Ventricular arrhythmias : A presentation of some illustrative cases and a review of the most recent management guidelines and supportive literature.

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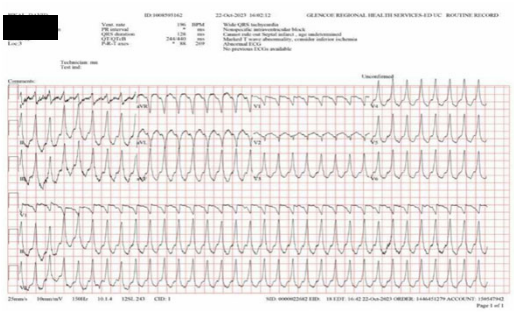
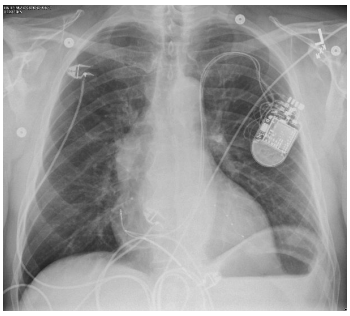




The fear of death follows from the fear of life. A man/woman who lives fully is prepared to die at any time. Mark Twain

1

I have no conflicts of interest to disclose.

2

Talk outline

- 1. Discuss illustrative cases that showcase the issues, therapeutic options, procedural technical challenges and nuances for patients with different flavors of ventricular arrhythmias:
- Frequent PVCs and non sustained VT in the presence of LV systolic dysfunction.
- Sustained monomorphic VT in the presence of prior myocardial infarction or ventricular scarring.
- Sustained monomorphic VT in the setting of surgically corrected complex congenital heart disease.

3

Talk outline continued

2. Show some clinical cases that highlight the issues, therapeutic options, procedural technical challenges and nuances for patients with different flavors of ventricular arrhythmias: (try my very best to stay out of the weeds to keep it interesting for everyone and not just the EPs)

- Frequent PVCs, non sustained VT and sustained VT in non ischemic cardiomyopathy (some epicardial cases).
- Epicardial access and ablation in patients with prior open heart surgery.
- Review a case of idiopathic verapamil sensitive VT
- Brief discussion of a case of polymorphic VT storm and management strategies.

3. Discuss the most recent expert consensus guidelines on the management of ventricular arrhythmias and review some of the supporting primary data.

4

Case 1

- 60 year old female with hypertension, hyperlipidemia, non-ischemic dilated cardiomyopathy (EF 20%) with frequent unifocal premature ventricular contractions(47% of QRS complexes on recent Holter) who presented for second opinion about ICD implantation.
- She had been on medical therapy with carvedilol 25 mg twice daily, entrestro 97/103 mg twice daily and spironolactone for more than 12 months and had been placed on Jardiance 10 mg daily 6 months prior and LVEF remained low.

5

Baseline ecg

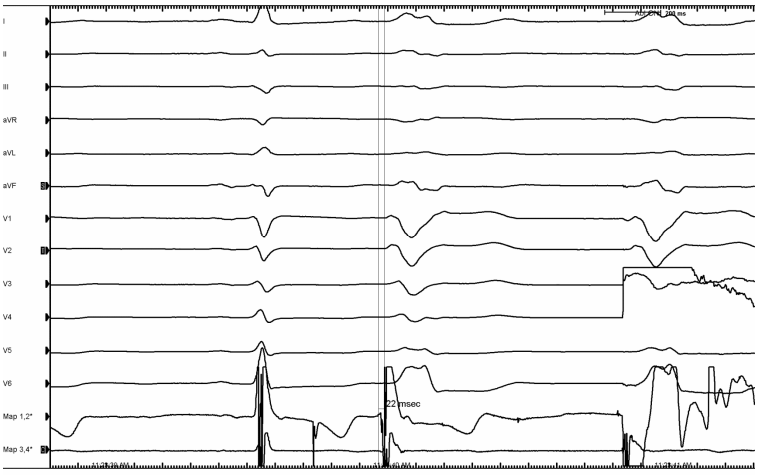
6

What shall we do

- 1. Implant ICD as requested.
- 2. Obtain CT coronary angiography to ensure that she has not developed new coronary artery disease since the original angiography was one year ago when she was just diagnosed with the cardiomyopathy also repeat cardiac MRI
- 3. Start her on amiodarone or other antiarrhythmic.
- 4. Do something else

7

Mapping to find a good site to ablate.



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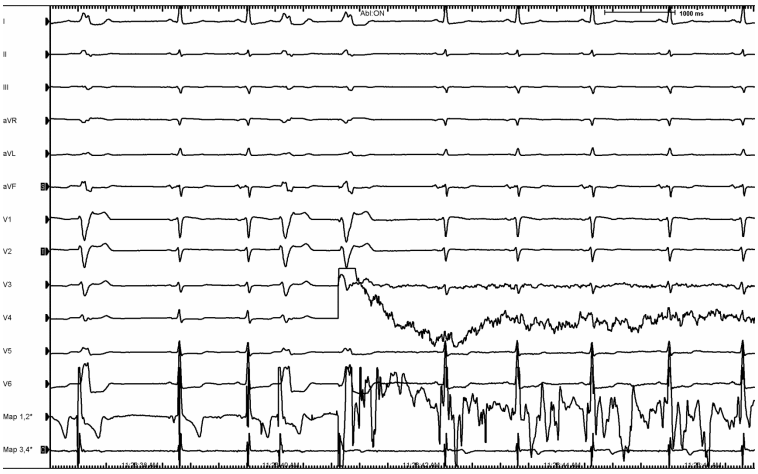
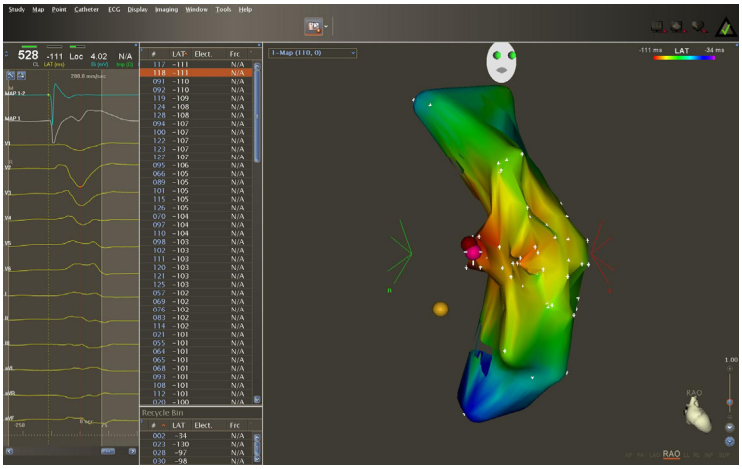
Ablation



PVC was forever silenced!

