Diagnosis and Management of Idiopathic PVCs

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Case

- 18 y/o man, active in collegiate athletics (football)
- Tooth extraction: bigeminy
- Echo EF: 48%, biatrial↑; Holter 48,000 PVCs
- ECG: next slide
- No syncope, CHF, CVA, palpitations
- SX; biochemical eval incl thyroid: all normal
- RVOT ablation and Ao cusp mapping failed elsewhere
- Next step?

Outside ECG

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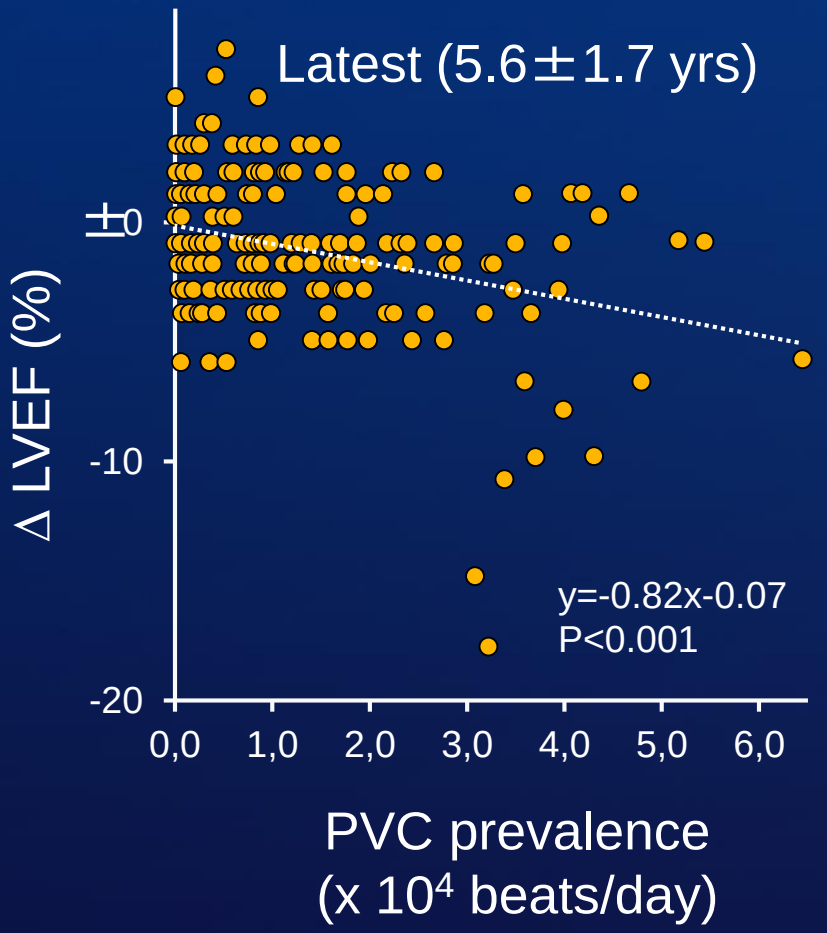
Case: Questions to Answer

- Are the PVCs causing ventricular dysfunction, or vice versa, and how can we tell?
- Are there PVC or other characteristics to predict LV dysfunction?
- Where are the PVCs coming from
- What medications should the PVCs respond to?
- How might we approach these for ablation?

Relationship Between Number of PVCs and Ventricular Dysfunction

- 239 consecutive patients
- >1000 beats/day (outflow)
- No heart disease
- Patients with Sx or ventricular dysfunction:
  - Ablated
  - Excluded from study
- Followed: 5.6 (1.7) yrs
- 13 patients had drop in EF more than 6%



Heart 95:1230, 2008

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Relationship Between Number of PVCs and Ventricular Dysfunction

75]

80]

13 pts with drop in LVEF: Time course

Predictors: Prevalence of PVCs (highest risk >20K)
Baseline EF

Other papers: PVC burden >26%. (Ban et al, Europace 2013)
PVC burden >20K/day (Del Carpio JCE 2011)

Time course: Years (slow progression) – adverse events

Majority of patients (85%): Can be reassured, and observed

Heart 95:1230, 2009

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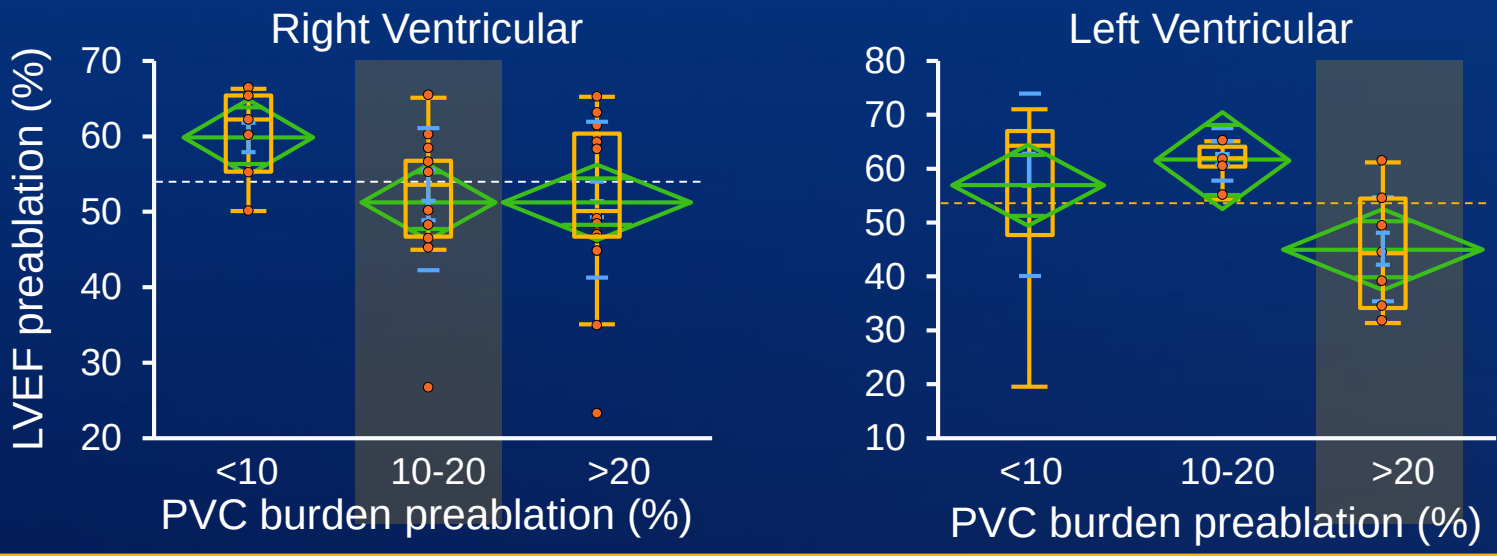
Site of Origin of PVC and Dysfunction



Site of Origin Impacts PVC Effect on Ejection Fraction

- >10K PVCs from RV (LBBB)
- >20K PVCs from LV (RBBB)
- QRS ≥ 140 msec associated with LV dysfunction

PVC duration ≥140 ms	-0.043	-0.080	-0.006	0.021
Fascicular PVC	0.068	0.004	0.131	0.035
Multiform PVC	-0.011	-0.065	0.043	0.703

- ?dysynchrony

Del Carpio…..Asirvatham JCE 2011

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Approach of when to treat (ablate) unifocal PVCs?

DX

- Confirm unifocal (12 lead Holter)
- Consider CT/MRI – confirm structurally normal
- Biochemical screen (thyroid)

Rx?

- Symptomatic, ↓ Ejection Fraction, ↑ LVEDD

Rx:

- Beta blockers/calcium blockers -uncommonly effective
- Membrane active drugs (1C, III): effective
- Ablation typically preferred

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Where is it coming from?

