CardioVascular Institute





A teaching hospital of Harvard Medical School

A Difficult case of VT Venice Arrhythmias 2015 Discussant: Andrea Natale

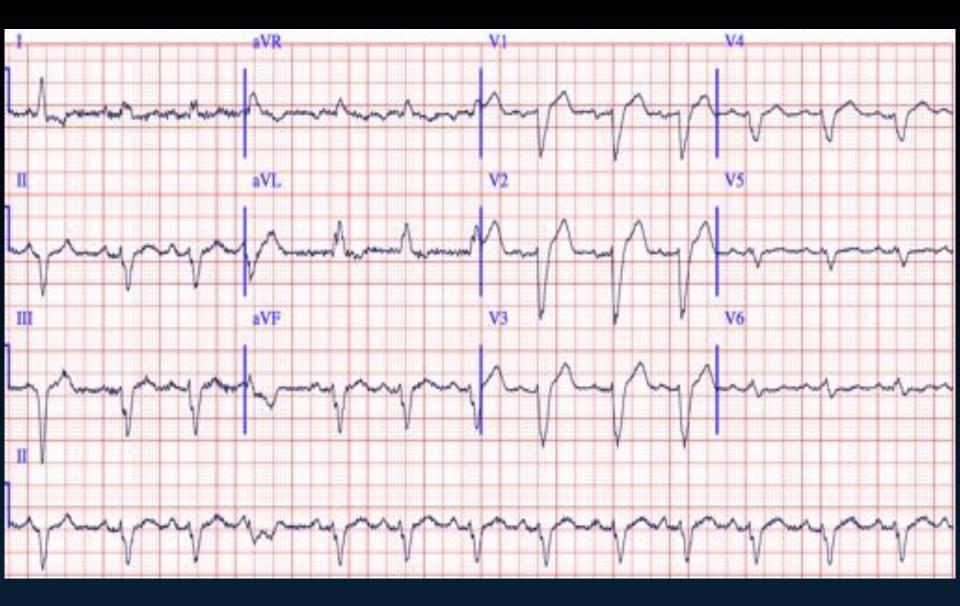
Elad Anter, MD

Director, Experimental Electrophysiology Laboratory Associate Director, Clinical Electrophysiology Laboratory Assistant Professor of Medicine, Harvard Medical School Beth Israel Deaconess Medical Center – Boston, MA

Clinical Presentation

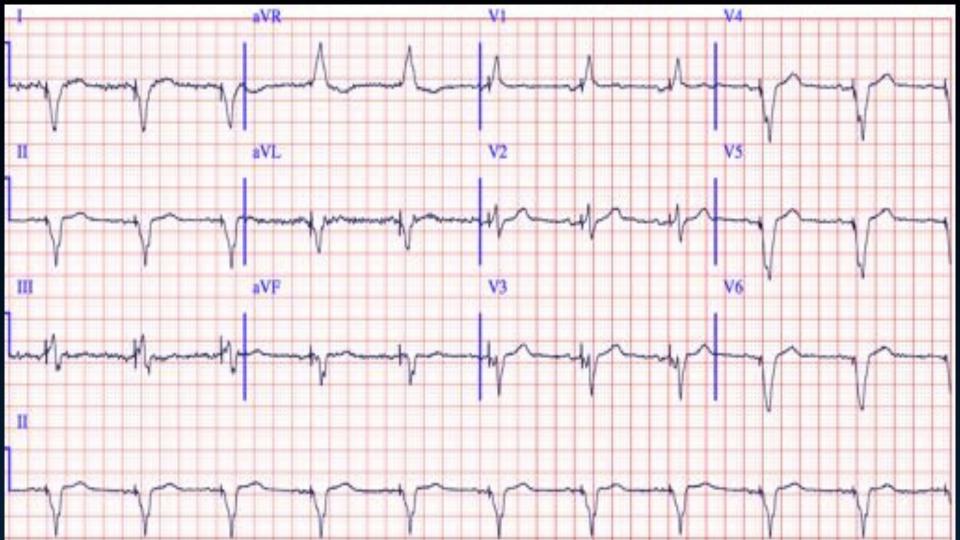
- 67-year-old man
- Chronic mixed cardiomyopathy (LVEF 25%, NYHA II)
 - LAD infarct 1996 (LVEF 45%; NYHA I)
 - 10 years later developed progressive LV dysfunction
 - ➢ LVEF from 45 -> 25% and new LBBB
 - VF arrest in 2012 -> CRT-D
 - Recurrent MMVTs requiring ICD shocks (>3 distinct EGMs)
 - Continue to have sustained VTs on chronic amiodarone therapy (400 daily)

Baseline ECG

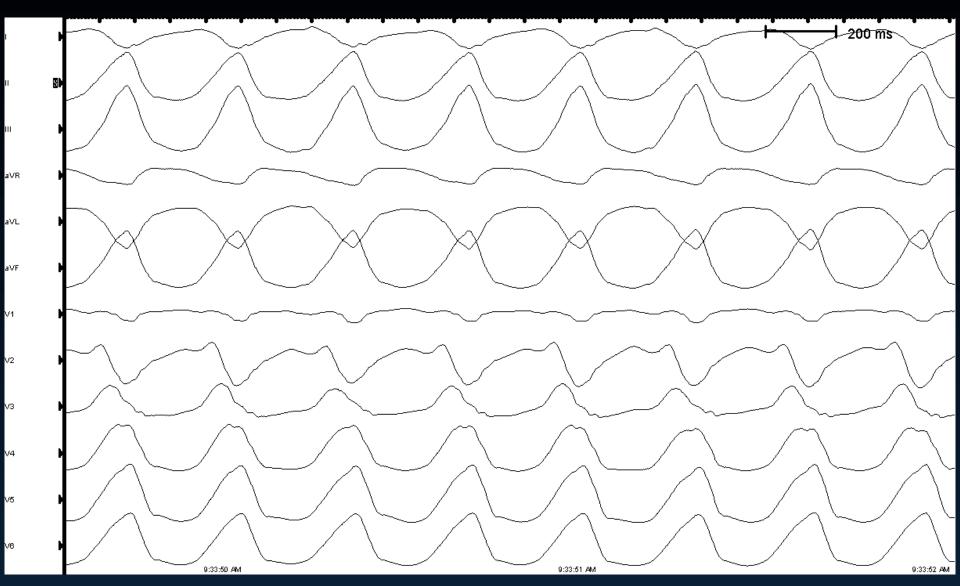


ECG after CRT

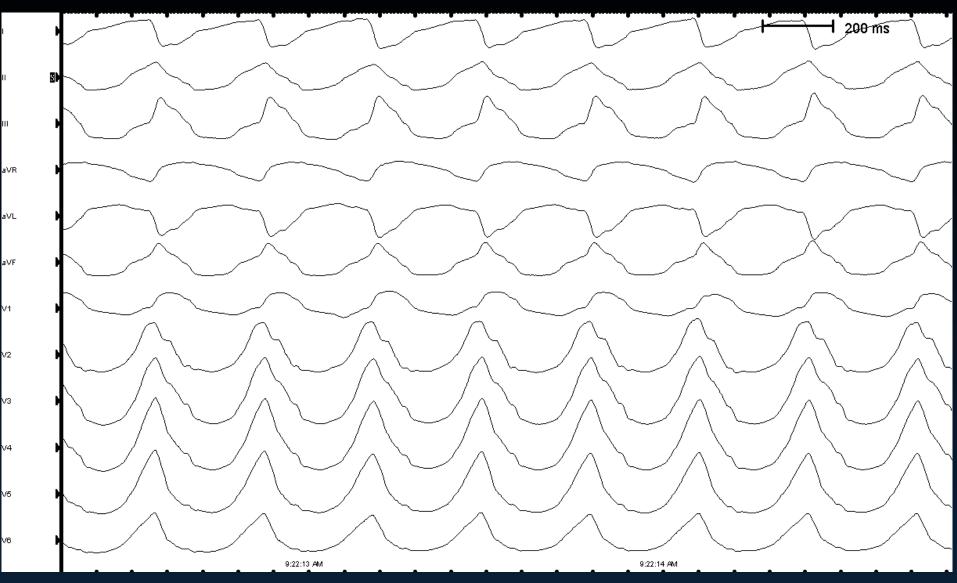
(No change in HF symptoms)



VT-1 induced with ES from RVA (TCL 320ms)