9

Activation Map and ablation lesion



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Success

- The activation map localized the PVC to the lateral free wall of the right ventricle just below the tricuspid annulus at about 10 o'clock in the 30 degree LAO position. After fine mapping of the earliest site which was ~20 ms pre-QRS with initial fractionation, a single ablation lesion at 35 watts resulted in immediate termination of ventricular ectopy. This ablation lesion was continued for 90 seconds.
- Her LVEF has since improved to 55 % from 20 % at time of initial evaluation and referral for ICD.

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Case 2

- 51 year old female with hypertension and highly symptomatic unifocal premature ventricular contractions that occur in a bigeminal pattern who presents for mapping and ablation of premature ventricular contractions. Therapy with beta blockers and eventually sotalol was ineffective and resulted in fatigue. LVEF was 40% possibly due to frequent PVCs

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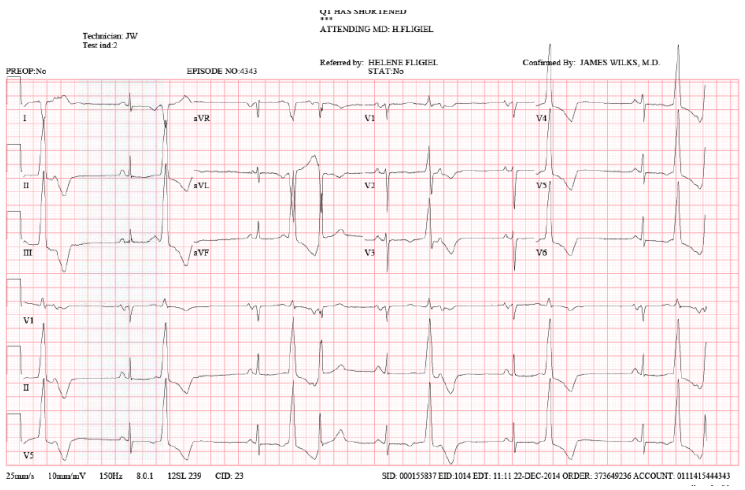
Clinical ECG



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Baseline ecg with PVCs in lab

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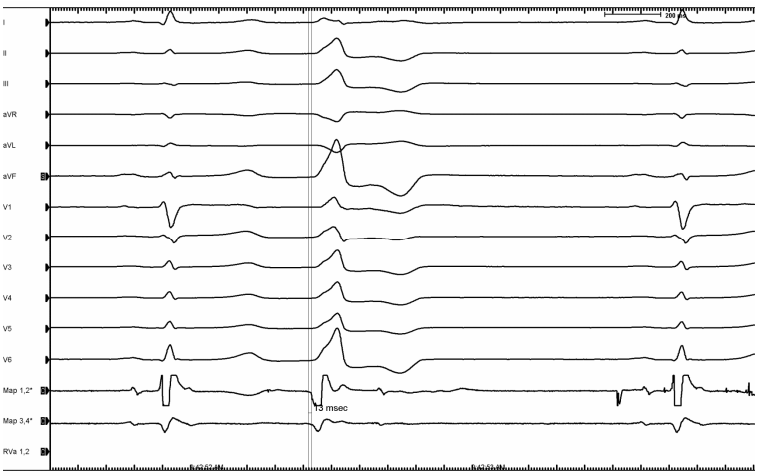
Good site to ablate?



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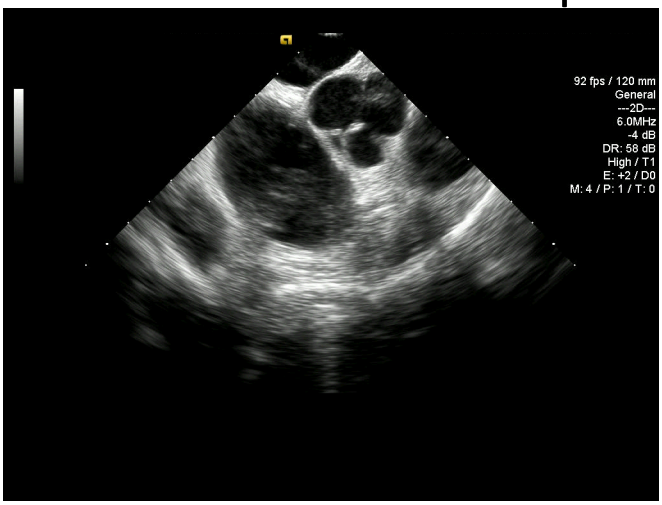
Aortic cusps



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Aortic cusps

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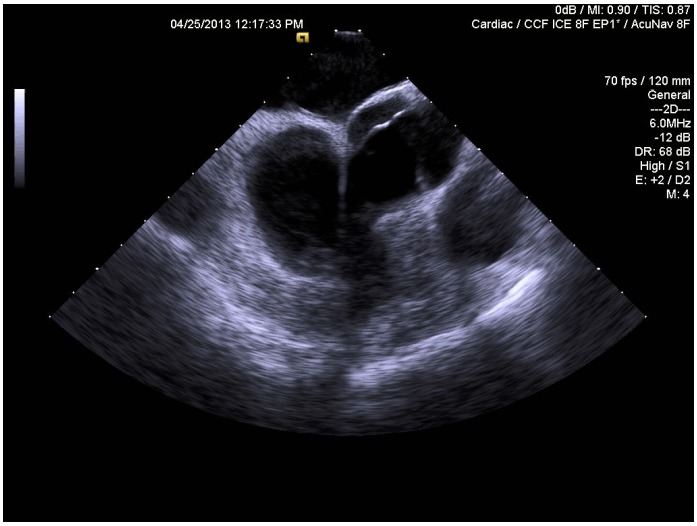
Have to be sure we are in a safe place to ablate



