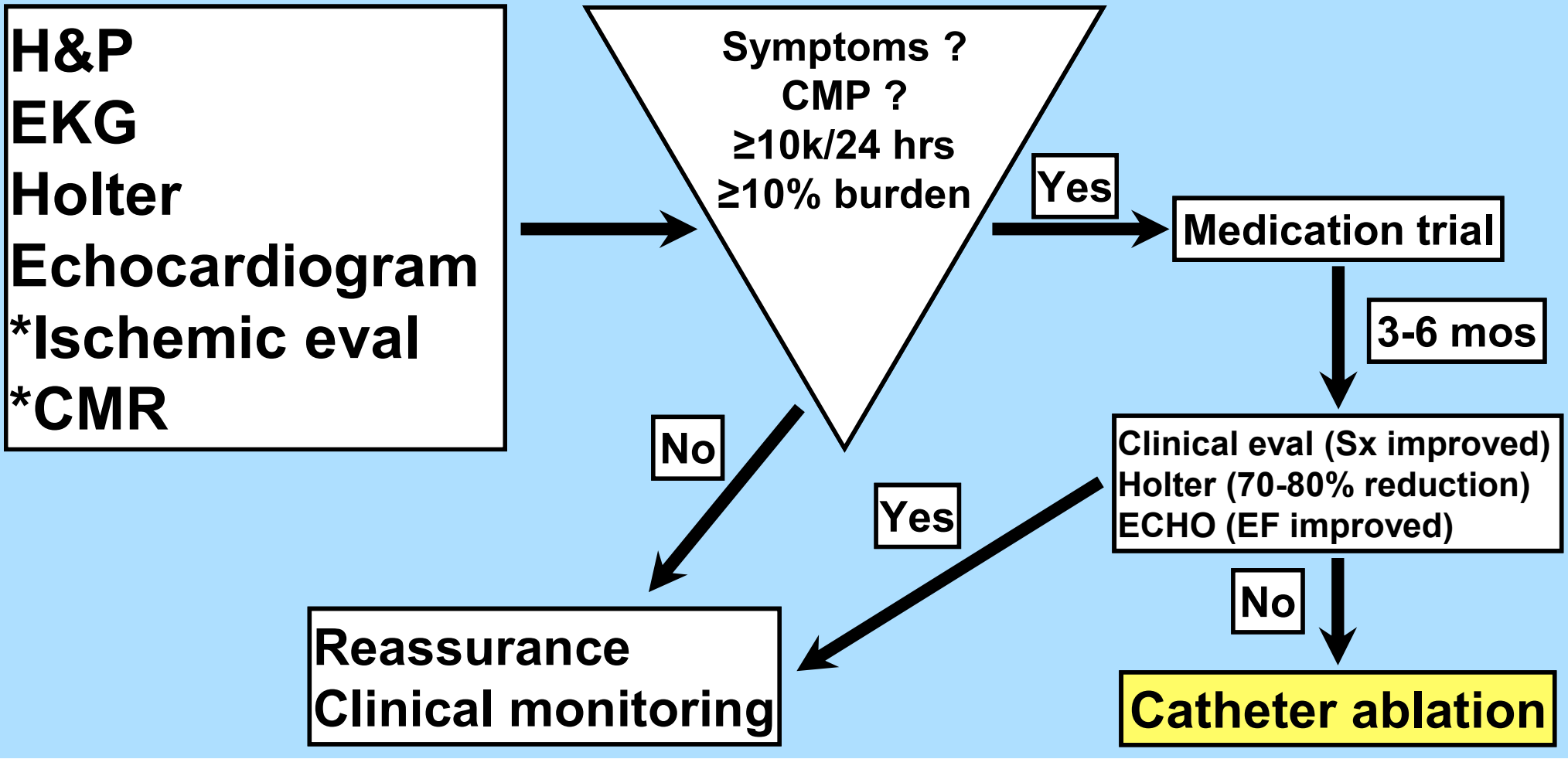
Hyman et al. *Heart Rhythm* 2018;15:159-68

Guidelines for PVC ablation summary

COR	LOE	Catheter ablation recommended/useful for:
I	B	Frequent and symptomatic PVCs from RVOT w/o SHD in preference to metoprolol/propafenone
I	B	Patients with CMP from frequent PVCs when AADs ineffective, not tolerated, or not preferred
I	B	Frequent and symptomatic non-OT RV/LV PVCs w/o SHD when AADs ineffective, not tolerated, or not preferred
IIa	B	Frequent and symptomatic PVCs from LVOT w/o SHD when AADs ineffective, not tolerated, or not preferred
IIa	B	Patients with SHD with frequent PVCs contributing to CMP when AADs ineffective, not tolerated, or not preferred
IIa	B	Patients with focally PVC triggered VF refractory to medical therapy
IIa	C	Nonresponders to cardiac resynchronization (CRT) when unifocal PVCs limit CRT pacing

Cronin et al. *Heart Rhythm* 2019

Algorithm for evaluation and management



Conclusions

★PVCs are commonly encountered arrhythmias

★Usually benign in the absence of structural heart disease

★PVCs may impart bothersome symptoms

★A burden of ≥10% idiopathic PVCs is associated with CMP

★Catheter ablation useful to improve symptoms and reverse CMP

~Thank you~