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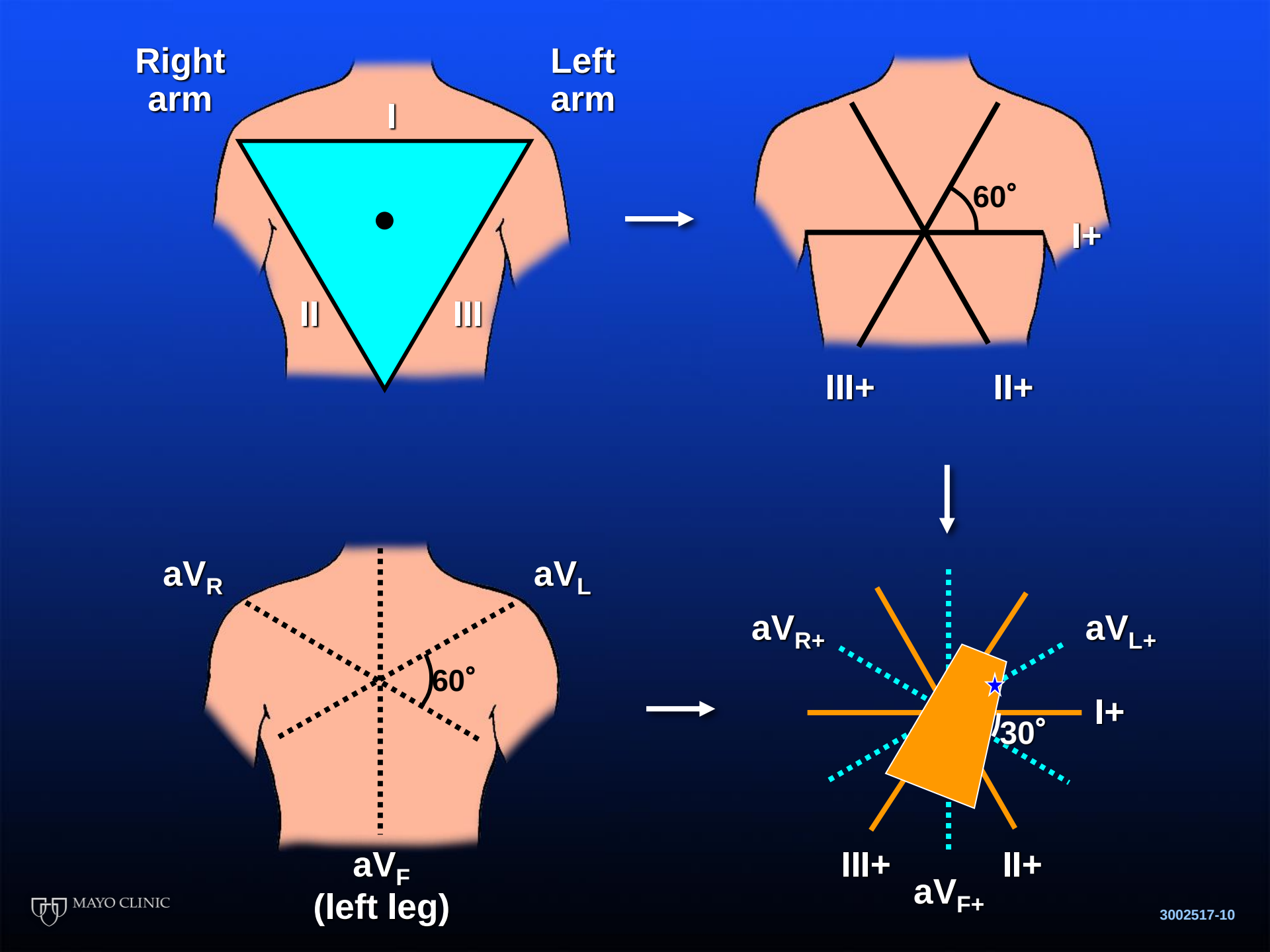
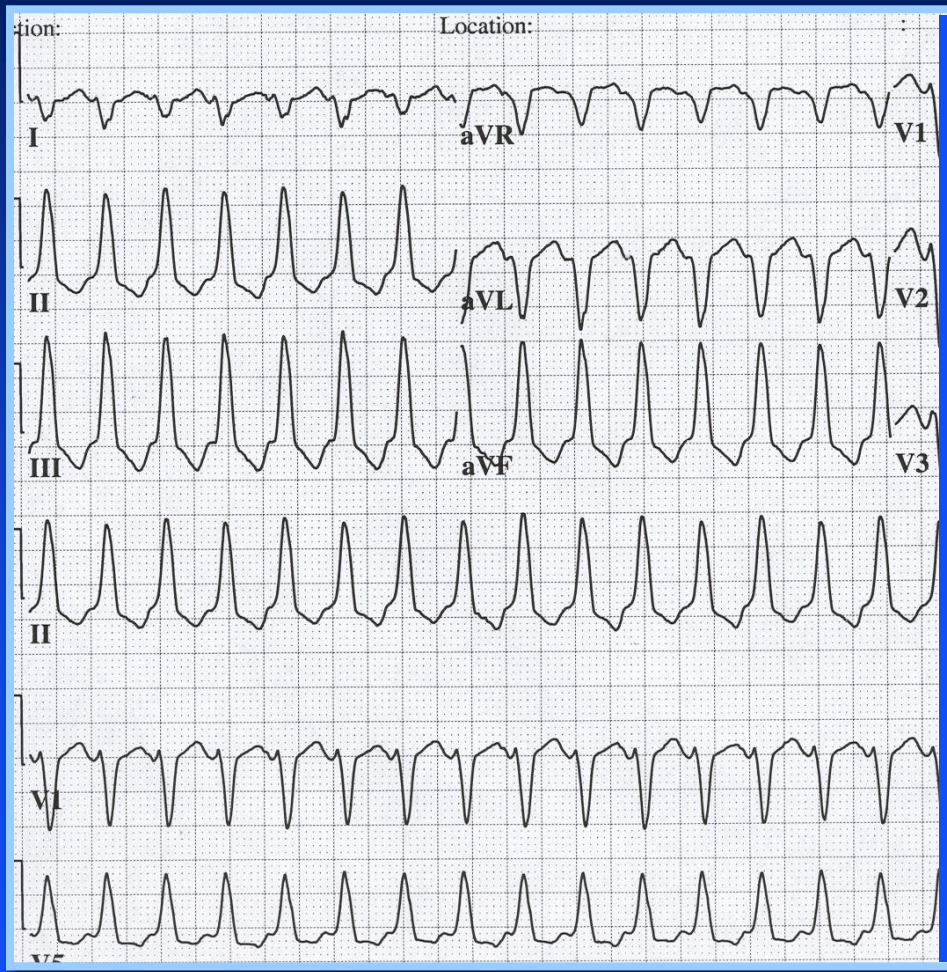
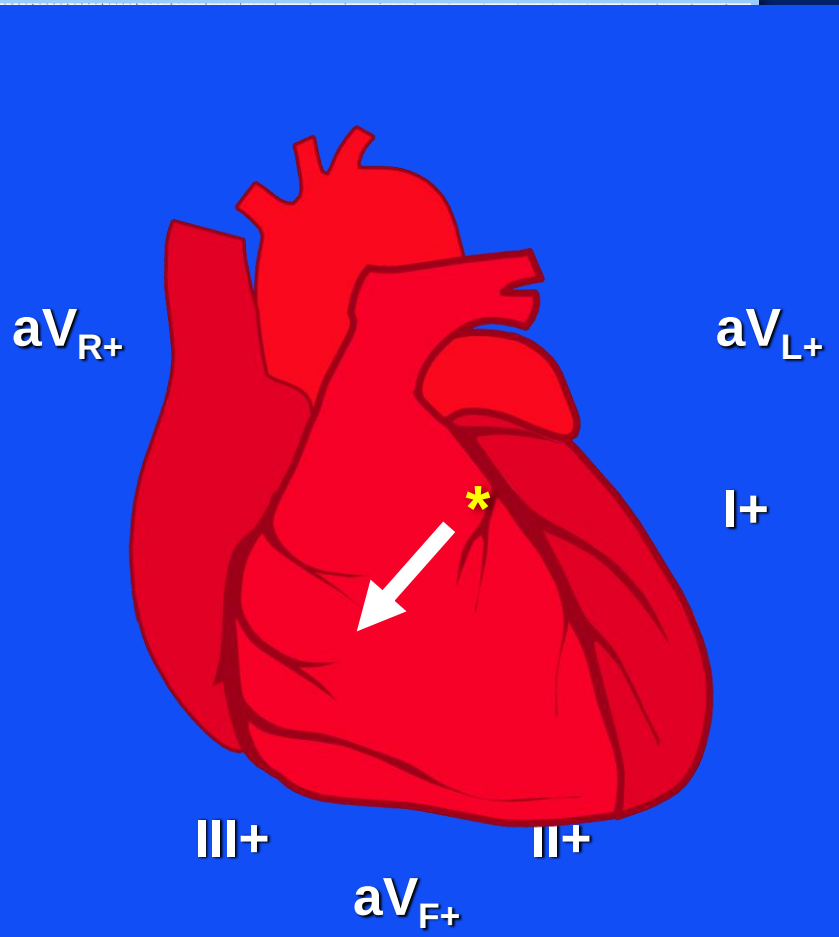

Right arm
Left arm
I
II
III
60°
I+
III+
II+
aV_R
aV_L
60°
aV_F
(left leg)
aV_R+
aV_L+
I+
30°
III+
II+
aV_F+

Septal RVOT

AVL more negative than AVR
Lead I is negative
Narrow QRS (<140)