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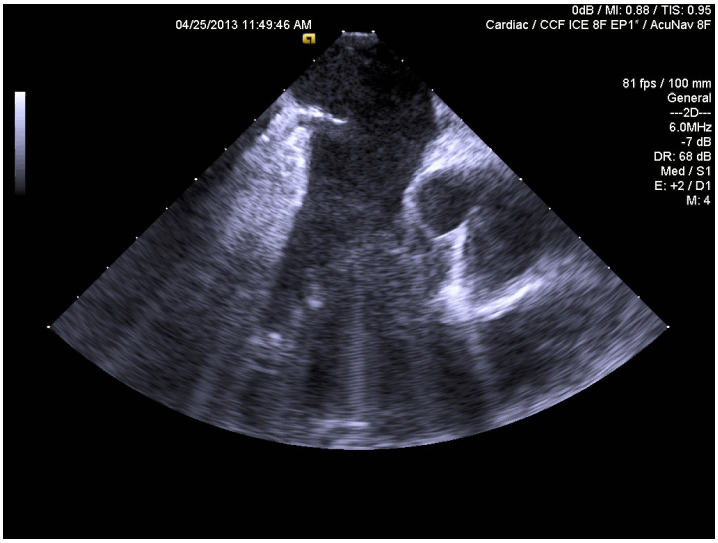

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Ablation

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Success

- After fine mapping of the earliest site which was ~15 ms pre-QRS with initial fractionation in the left coronary cusp, a single ablation lesion at 25 watts resulted in immediate termination of ventricular ectopy. This ablation lesion was continued for 60 seconds. After this single ablation lesion there were no premature ventricular contractions observed over a one hour waiting period. The procedure was terminated.
- Her EF is now 60% and she feels great on no medical therapy.

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Case 3

- 68 year old female with non ischemic cardiomyopathy with LVEF of 35%, frequent PVCs (53% on recent holter presents for EP study and ablation of PVC focus.
- She failed therapy initially with sotalol and eventually amiodarone.

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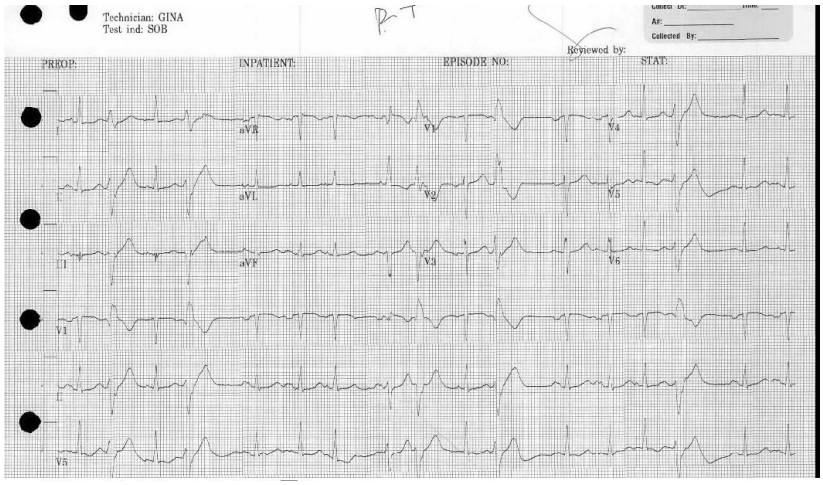
12 lead of PVC



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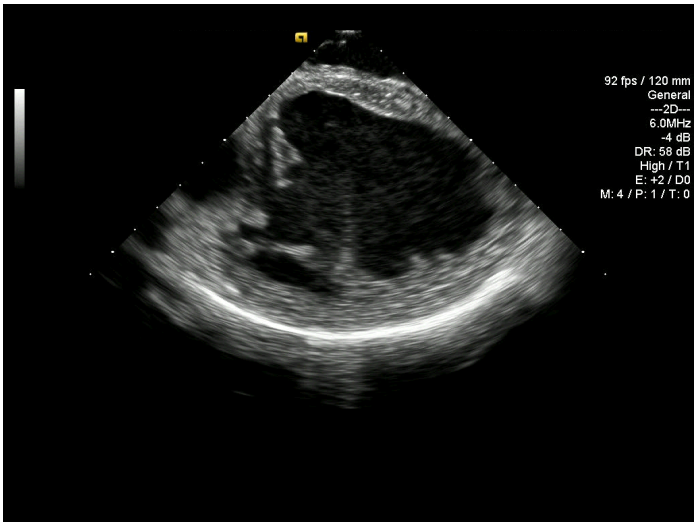
Intracardiac echo



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Clinical Progress

- Patient had successful mapping and ablation of PVC focus which was mapped to the base of the posteromedial papillary muscle. Her LVEF normalized.

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Case 4

- 48 year old female with sickle cell disease(SC), paroxysmal atrial fibrillation, nonischemic dilated cardiomyopathy with severe left ventricular systolic dysfunction, prior dual chamber ICD implant 2010, history of frequent PVCs for which she had an ablation in 2018 who is admitted with recurrent sustained monomorphic ventricular tachycardia and PVCs(with identical morphology to previous PVC as well as clinical VT) who is referred for redo VT ablation.