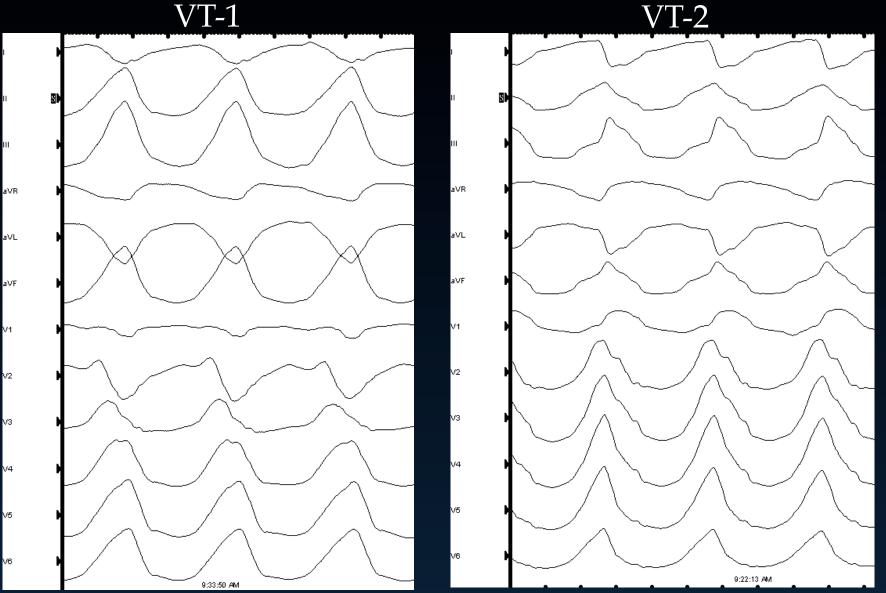


VT-1 induced with ES from RVA (TCL 305ms)

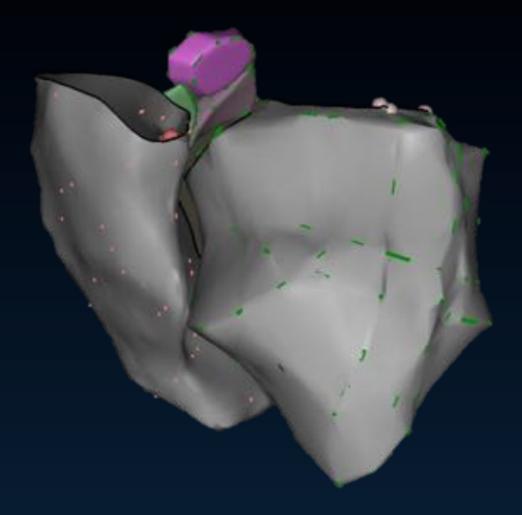


Two inductions with 2 MMVTs

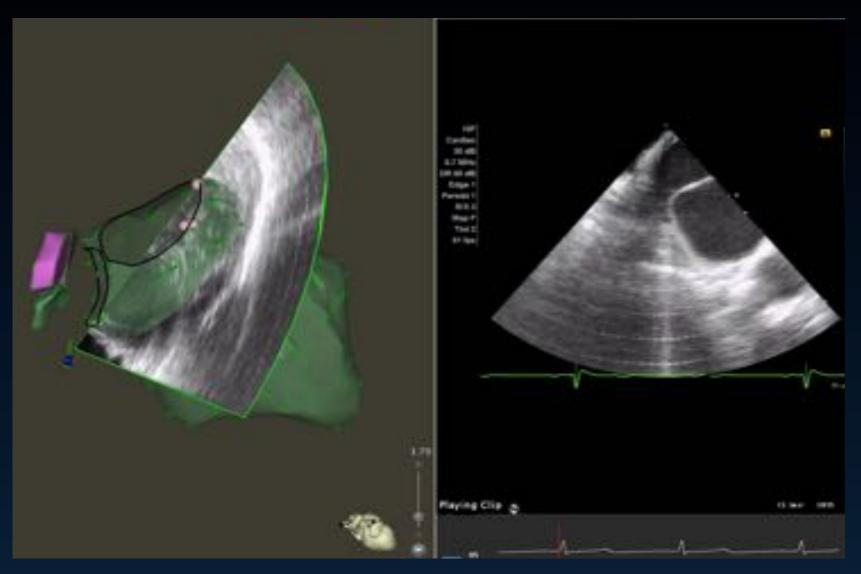
(Both non-tolerated, failed ATP – DCCV)



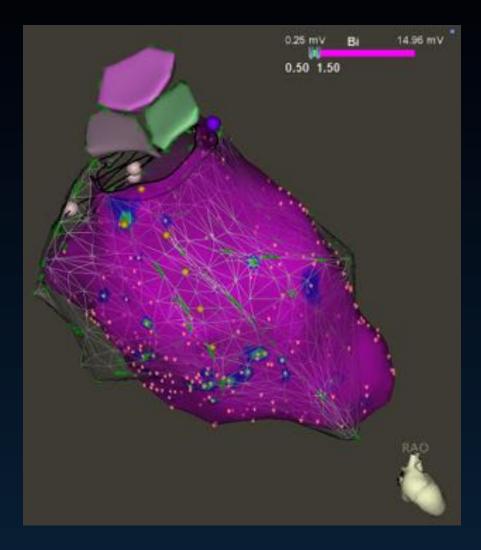
Carto Sound shell

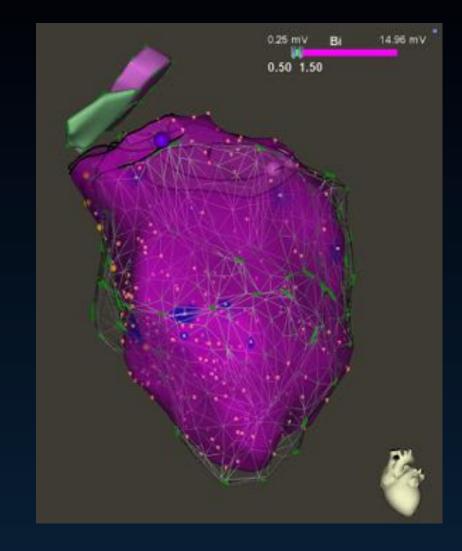


Carto Sound shell (focus on ventricular OT and AMC)