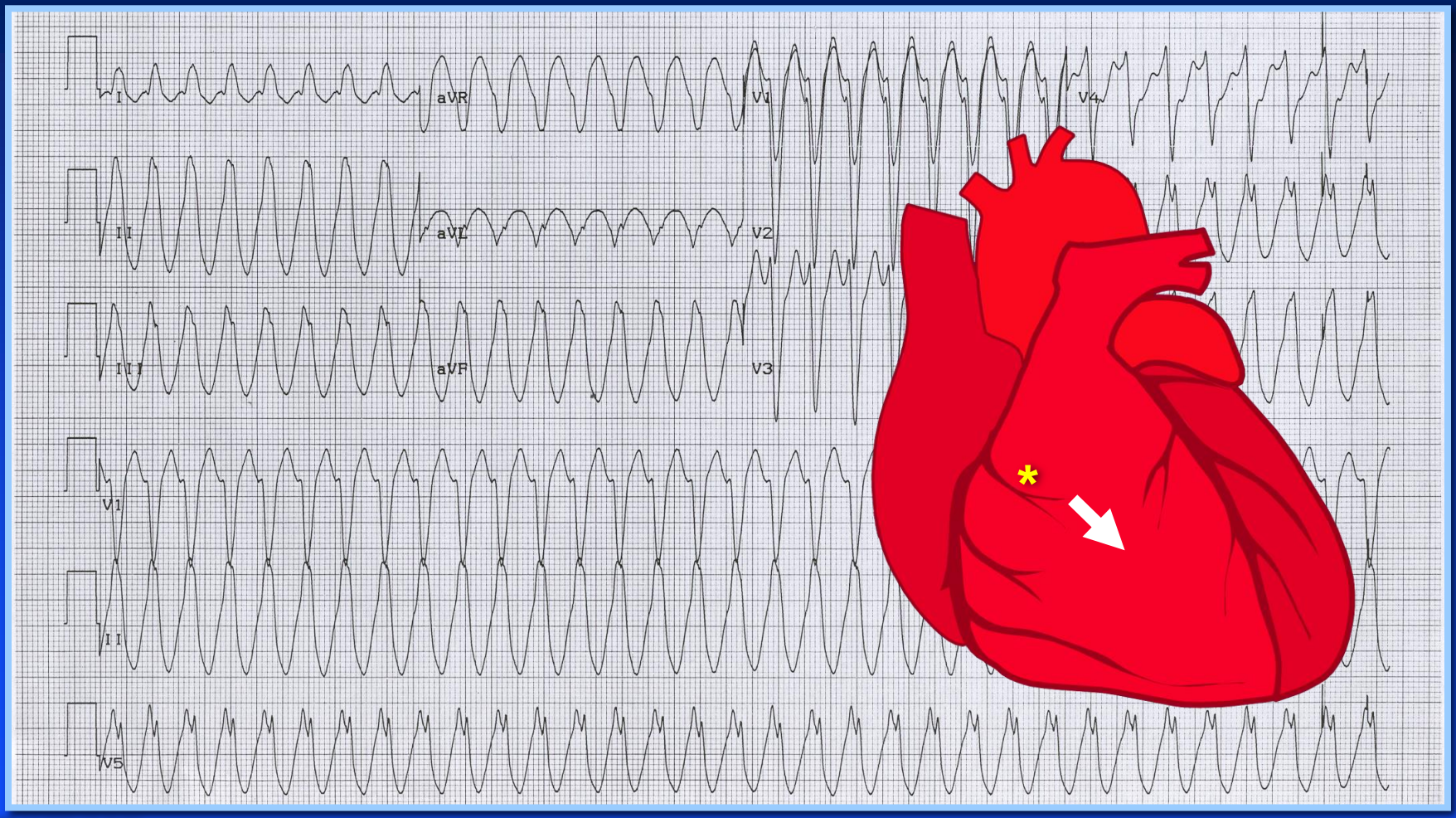




Lateral RVOT

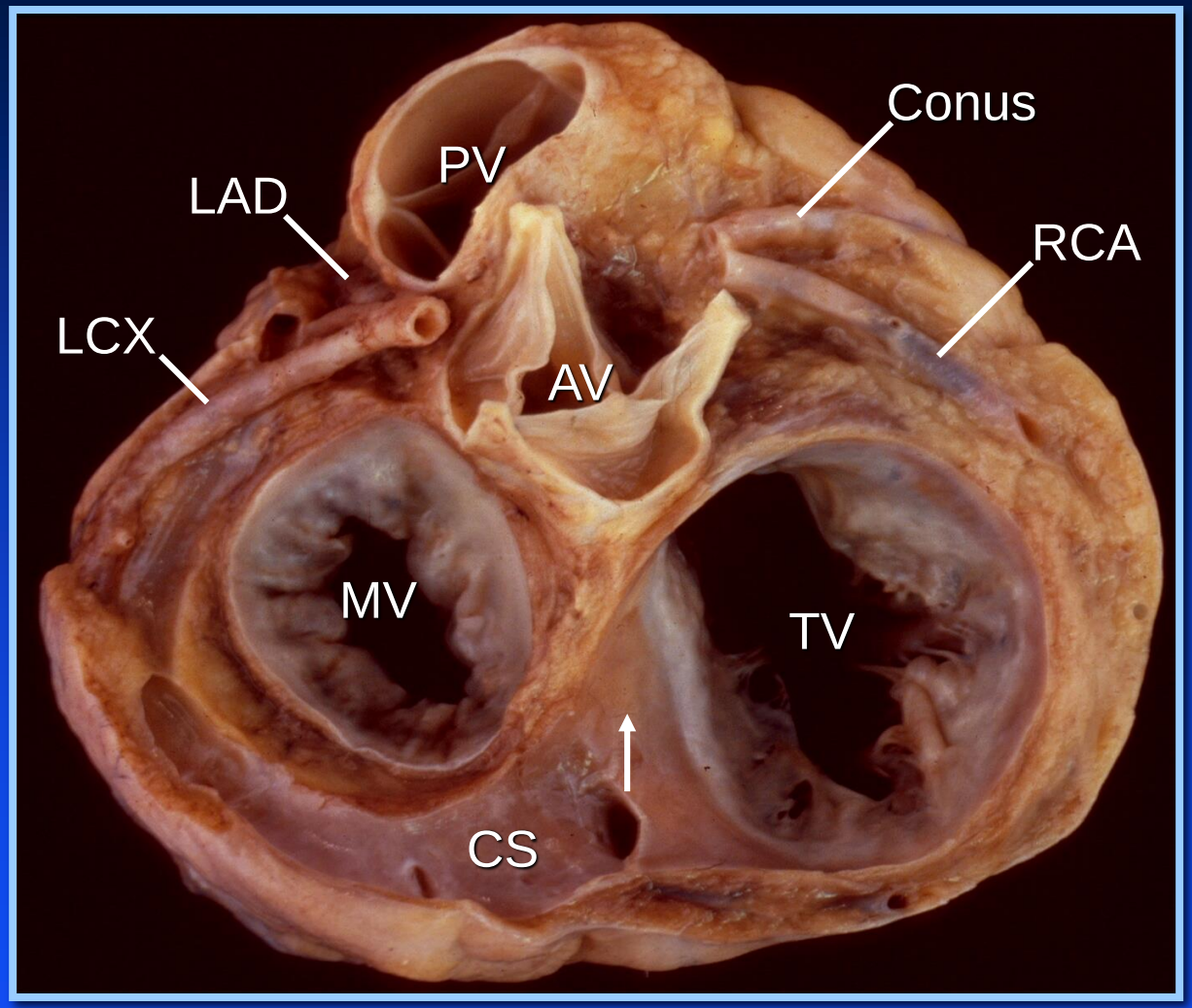
AVR more negative than AVL

QRS widerLead I is positive

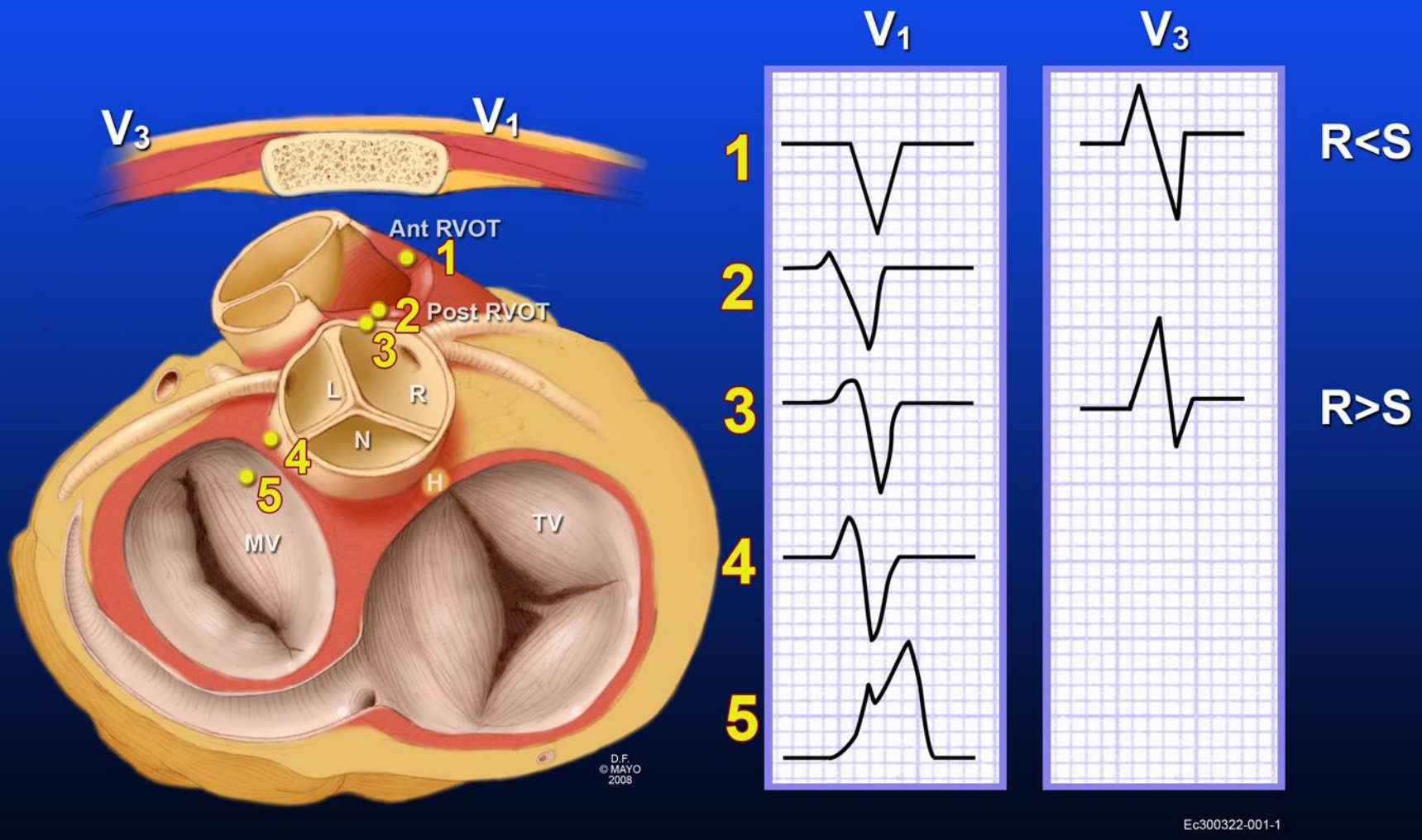


Normal Heart

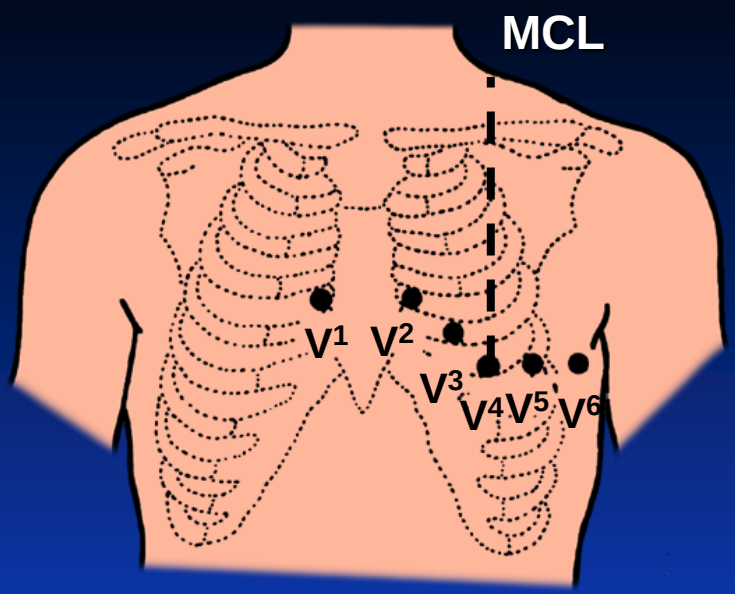
Valves & Coronary Arteries

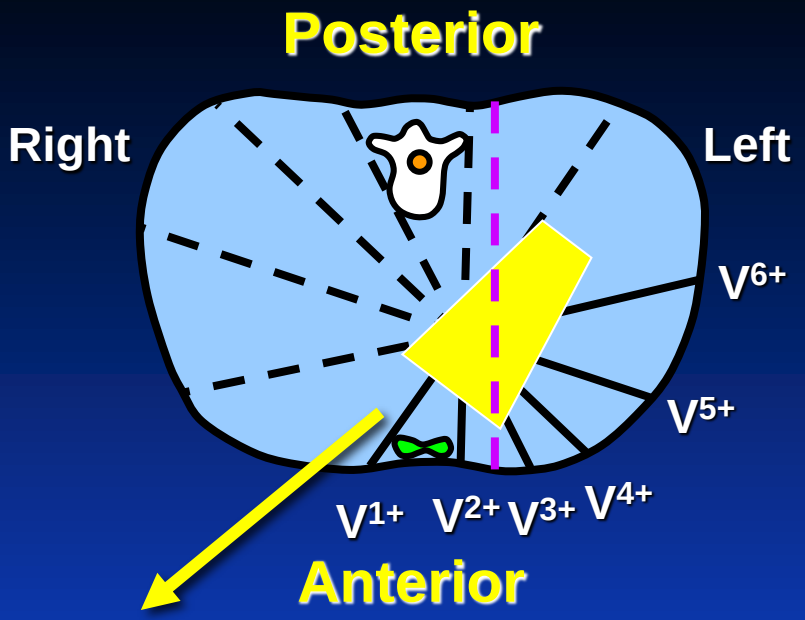


Cardiac base

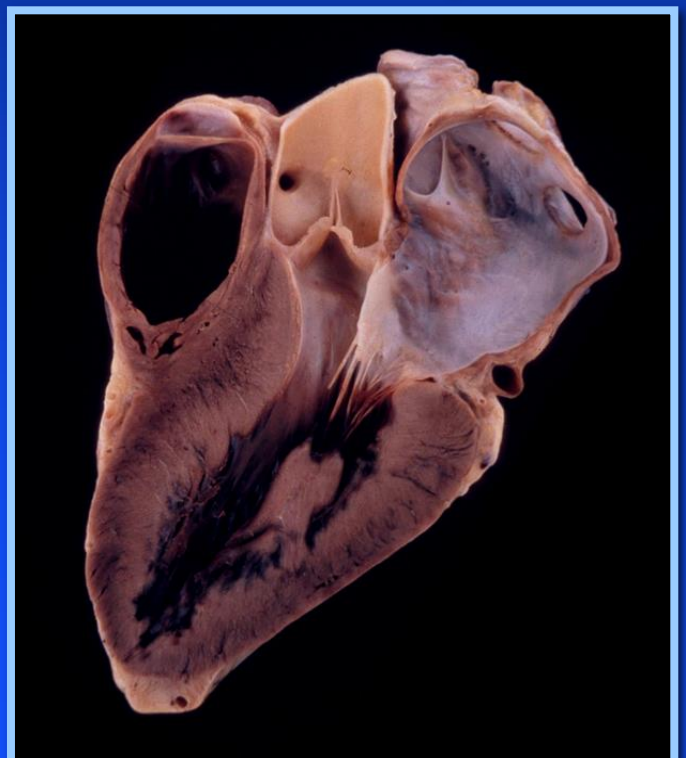


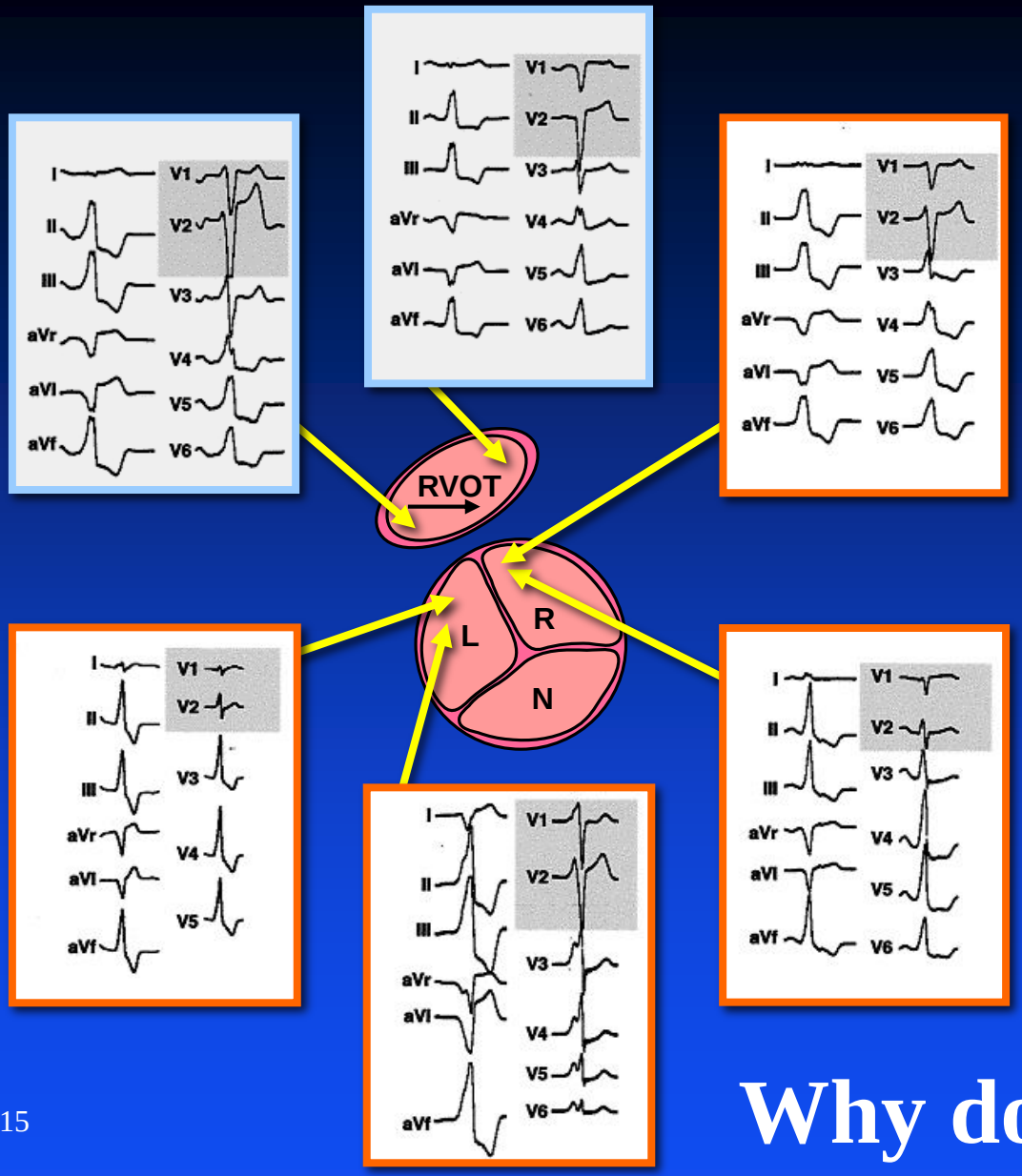