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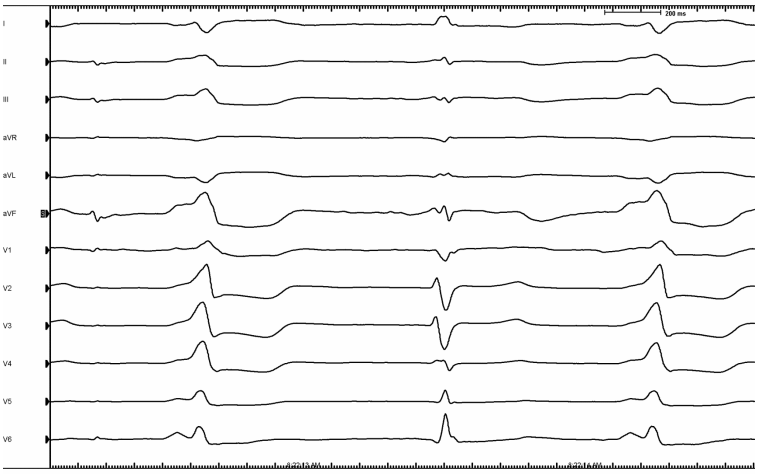
Clinical PVCs



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Criteri for epicardial VT circuit/ Focus

Slurring of the initial portion of the QRS and delayed activation

- Pseudo delta ≥ 34 ms
- Intrinsicoid deflection time ≥ 85 ms
- Shortest RS complex in precordial leads ≥ 121 ms
- Maximum deflection index ≥ 0.55
- QS or rS in lead 1

Berruezo etal Circulation 2004, Daniels etal Circulation 2006, Bazan etal Heart Rhythm 2007

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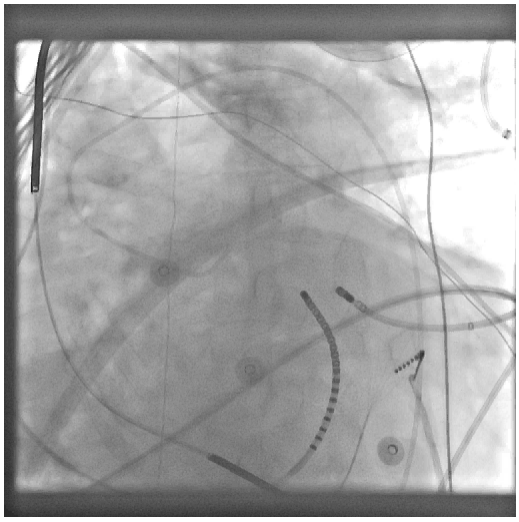
Went straight for mapping epicardial surface of heart



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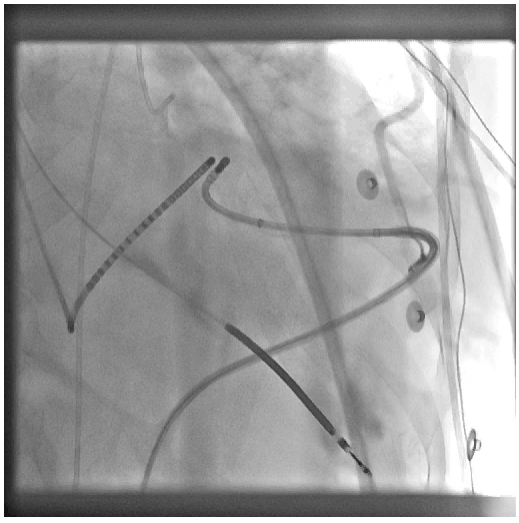

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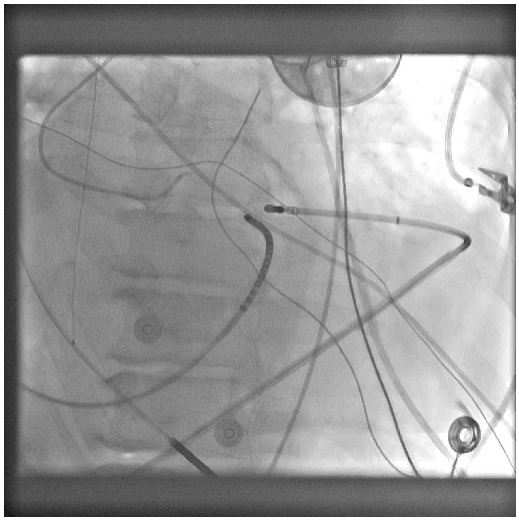
Too close for comfort



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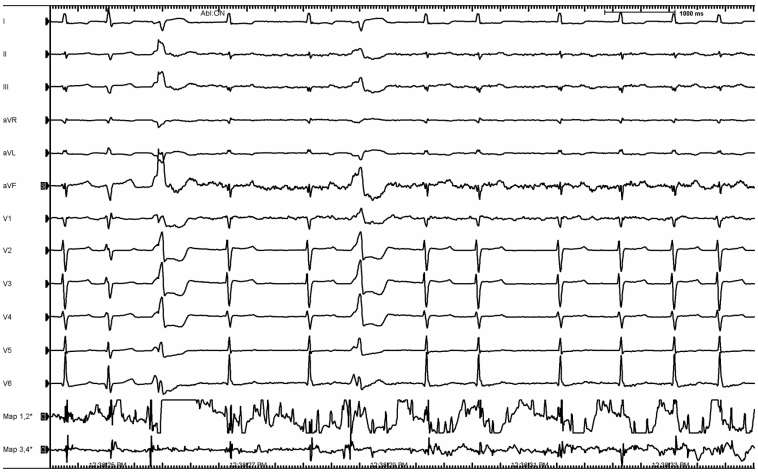
Ablation



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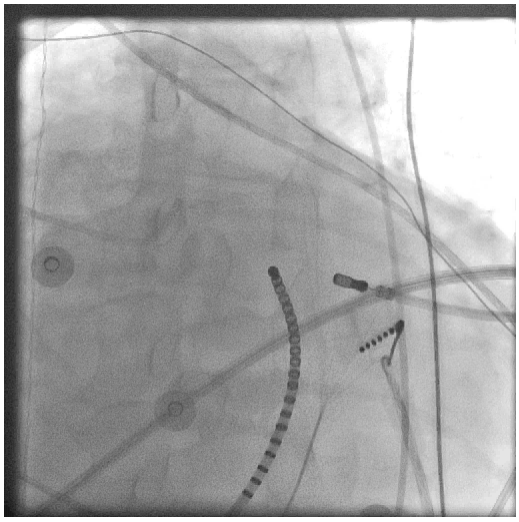
Small marginal branch sacrificed



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Case 5 :Brief History

- 37 year old female with history of frequent PVCs and mild LV dysfunction (EF 45-50%)who collapsed at work. Had CPR by coworkers and three shocks by Automated external defibrillator which was luckily present in the office where she works.
- On presentation she has chest soreness and no other complaints. Physical examination shows anterior chest bruising and is otherwise unremarkable.