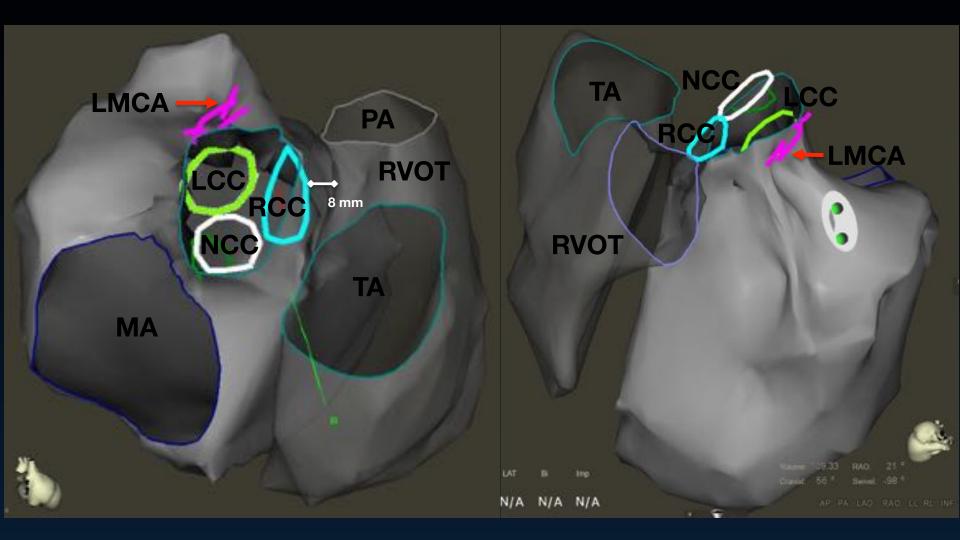


Carto Sound Shell

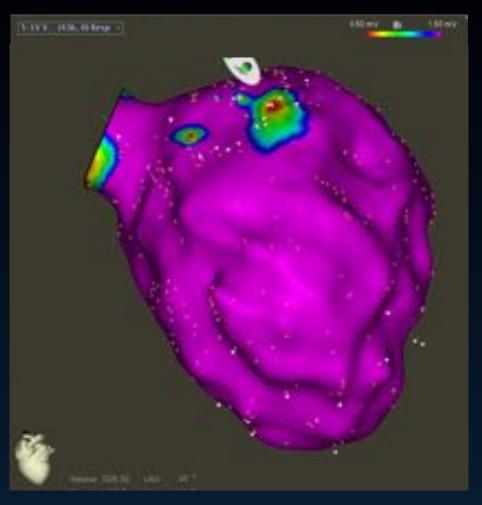


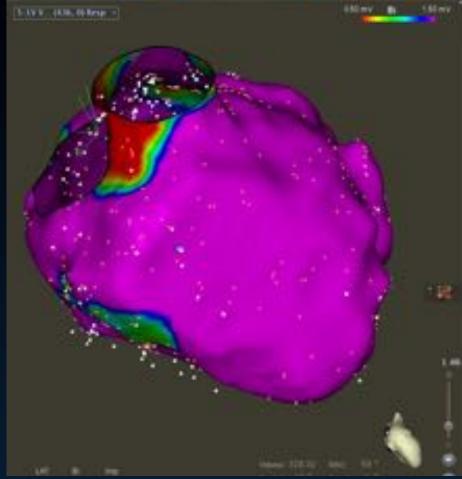


Carto Sound Shell

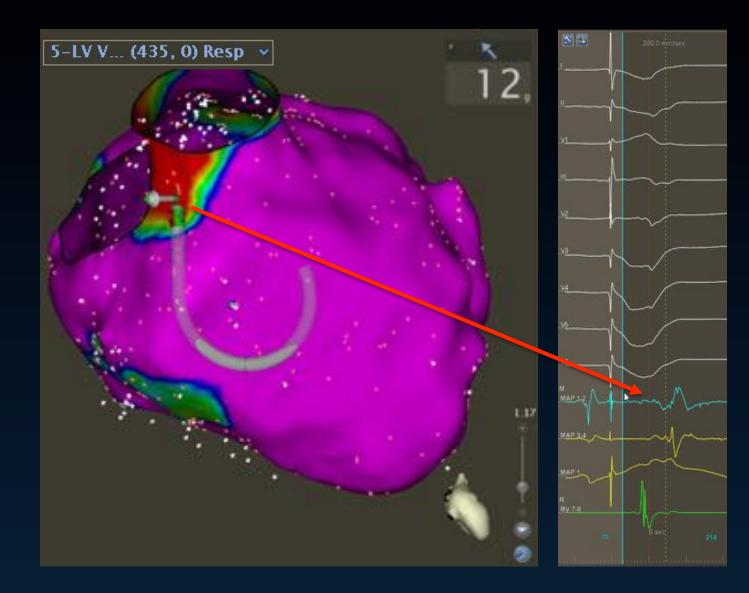


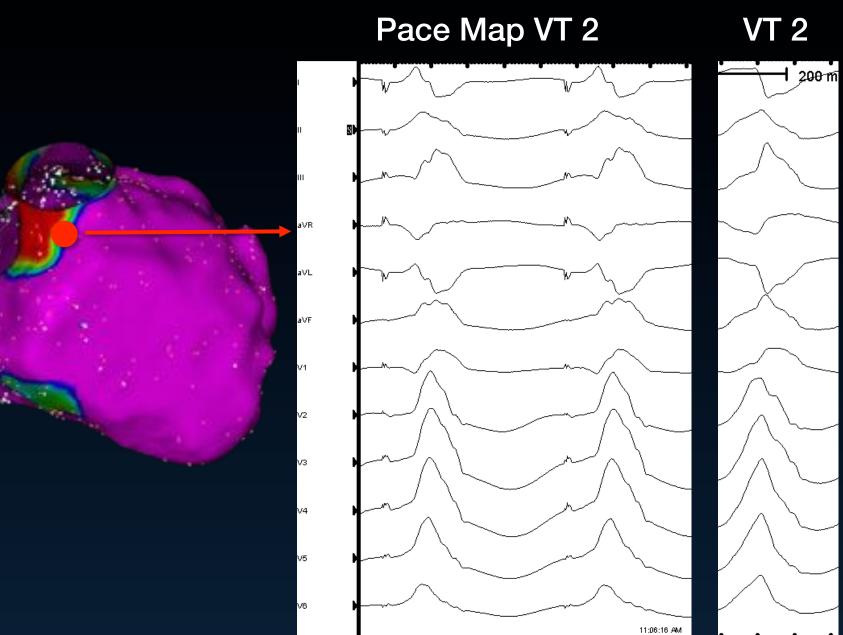
LV Endocardial Bipolar Voltage Map

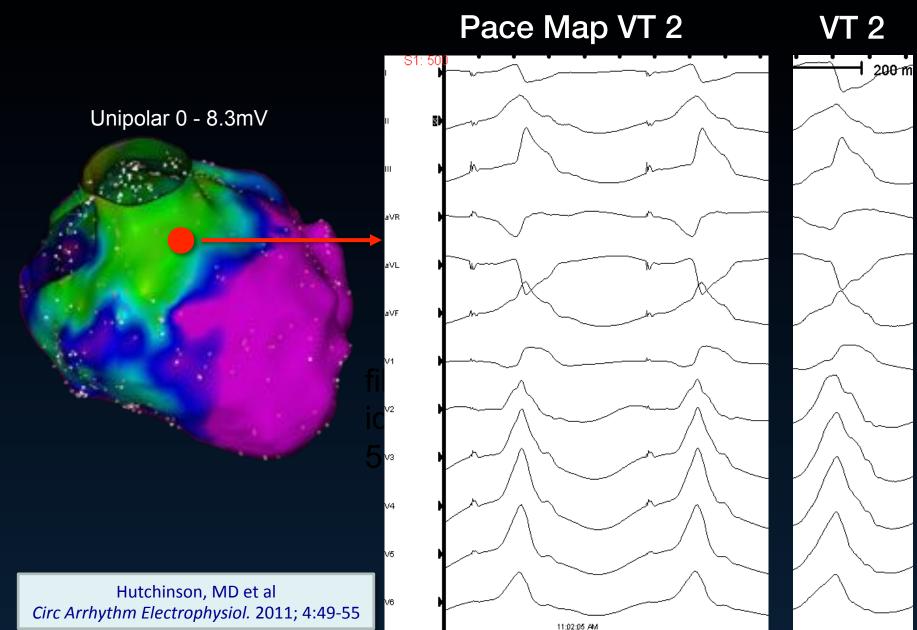


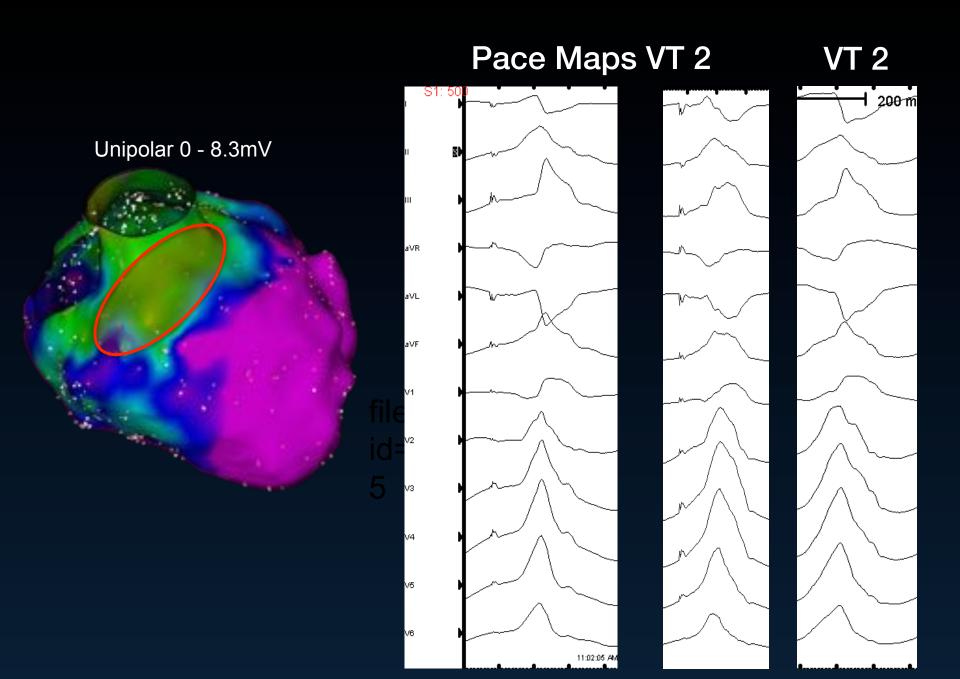


LV Endocardial Bipolar Voltage Map

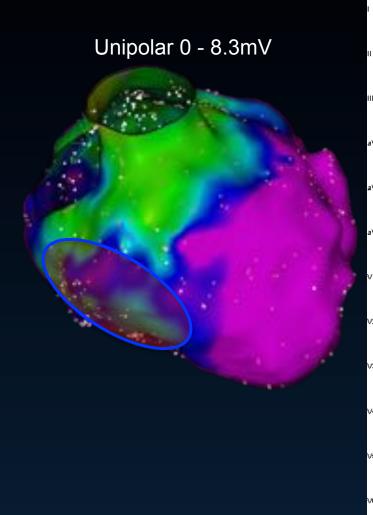