Modified from Tabetabei and Asirvatham JCE 2009


MCL
V1
V2
V3
V4
V5
V6


Posterior
Right
Left
V6+
V5+
V1+
V2+
V3+
V4+
Anterior

V1





Aortic Cusp if:
R-wave duration > 50% QRS
R/S > 30% (V1 or V2)

Transition in V3 differentiates RVOT from Aortic CUSP VT/PVC

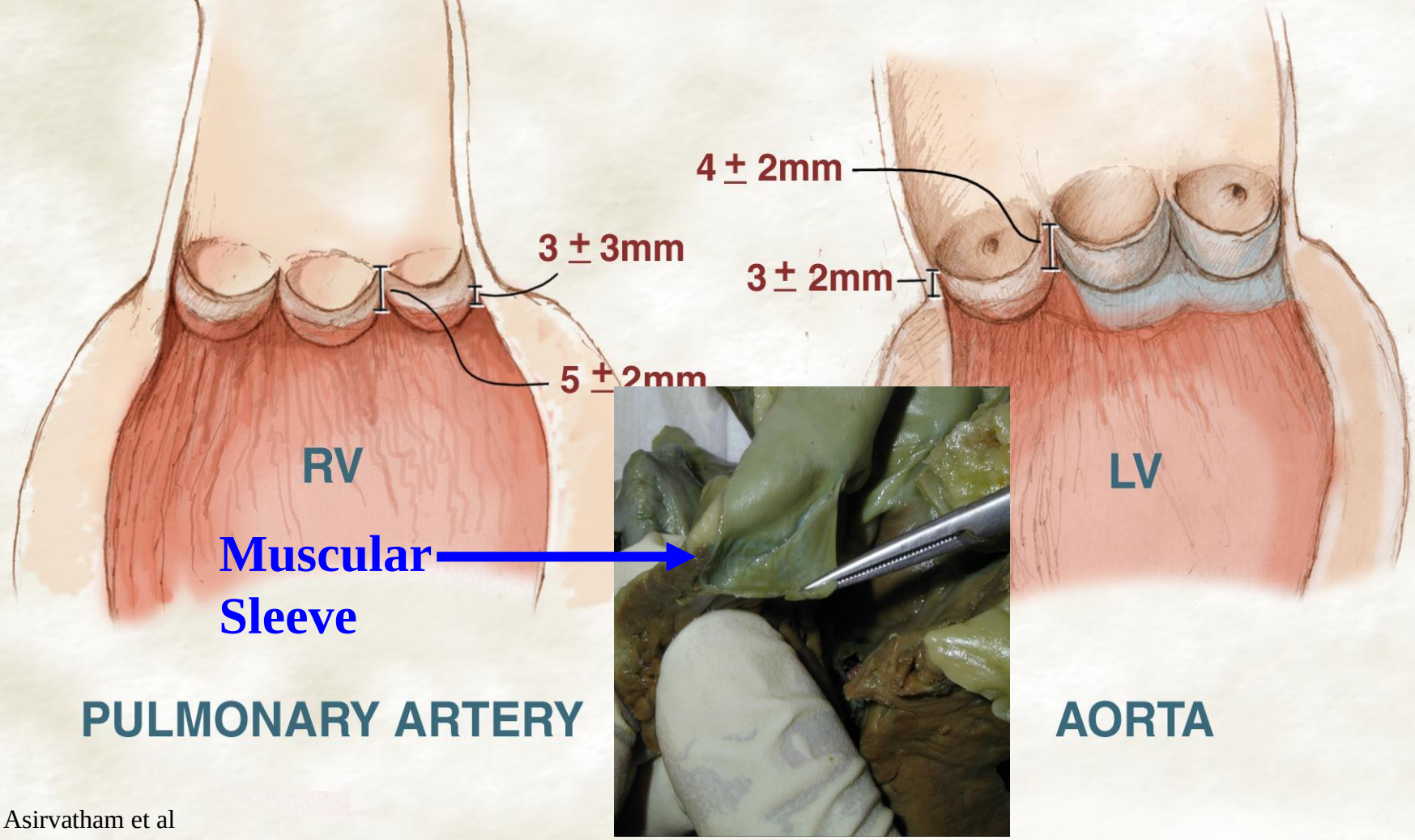
N=15

Ouyang et al: JACC 39:500, 2002

Why do PVCs/VT come From Valve cusps?

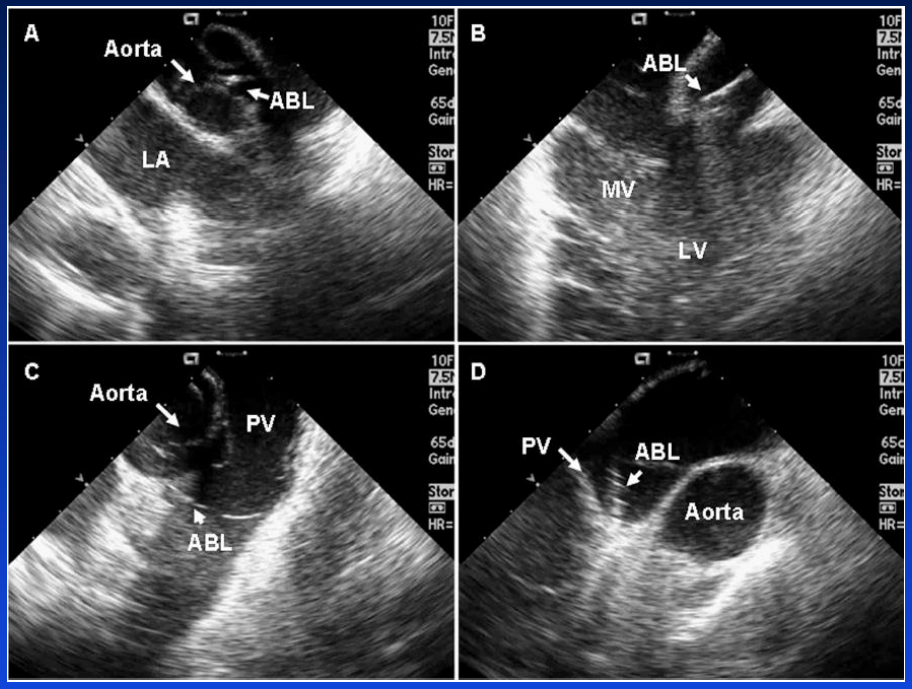
MUSCULAR EXTENSIONS INTO THE GREAT VESSELS