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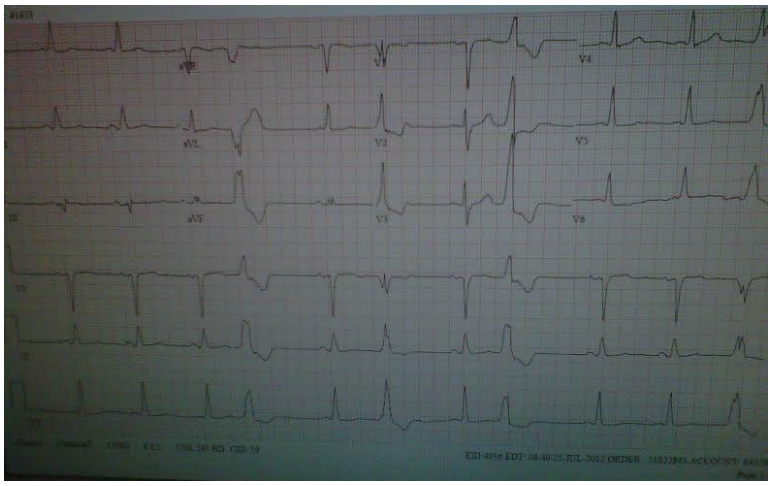
ECG on presentation



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What shall we do

- 1. Cardiac CTA or invasive angiography ?
- 2. Cardiac MRI then implant ICD and do nothing else?
- 3. Implant ICD and do something else?
- 4. Something else?

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Ablation

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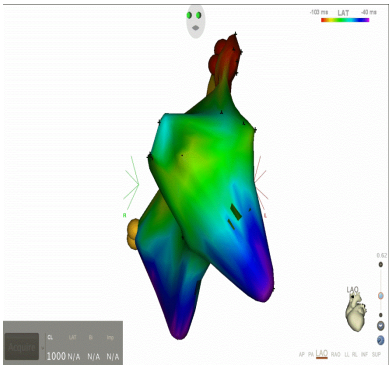
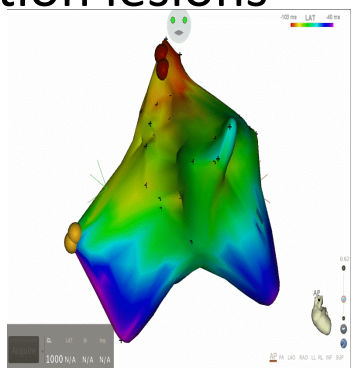
LV map and Ablation lesions





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Clinical course

- She declined an ICD. She wore a Wearable Cardioverter defibrillator(LifeVest for 6 months).
- Had numerous Holters that showed no PVCs.
- EF now 60 %.
- She no longer wears the Life Vest and is doing well raising her daughter on no cardiac medications.

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Case 6 Brief History

- 40 year old man with repaired tetralogy of Fallot with enlarged RV, Pulmonary insufficiency, mild pulmonary hypertension and sustained monomorphic VT refractory to sotalol and mexiletene and requiring ICD shocks in the past who presents to EP lab for mapping and ablation for recurrent symptomatic tachycardia episodes.

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Surgical and EP history

- He had a shunt Blalock Taussig Shunt at age 4 months of age and a complete repair at age 5 yrs.
- He developed marked RV and RA enlargement, pulmonary stenosis and regurgitation with RV dysfunction.
- On 12/7/04 he underwent reconstruction of pulmonary valve annulus, excision of main PA with insertion of 28mm pulmonary homograft between RV and distal MPA.
- In July 2012 he had an ICD implantation for easily inducible sustained monomorphic ventricular tachycardia after admission with syncope.

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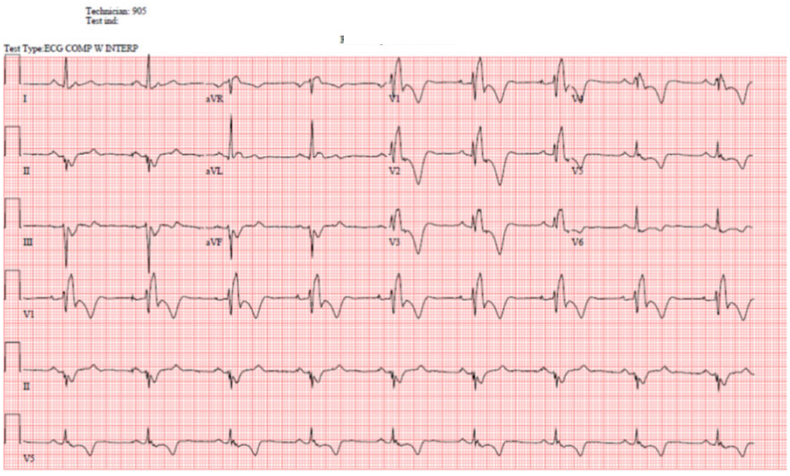
Baseline ecg



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Induction of tachycardia
500-290-240