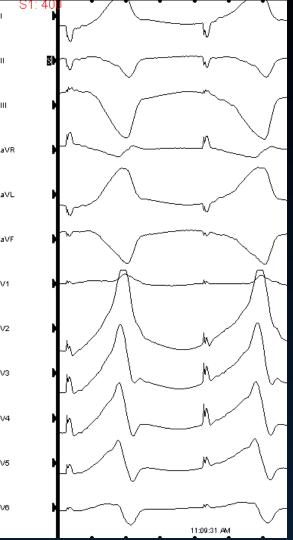


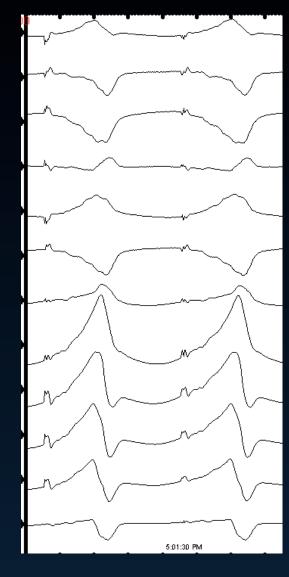




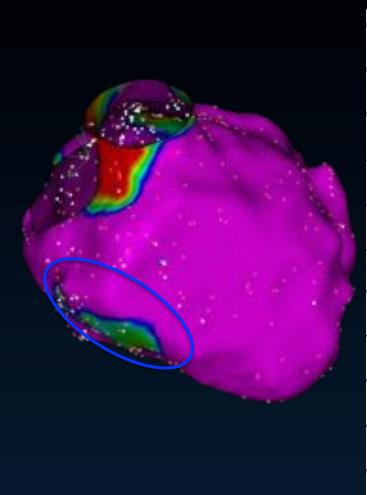
Pace Maps from inferior septum

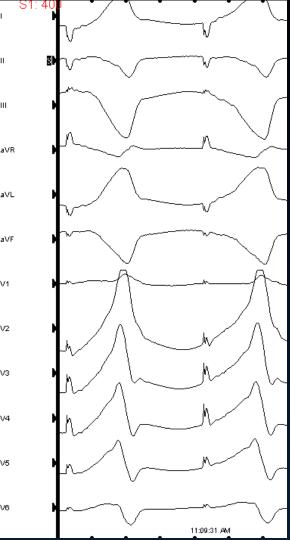


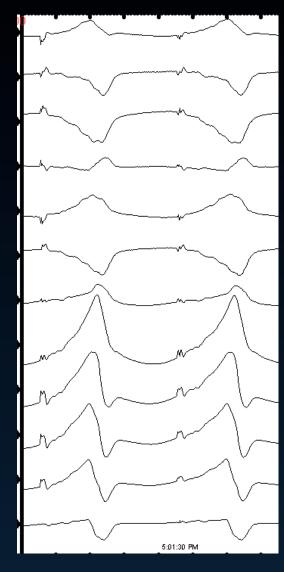




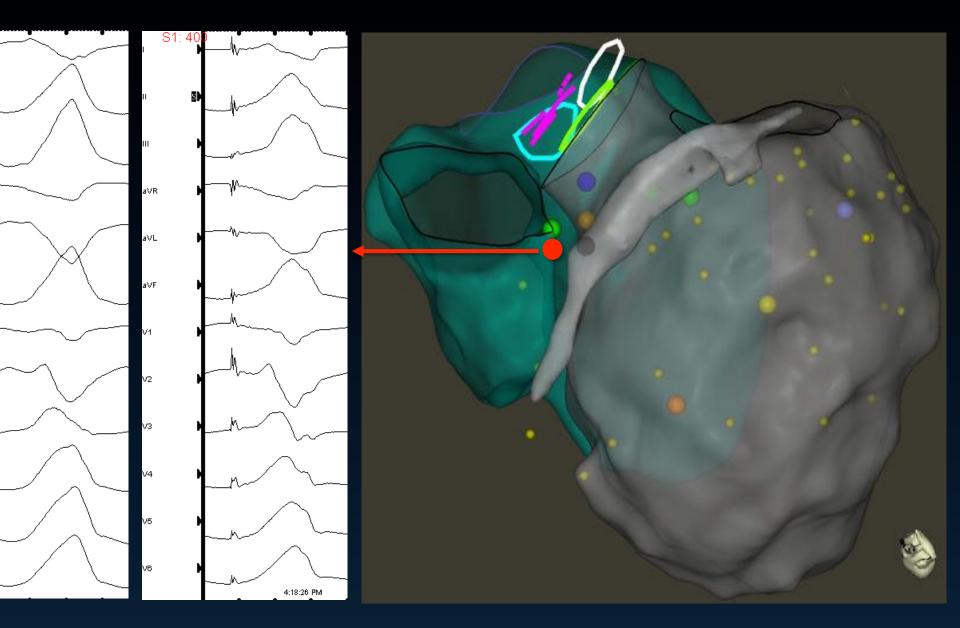
Pace Maps from inferior septum



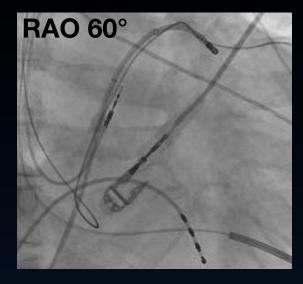


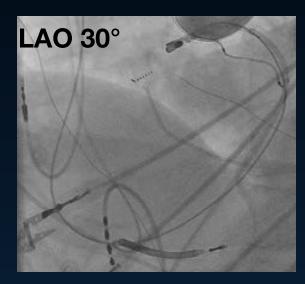


Pace Map VT-1



Pace Map VT-1 from AIV