Are Common: Found in **360/480** (75%) Hearts Examined



Asirvatham et al

Discrete Great Arterial Potentials

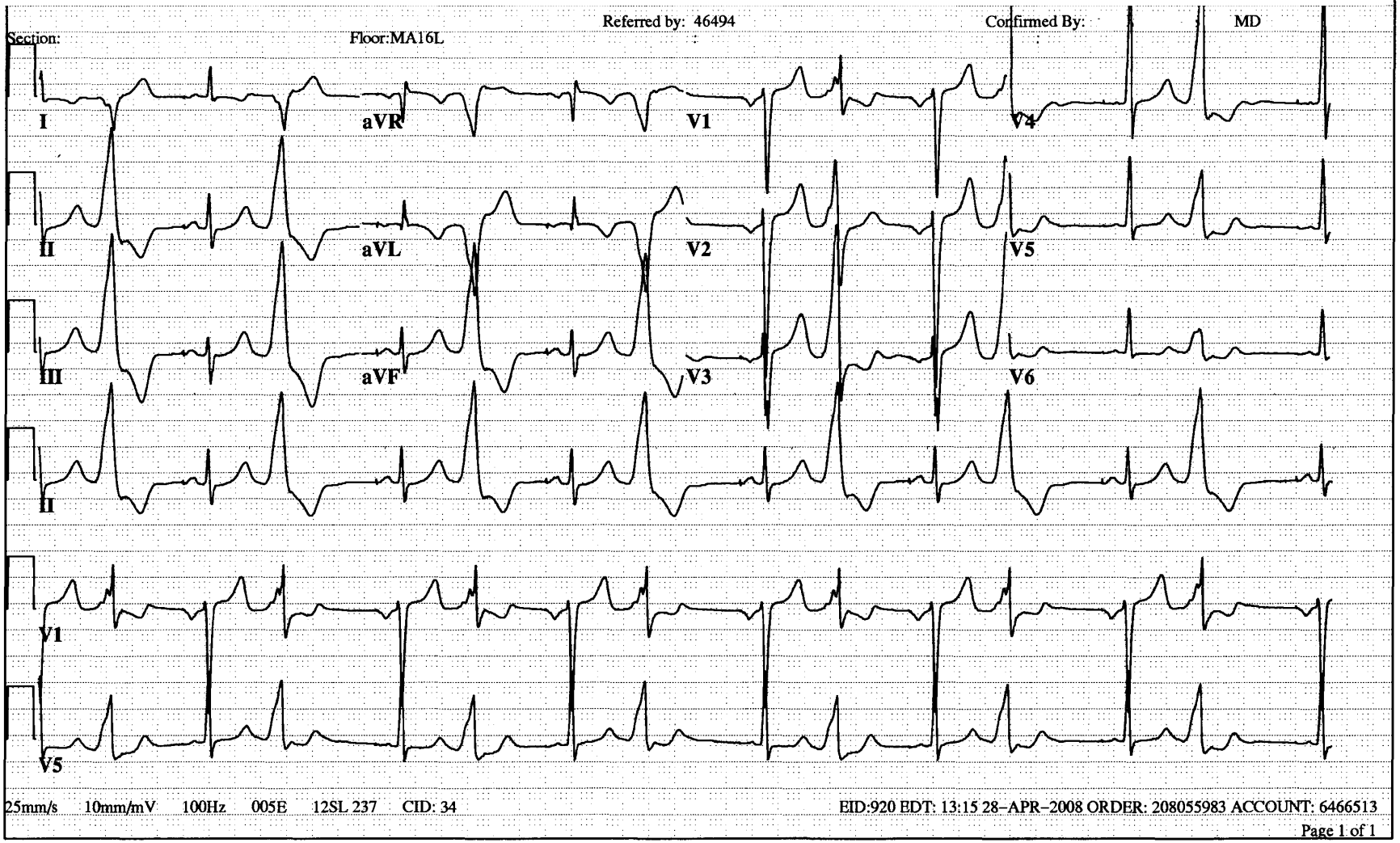


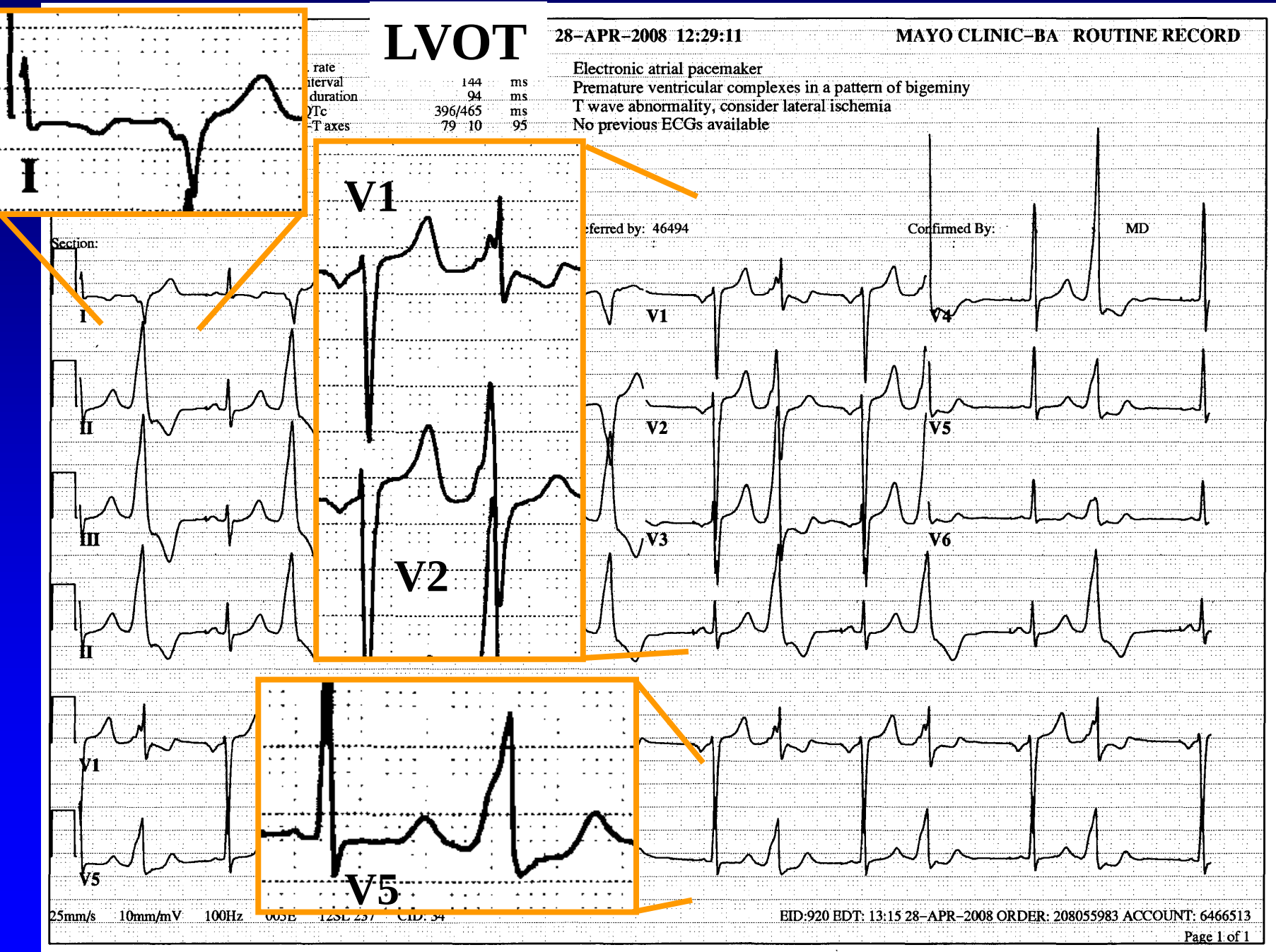
- How do we identify the potentials
- How do we confirm they are arrhythmogenic?

Srivatthsan … Shen Circ Arrhythmia EP, 2008

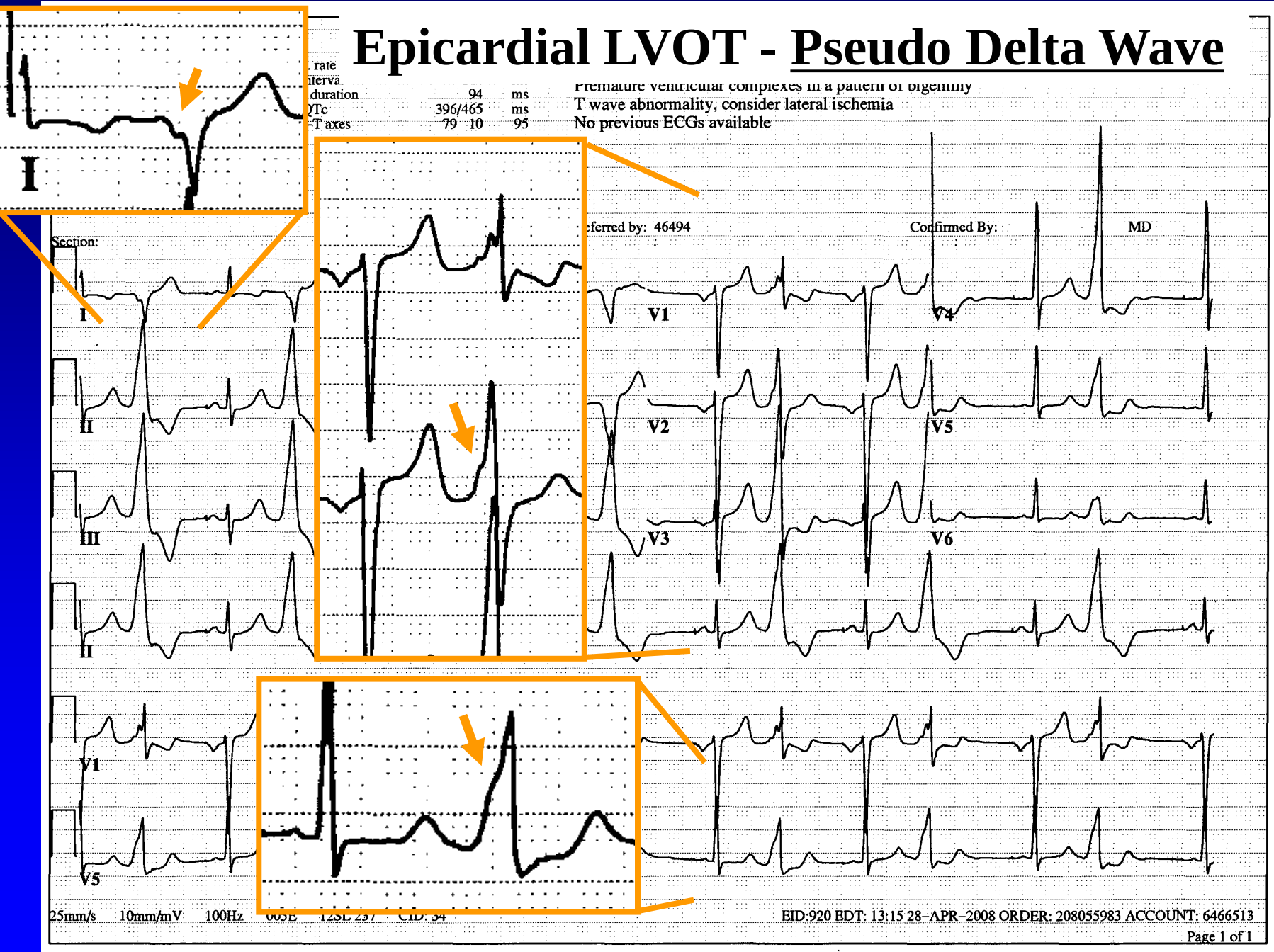
These PVCs are coming from:

1. Lateral RVOT
2. Septal RVOT
3. LVOT
4. Posterior LV



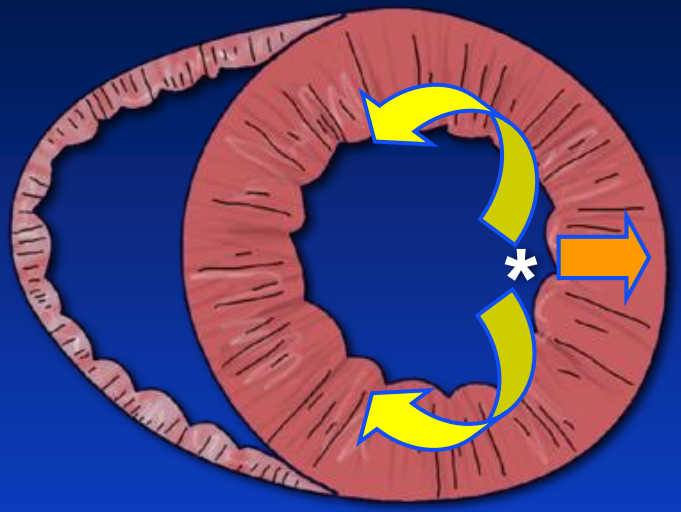

LVOT
28-APR-2008 12:29:11
MAYO CLINIC-BA ROUTINE RECORD
Electronic atrial pacemaker
Premature ventricular complexes in a pattern of bigeminy
T wave abnormality, consider lateral ischemia
No previous ECGs available
rate
144 ms
duration
94 ms
QTc
396/465 ms
P-T axes
79 10 95
Referred by: 46494
Confirmed By:
MD
Section:
I
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6
25mm/s
10mm/mV
100Hz
EID:920 EDT: 13:15 28-APR-2008 ORDER: 208055983 ACCOUNT: 6466513
Page 1 of 1