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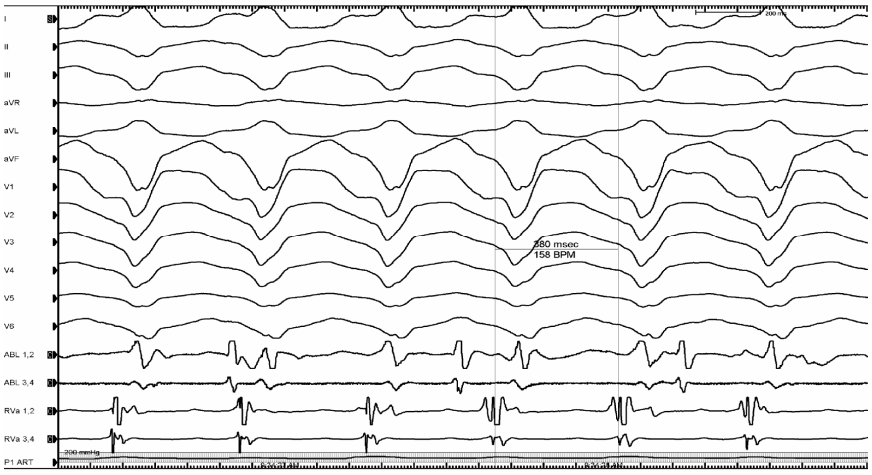
Tachycardia CL



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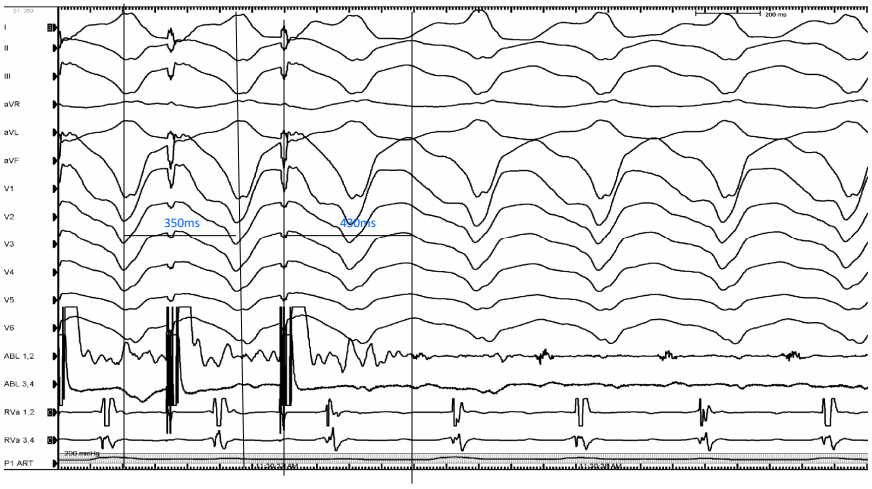
What do you think about this site for ablation



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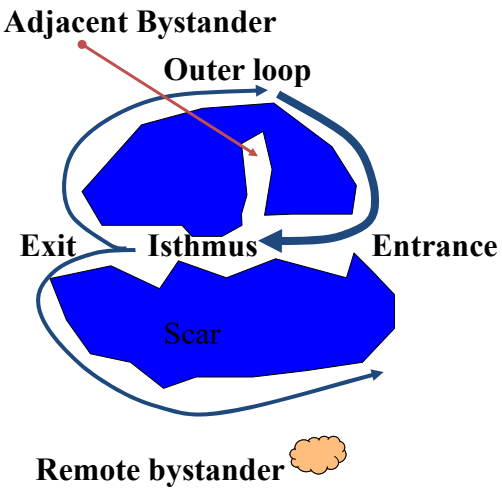
Where is mapping catheter?



A. Inner loop
B. Outer loop
C. Central isthmus
D. Adjacent bystander
E. Remote bystander
F. Proximal Isthmus
G. Exit site of Isthmus

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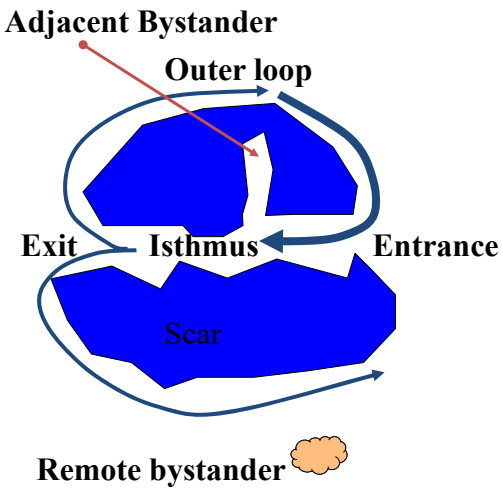
Where is mapping catheter?



A. Inner loop
B. Outer loop
C. Central isthmus
D. Adjacent bystander
E. Remote bystander
F. Proximal Isthmus
G. Exit site of Isthmus

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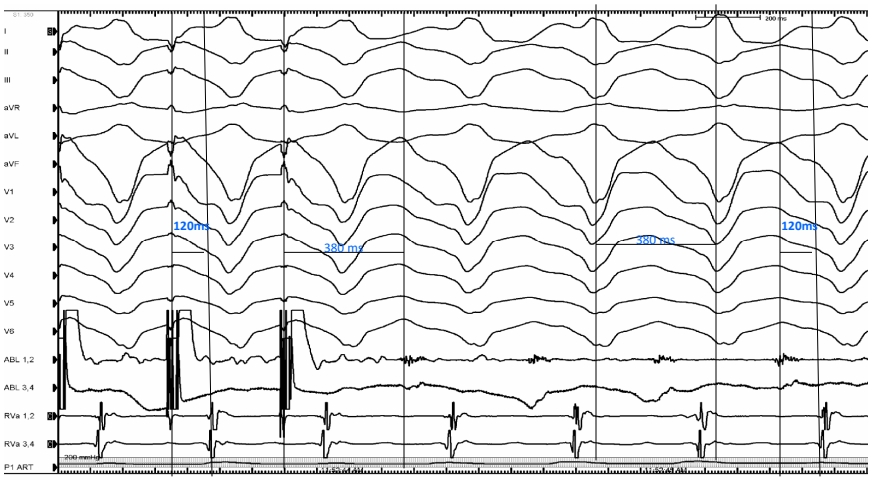
How about here



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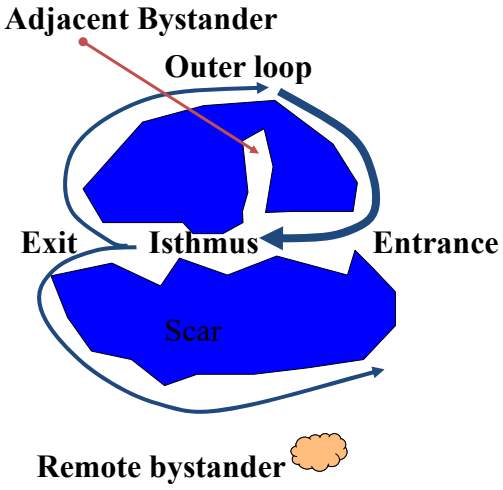
Where is mapping catheter?