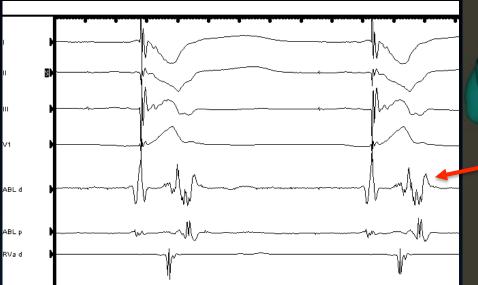


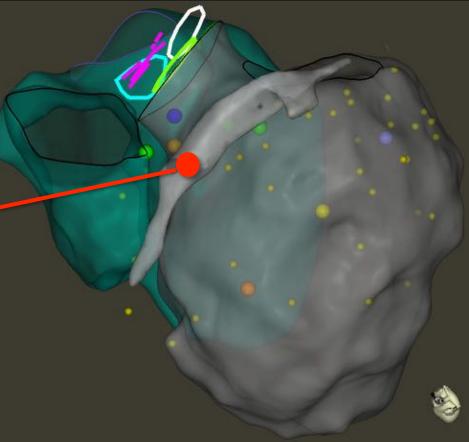




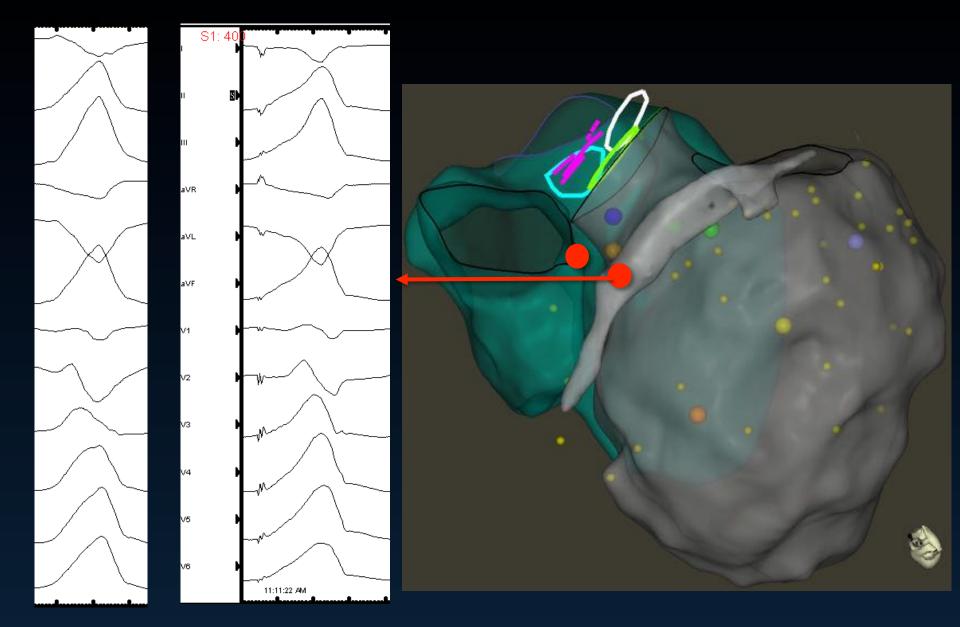
Pace Map VT-1 from AIV

RAO 60°

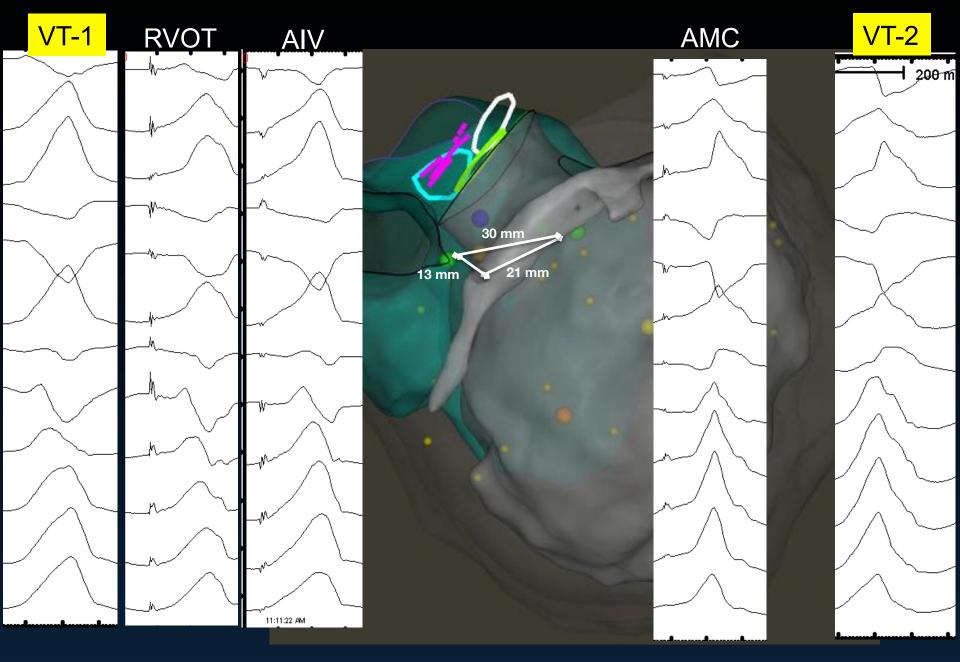




Pace Map VT-1 from AIV

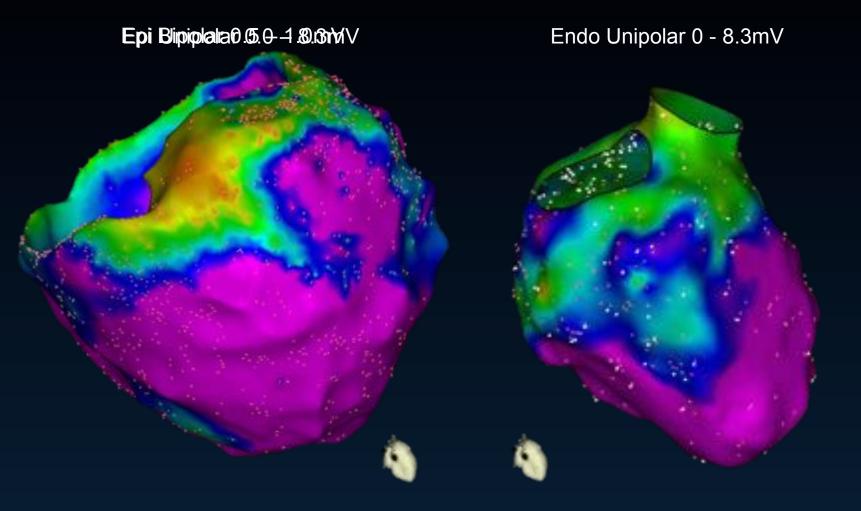


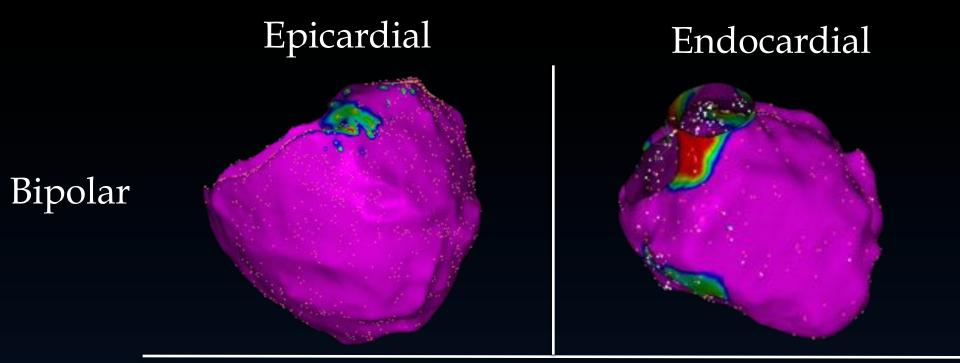
Data from Pace Maps

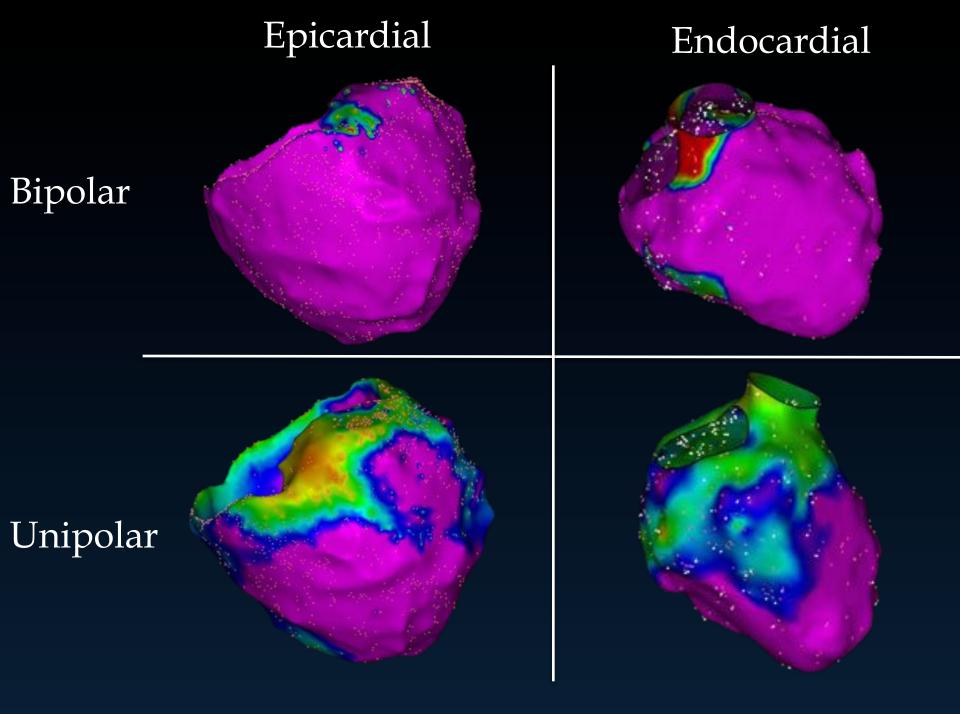


Epicardial Voltage Map

Mapped with DecaNav, >2,000 points