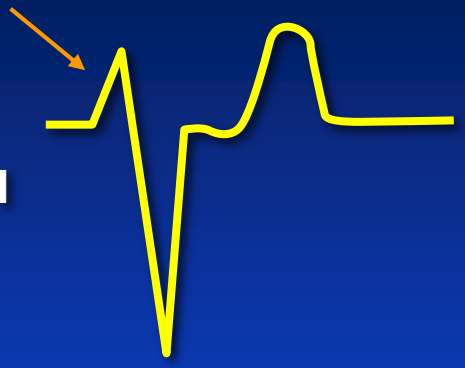
Epicardial LVOT - Pseudo Delta Wave



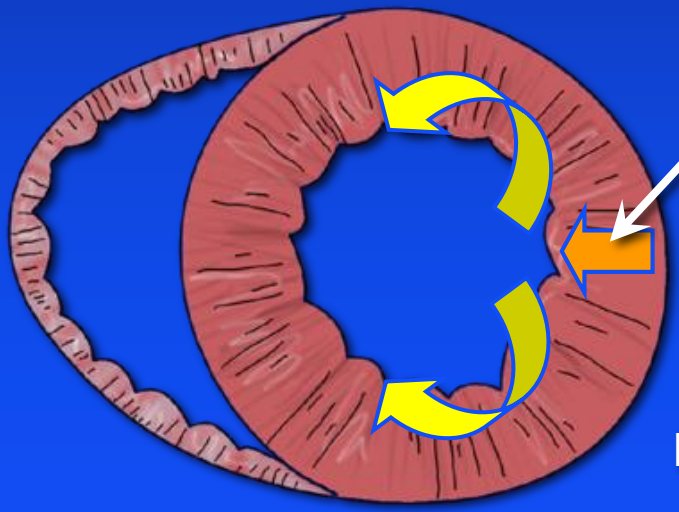
ECG and VT Source

Endocardial

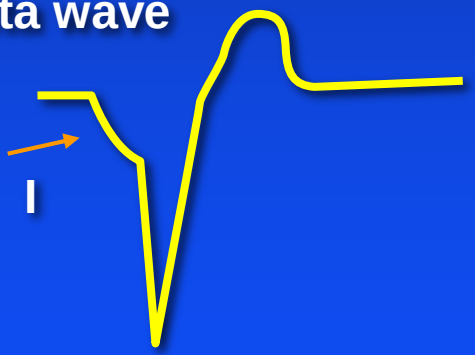




Epicardial



Cause of pseudo delta wave



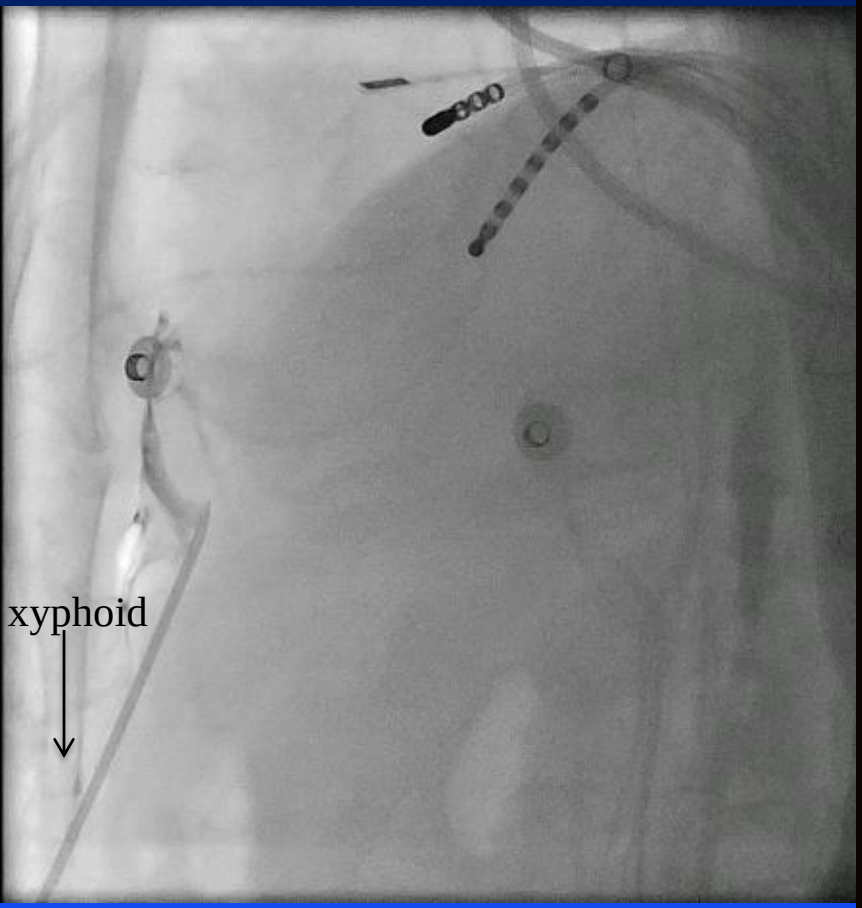
Pseudo delta wave

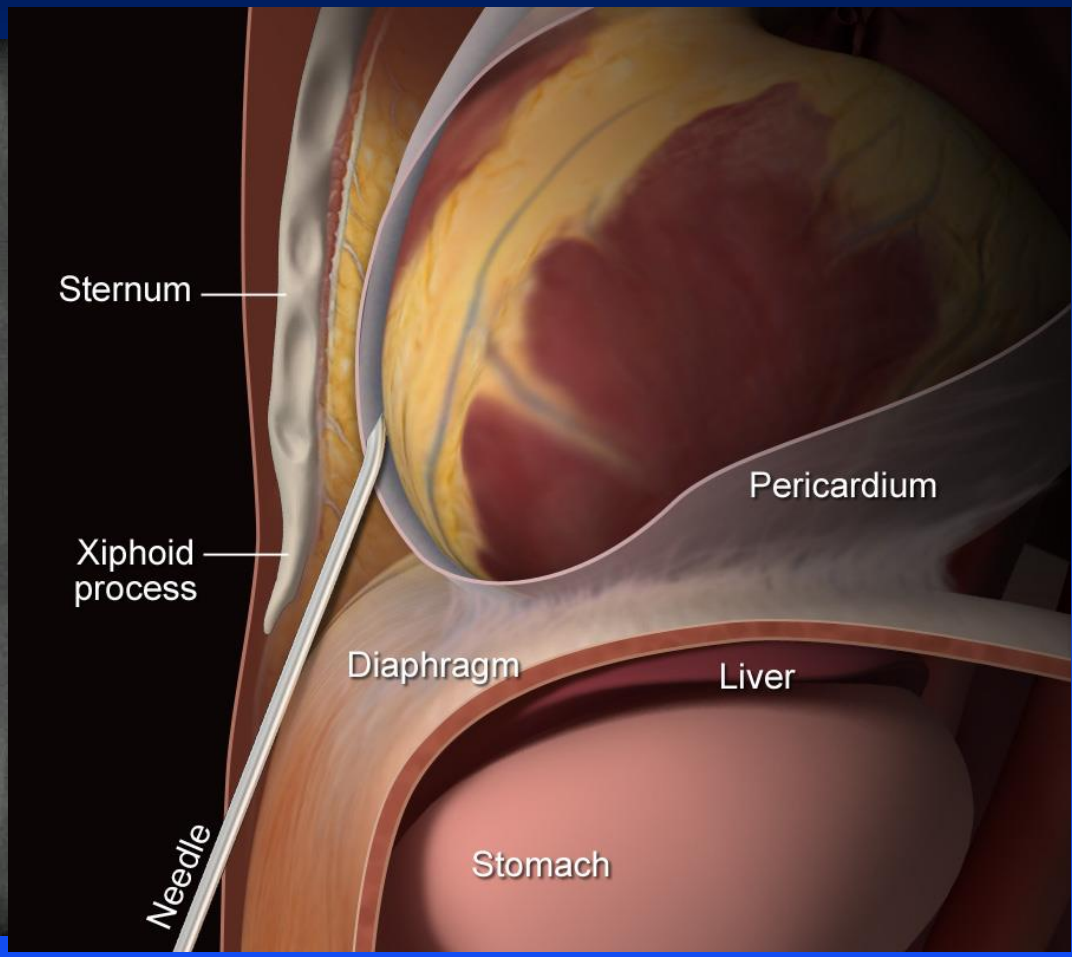
Pseudo delta >34ms: sens 83%, spec 95%

Berruezo Circ 2004

Lateral View – Percutaneous epicardial access

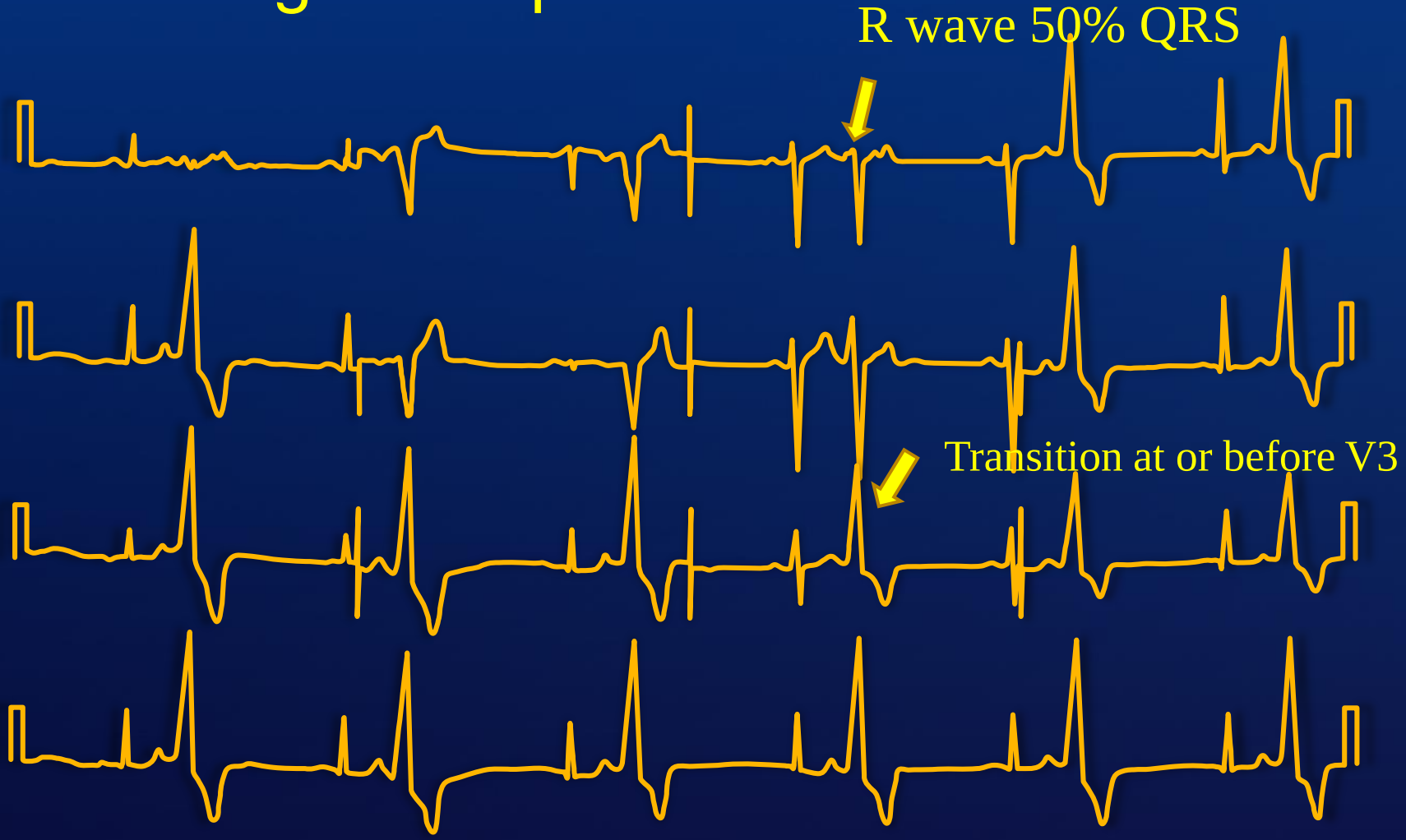
head





feet

Returning to our patient…



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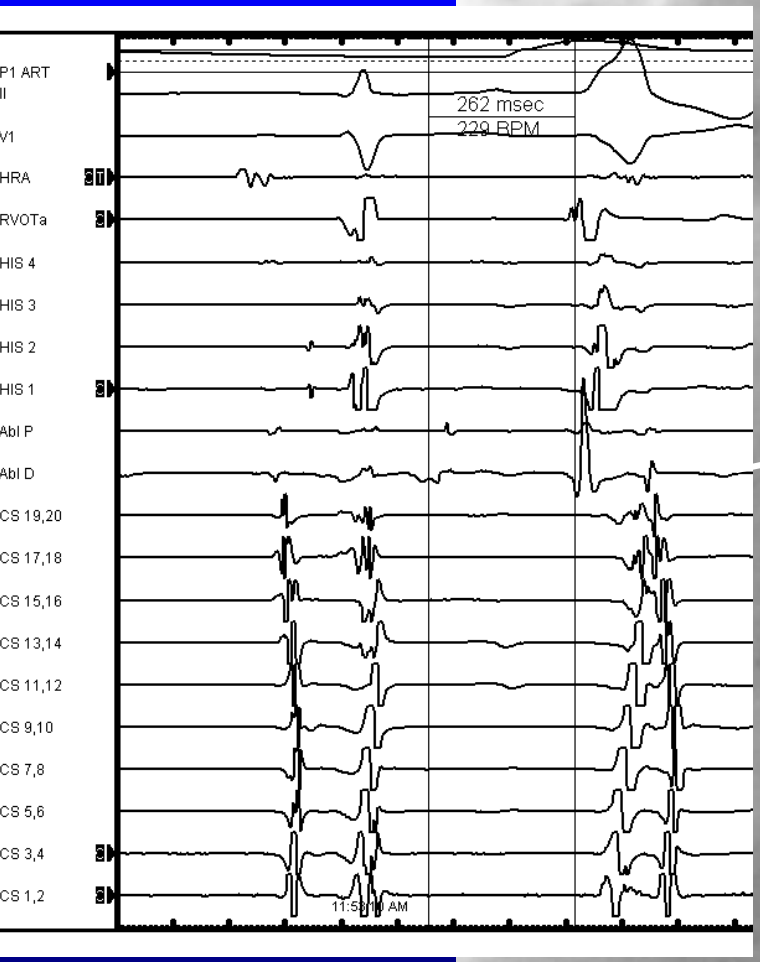
Aortic Valve
Cusp > -200 mse



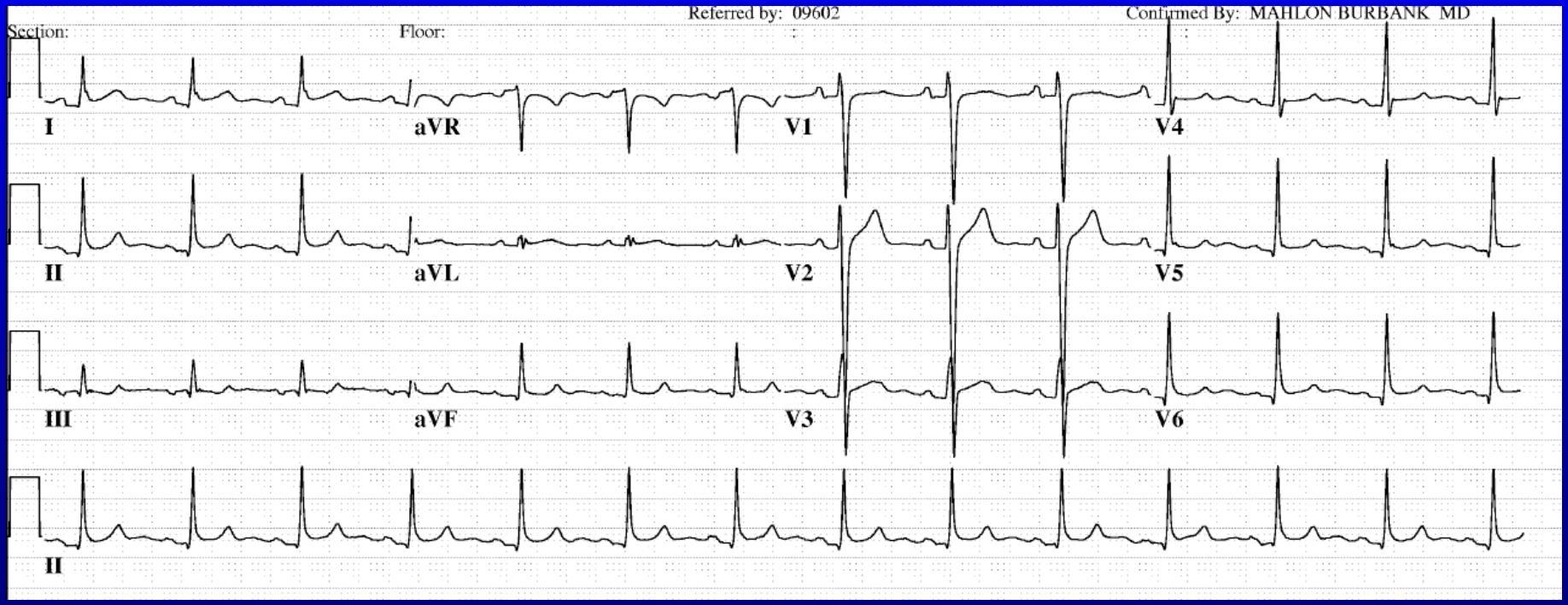
RVOT -25

Epicardial -35

DCS -30

Bracketing Complex Anatomy

Post Procedure: No Ectopy
Predismissal echo: EF 47%
Echo 2 yrs later: EF 50-65%



Key Points

- Just as smooth muscle in pulmonary veins is associated with AF, muscle in great arteries associated with ventricular arrhythmia

- Frequent PVCs can cause “tachycardia induced” cardiomyopathy

Take Home Points

1. Rule out structural disease
2. If PVCs unifocal → may be PVC CM
   - 12 lead Holter helpful
   - >20K PVCs/day (RBBB) or >10K PVC (LBBB) → increased risk
3. Progression is slow; if EF normal can follow clinically (Holter, echo)