A. Inner loop
B. Outer loop
C. Central isthmus
D. Adjacent bystander
E. Remote bystander
F. Proximal Isthmus
G. Exit site of Isthmus

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We have studied the beast : now we shall slay it!

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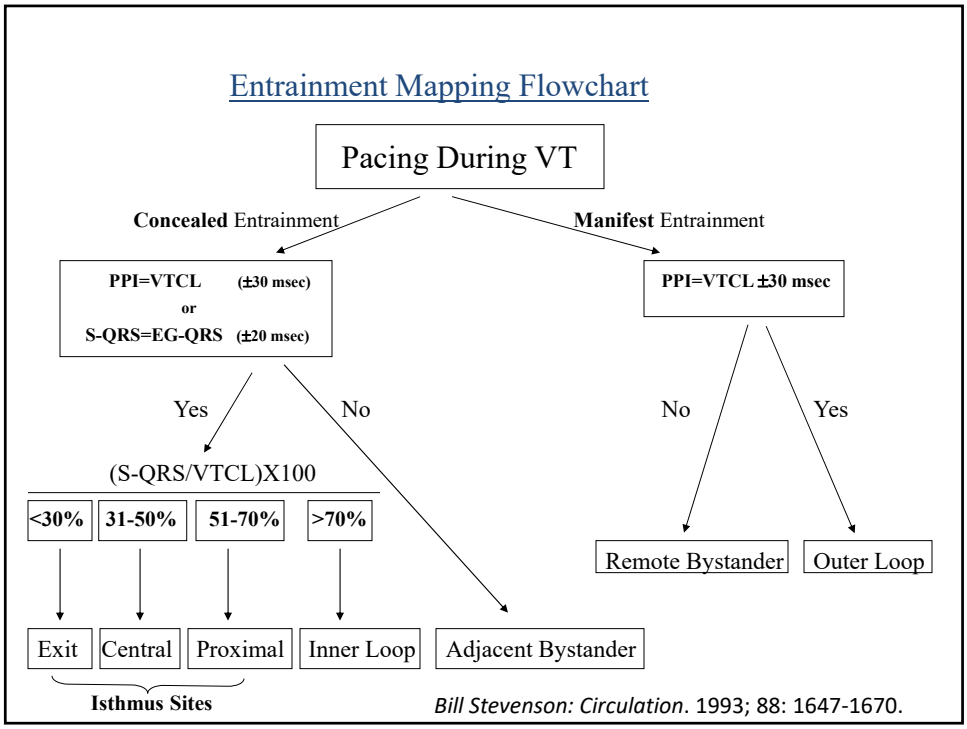

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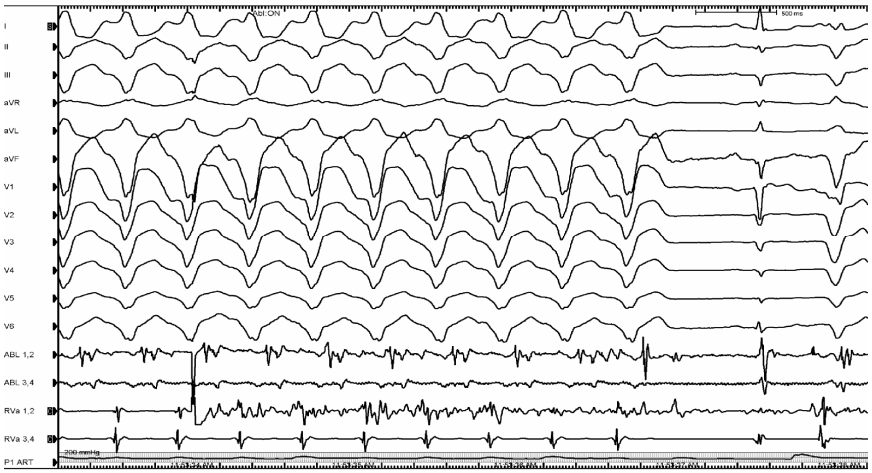
Termination with ablation



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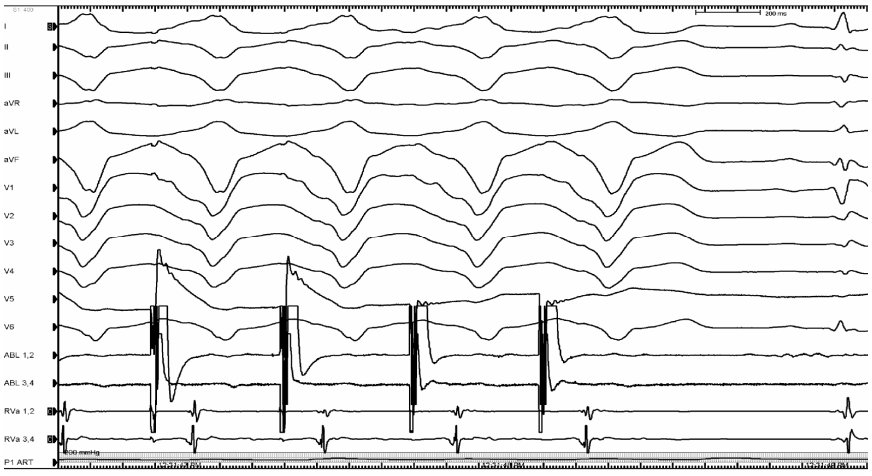
Perfect pacemap of VT