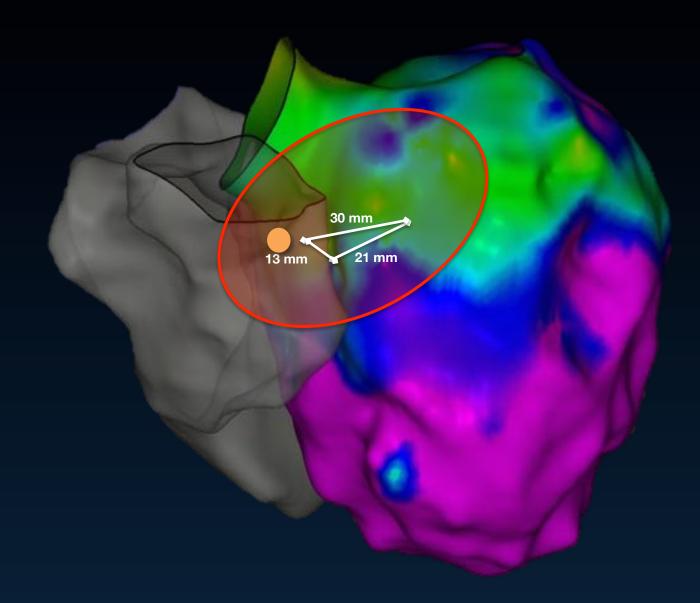




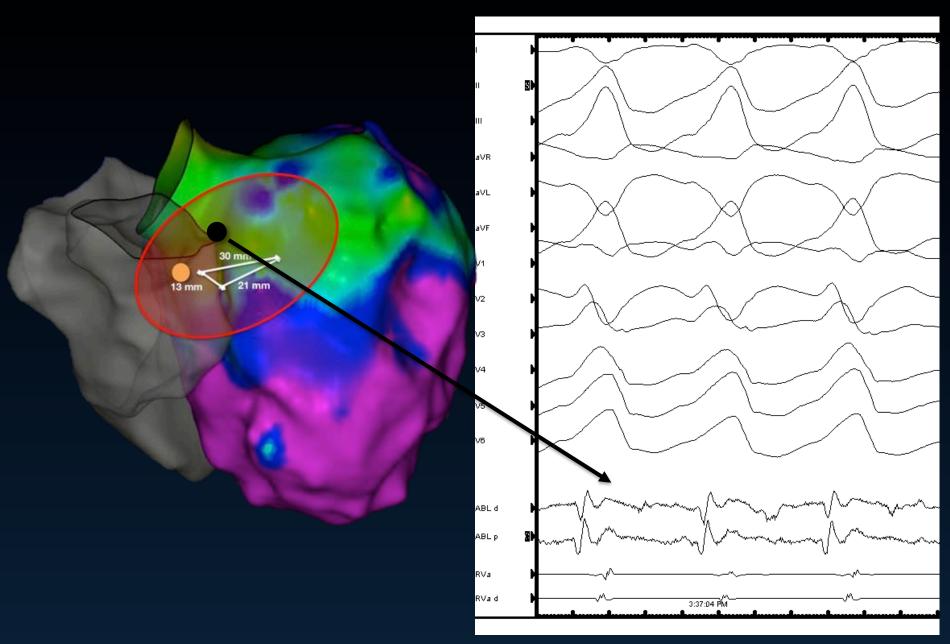


Ablation Strategy

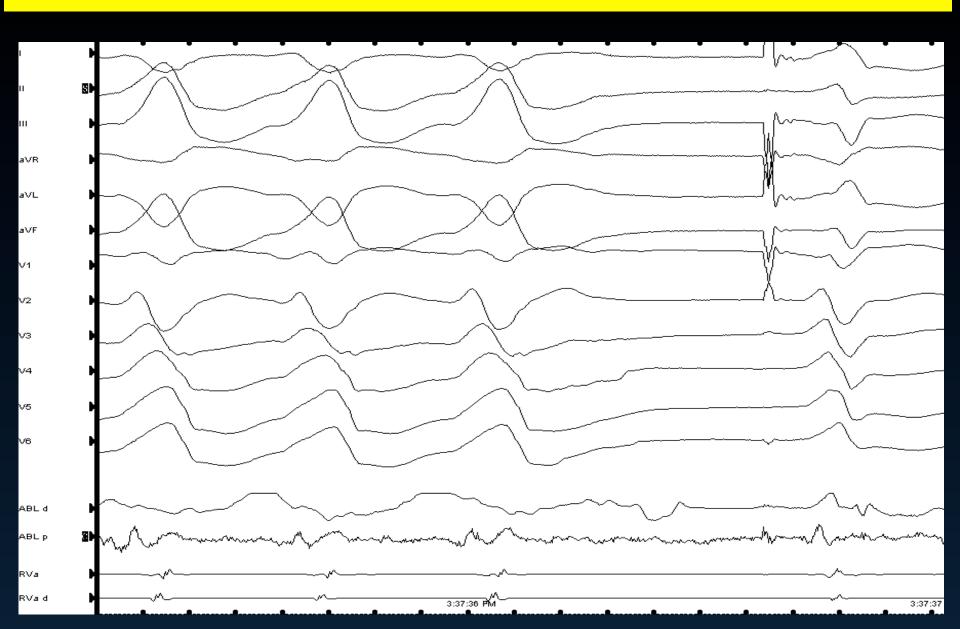
- Substrate is not in the endocardium and not in the epicardium
- Substrate is probably in the midmyocardium
- Ablation from endocardium based on unipolar abl
- Ablation parameters: Tissue contact ≥ 15 gr Power = 30-40W Time = 90 - 240 seconds
- End point of each ablation lesion lack of capture at 20mA@2ms