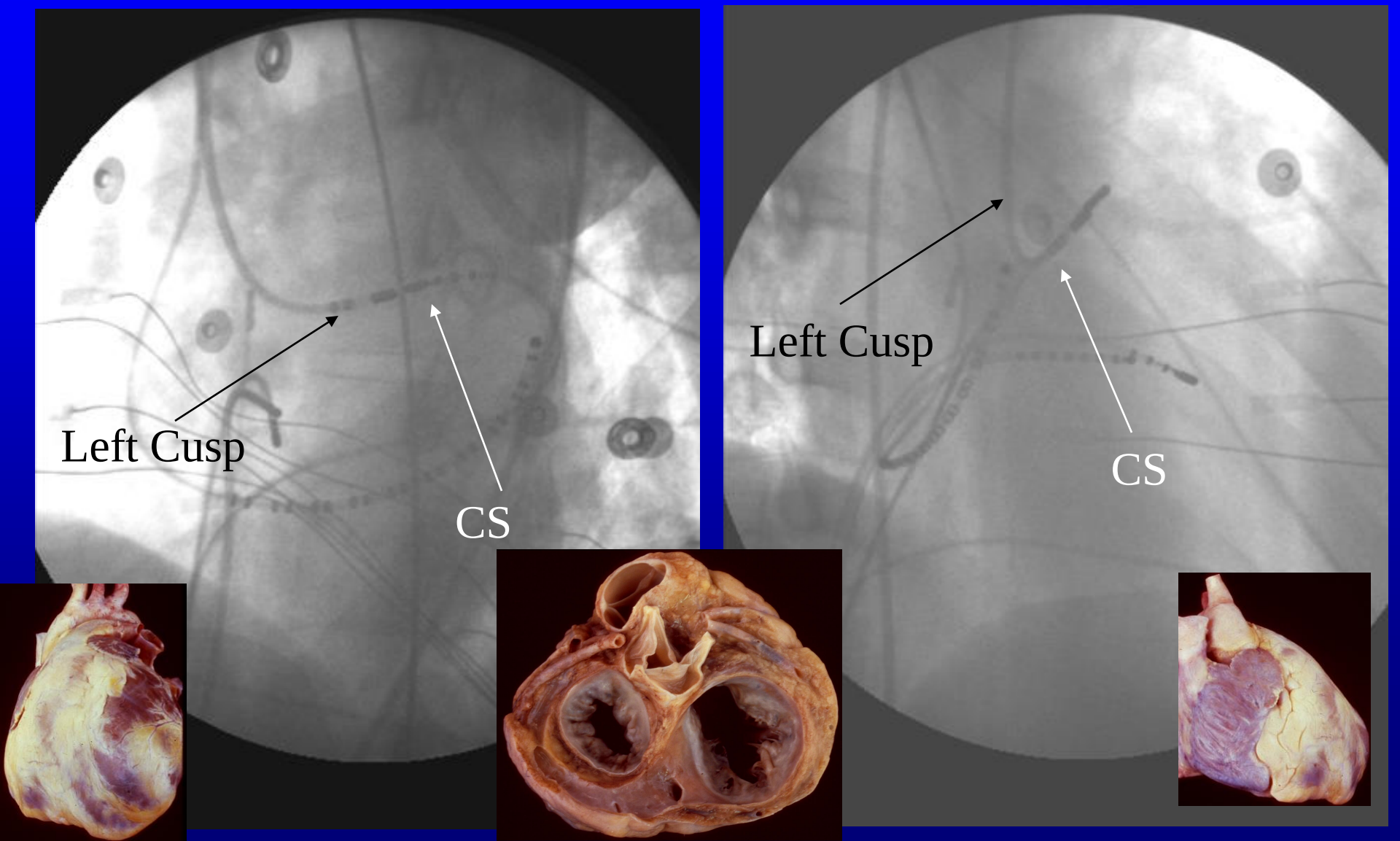
Thank You

Conclusions

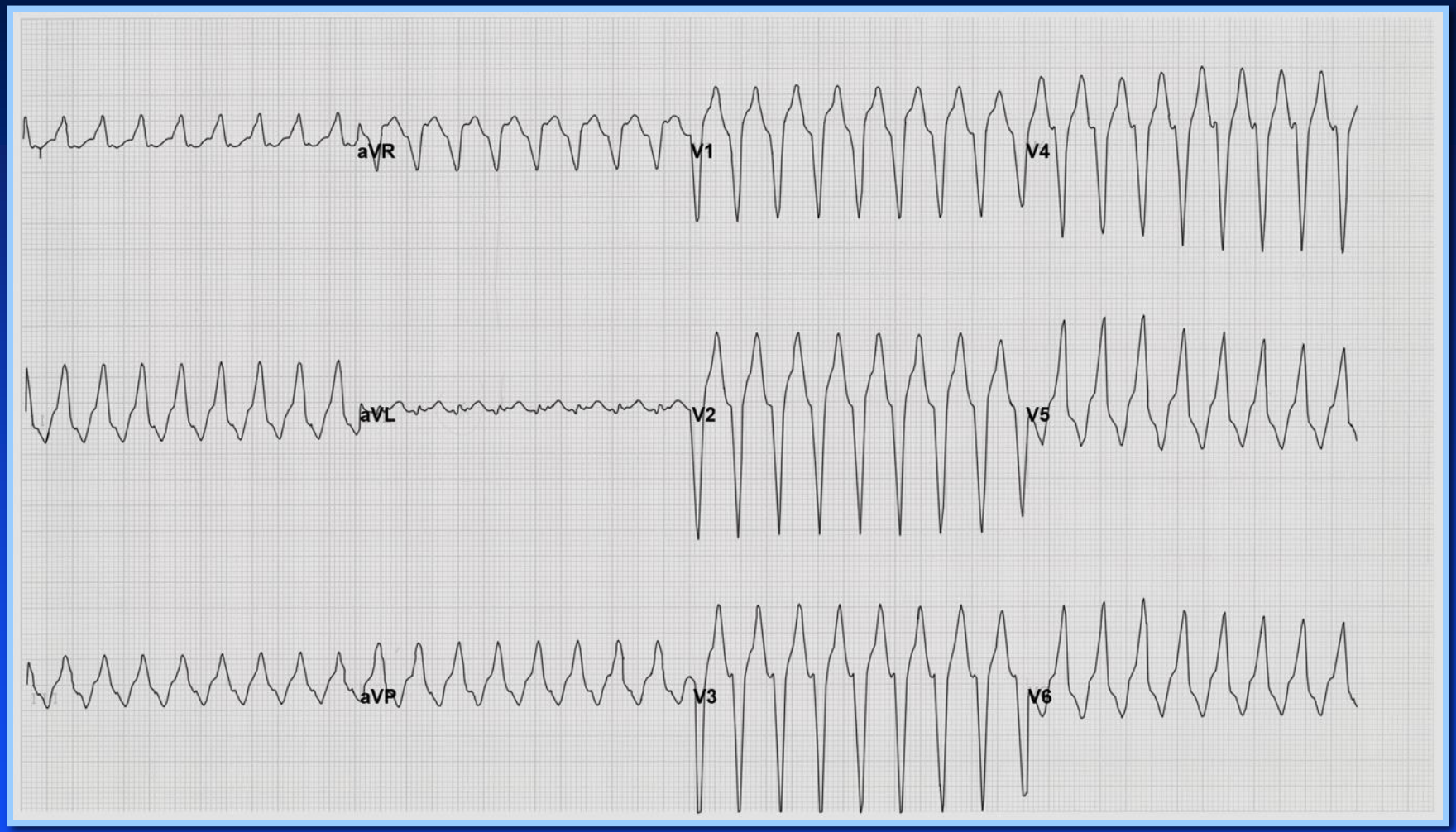
Mapping in Left Coronary Cusp (not our case)

LAO

RAO



AVL positive – near His

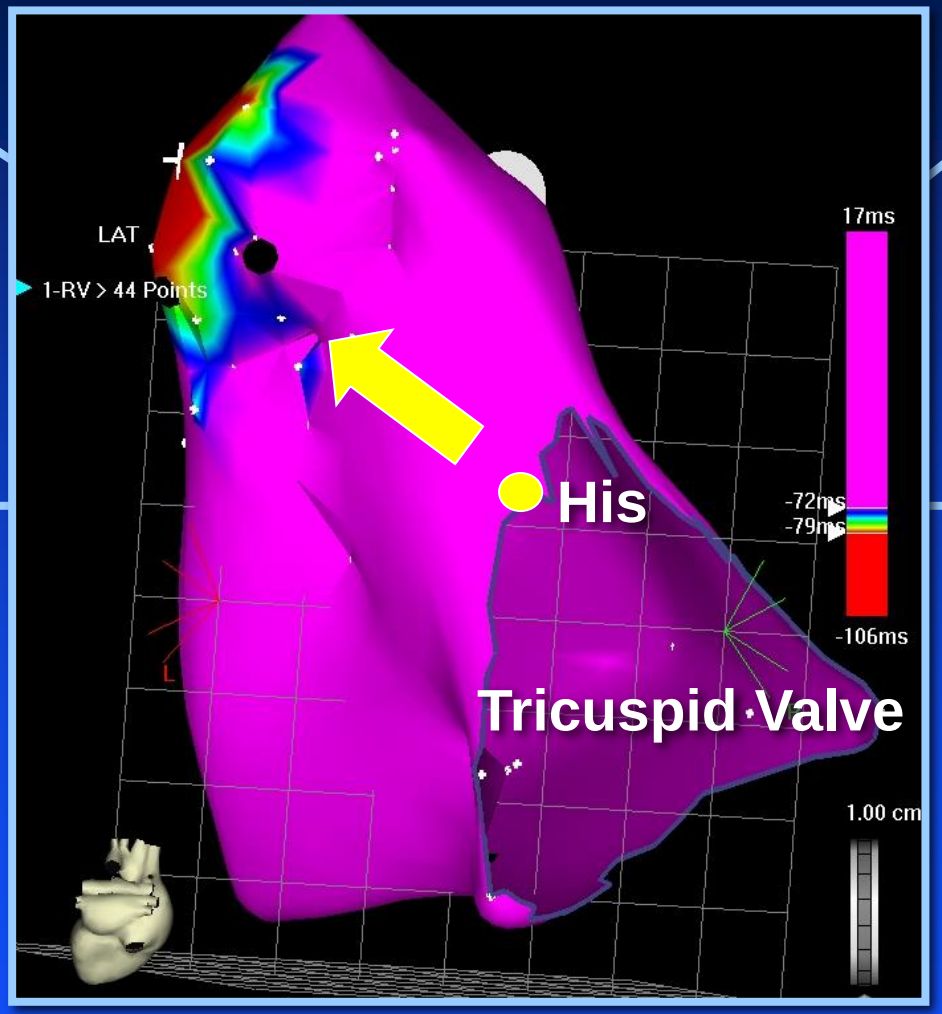


aVL "+" : near His bundle

aVL

aVR

I



(posterior view)

Summary of Proposed ECG Criteria

- Psuedo delta wave (≥35 msec) (CM)
- Delayed intrinsicoid deflection (>85 msec to V2 peak) or RS >121 msec (CM)
- MDI >0.55 (idiopathic VT, GCV)
- Q waves in region opposite origin (CM)

Substrates vary and experience limited; ECG is guide and useful adjunct, but is location specific and has important limitations

Berruezo: Circ, 2004
Bazan: Heart Rhythm, 2006
Bazan: Heart Rhythm, 2007
Daniels: Circ, 2006