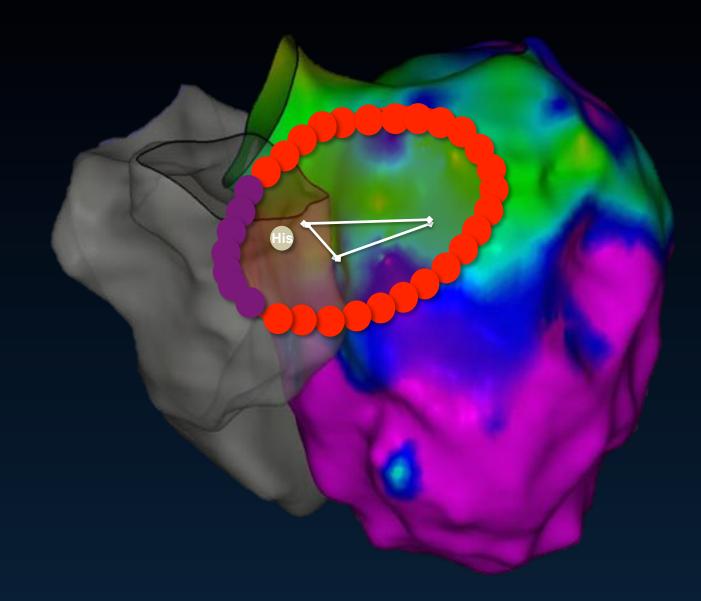
MDP during VT-1 at R/L junction



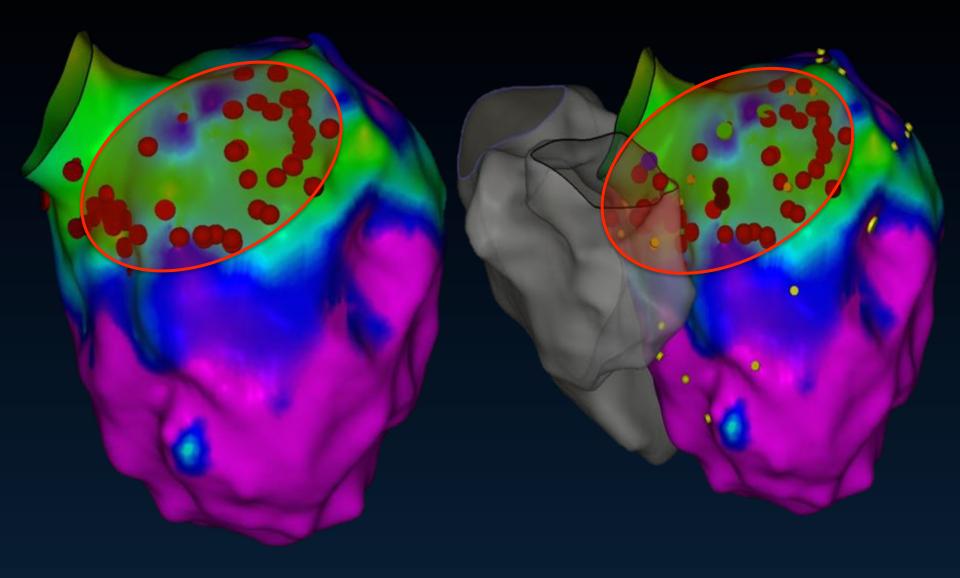
Terminates after 29 seconds of RF

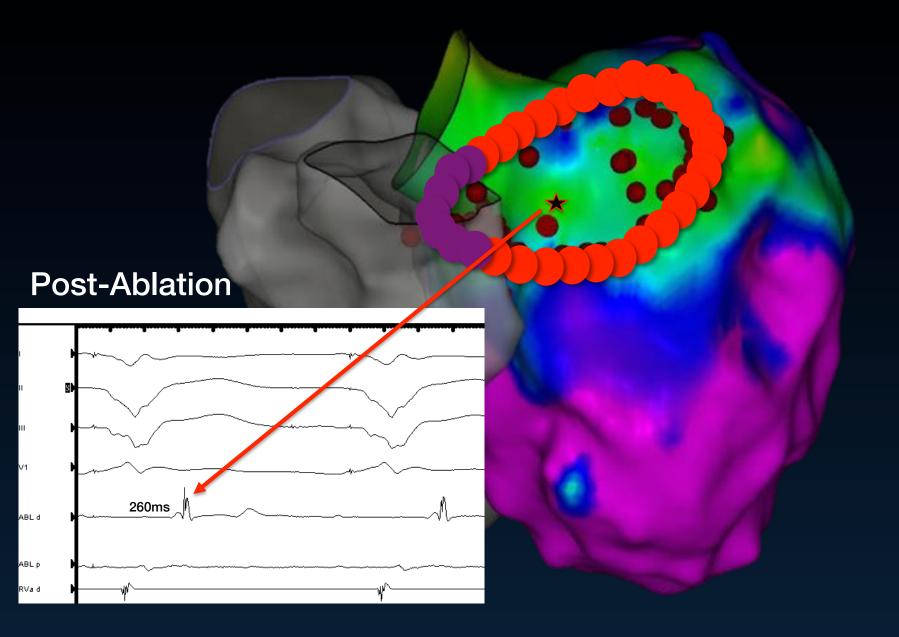


Core Isolation

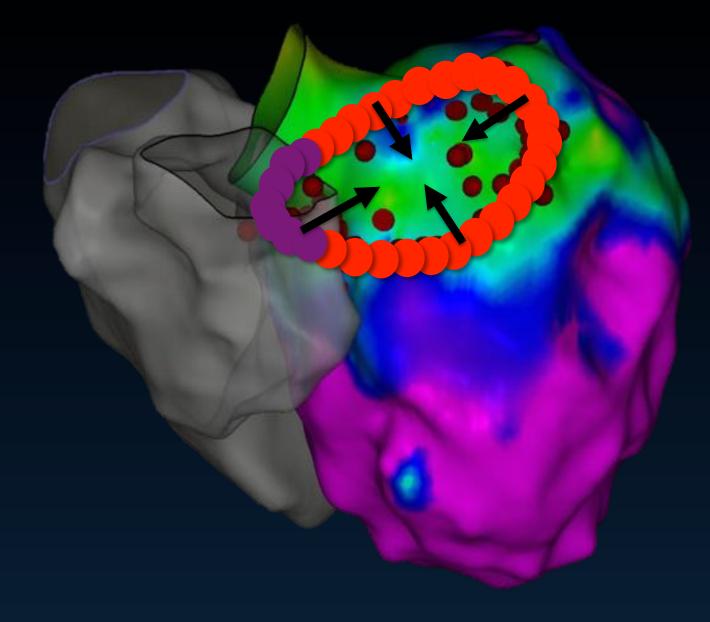


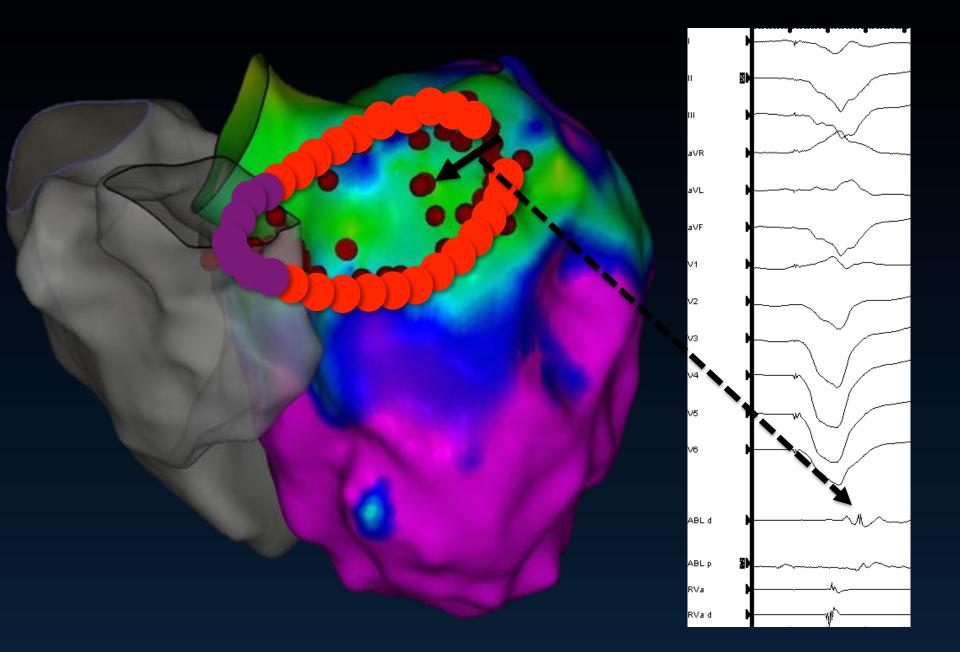
Core Isolation strategy

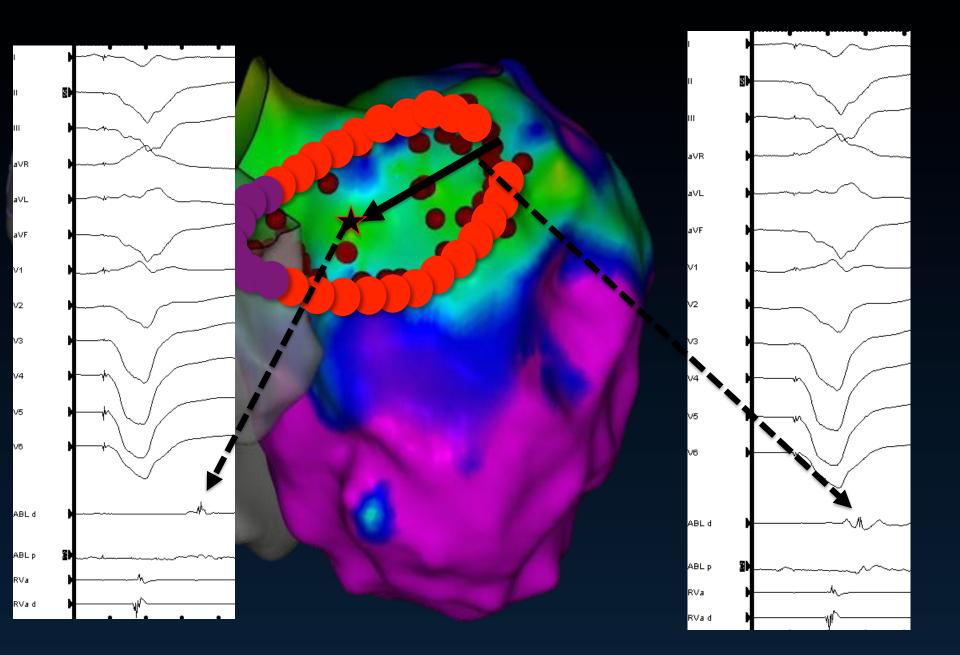


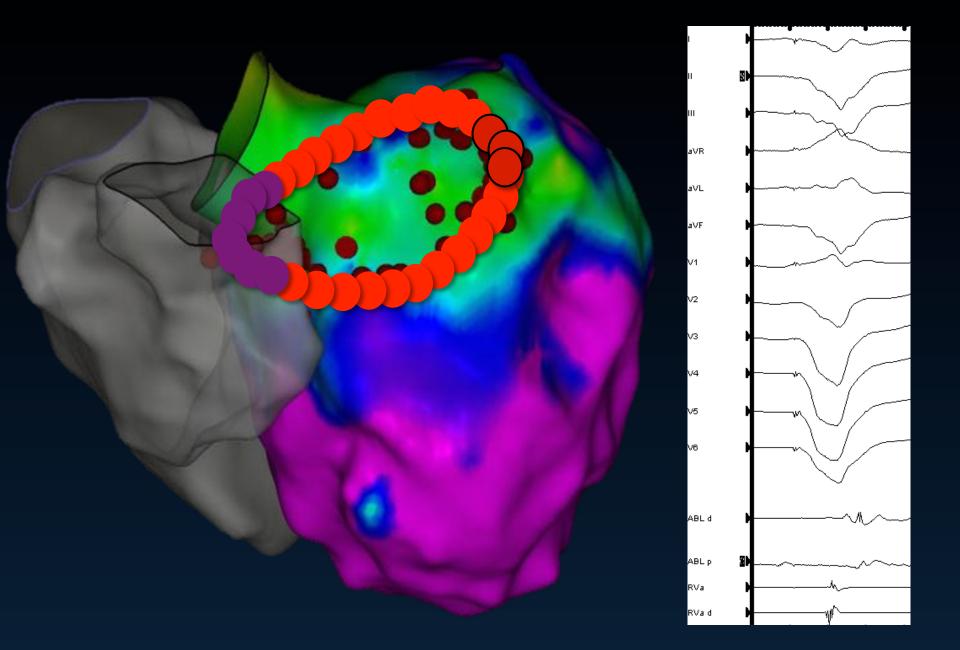


Core Isolation: Identifying the leak

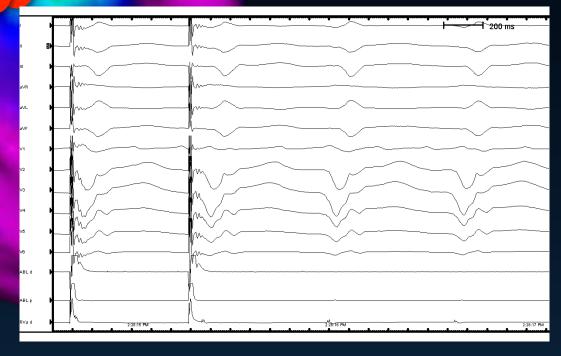




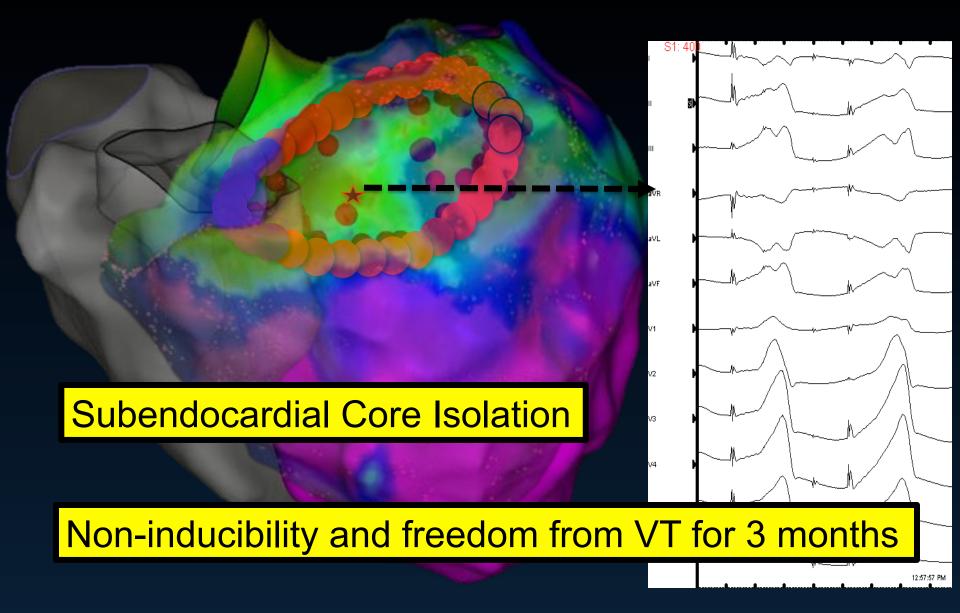




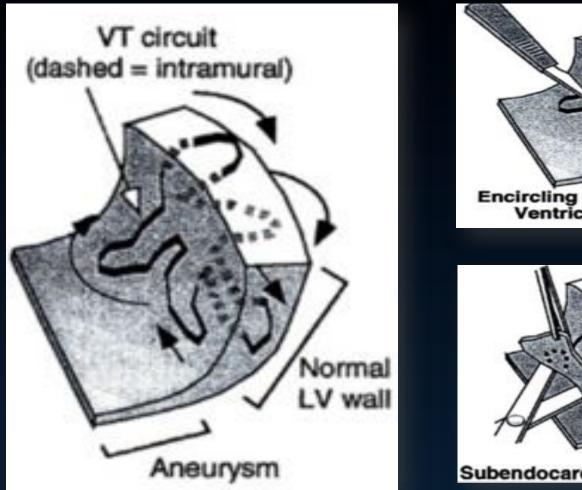
Entrance block Lack of capture at 20mA@5ms



Capture at the opposing epicardial surface



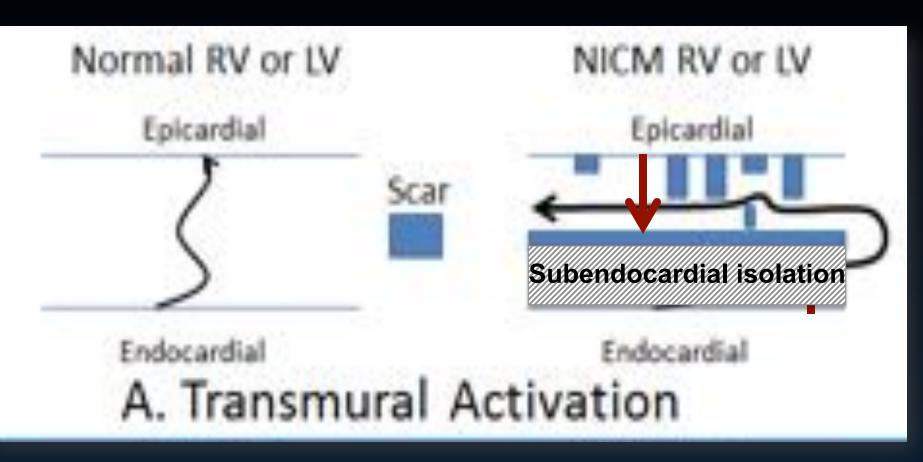
Does isolation of the core needs to be transmural ?



Encircling Endocardial Ventriculotomy Subendocardial Resection

From Miller JM et al. 1997 Williams & Wilkins Baltimore, MD:641-684

Does isolation of the core needs to be transmural ?



Tzou W eat al; Circ EP 2015

Core Isolation as VT endpoint

Core Isolation of Critical Arrhythmia Elements for Treatment of Multiple Scar-Based Ventricular Tachycardias

Wendy S. Tzou, MD*; David S. Frankel, MD*; Timothy Hegeman, DO; Gregory E. Supple, MD; Fermin C. Garcia, MD; Pasquale Santangeli, MD; David F. Katz, MD; William H. Sauer, MD; Francis E. Marchlinski, MD

Background—Radiofrequency ablation of multiple or unmappable ventricular tachycardias (VTs) remains a challenge with unclear end points. We present our experience with a new strategy isolating core elements of VT circuits.

Methods and Results—Patients with structural heart disease presenting for VT radiofrequency ablation at 2 centers were included. Strategy involved entrainment/activation mapping if VT was hemodynamically stable, and voltage mapping with electrogram analysis and pacemapping. Core isolation (CI) was performed incorporating putative isthmus and early exit site(s) based on standard criteria. If VT was noninducible, the dense scar (<0.5 mV) region was isolated. Successful CI was defined by exit block (20 mA at 2 ms) within the isolated region. VT inducibility was also assessed. Forty-four patients were included (mean age, 63; 95% male; 73% ischemic cardiomyopathy; mean left ventricular ejection fraction, 31%; 68% with multiple unstable VTs [mean, 3+2]). CI area was 11+12 versus 55+40 cm² total scar area. Additional substrate modification was performed in 27 (61%), and epicardial radiofrequency ablation was performed in 4 (9%) patients. CI was achieved in 37 (84%) and led to better VT-free survival (log rank P=0.013).

Conclusions—CI is a novel strategy with a discrete and measurable end point beyond VT inducibility to treat patients with multiple or unmappable VTs. The CI region can be selected based on standard characterization of suspected VT isthmus surrogates thus limiting ablation target size. Exit block within the isolated area is achievable in most and may further improve long-term success. (Circ Arrhythm Electrophysiol. 2015;8:353-361. DOI: 10.1161/CIRCEP.114.002310.)

Summary

- The use of unipolar voltage (endocardial and epicardial) enhances our ability to identify abnormal substrate "scar" that is further away from the recording electrodes
- Normal bipolar endocardial and epicardial voltage coupled with abnormal endocardial and epicardial unipolar voltage may suggest a midmyocardial substrate
- Isolation of the arrhythmogenic core may not require transmural lesions formation as lesions only have to reach the existing layer of midmyocardial scar
- Core isolation may be a feasible alternative endpoint for VT ablation