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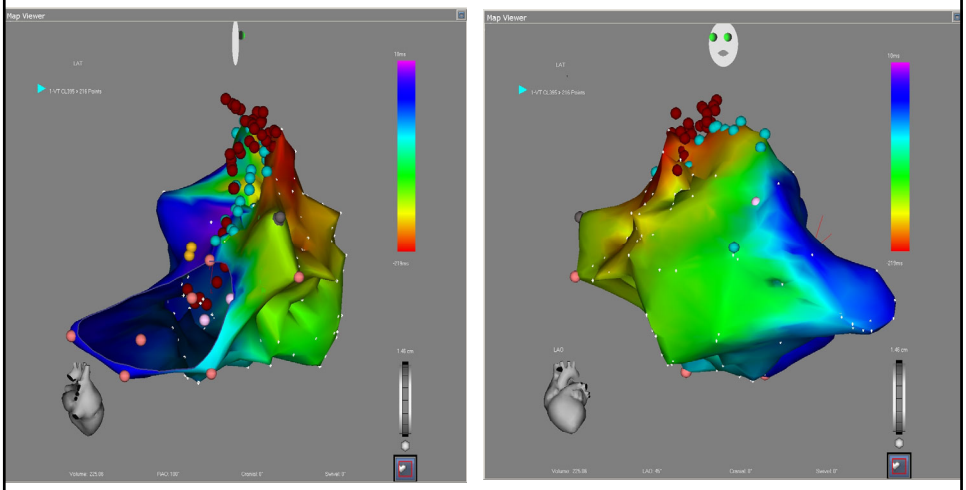
RV map and ablation points



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Procedure Note : Success !

- Double potentials consistent with his prior surgery were found and mapped extending from the high RVOT downward. Early activation during the VT that was mid/early diastolic was found just anterior to the line of double potentials in the high RVOT despite the marked superior axis. Entrainment here showed concealed entrainment with 12/12 pace-map matching and return cycle matching the VT CL.
- Further mapping at the high RVOT site located a very early diastolic potential of high amplitude and near merging of electrograms along the double potential line. This was just cranial to where our prior ablation had started. Ablation here immediately terminated the VT after 7 beats (2.8 sec) with pre-termination prolongation between the double potentials. We applied lesions at surrounding the successful site and along the line of double potentials that extended cranially toward the pulmonic valve.
- After the ablation, no further VT could be induced, including with triple extrastimuli at 2 PCLs from the RVA and RVOT and rapid burst pacing .

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Not inducible at end with triple extrastimuli

RVOT pacing

56

(Case from Cleveland Days)

- 66 year old male with ischemic cardiomyopathy with EF of 35%,
  revascularization with coronary artery bypass surgery 21 years ago (LIMA-LAD, SVG - LCx,and prior ICD with recurrent ICD shocks for fast monomorphic VT despite escalating doses of sotalol and mexiletine. He had 3 prior endocardial ablations at revered institutions who presents for EP study and ablation of VT circuits and triggers. He was allergic to amiodarone

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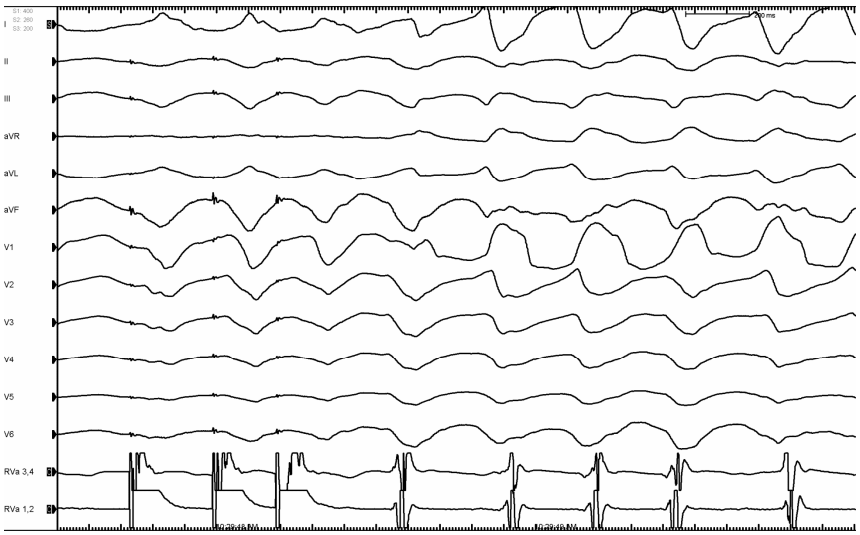
Induction of tachycardia



BP 40/0------ patient needs emergent cardoversion

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Now what ?

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Criteria for epicardial VT circuit

Slurring of the initial portion of the QRS and delayed activation

- Pseudo delta ≥ 34 ms
- Intrinsicoid deflection time ≥ 85 ms
- Shortest RS complex in precordial leads ≥ 121 ms
- Maximum deflection index ≥ 0.55
- QS or rS in lead 1

Berruezo etal Circulation 2004, Daniels etal Circulation 2006, Bazan etal Heart Rhythm 2017

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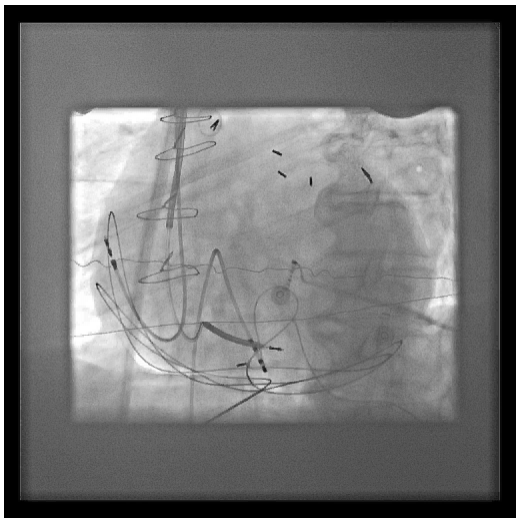
Decide to go epicardial



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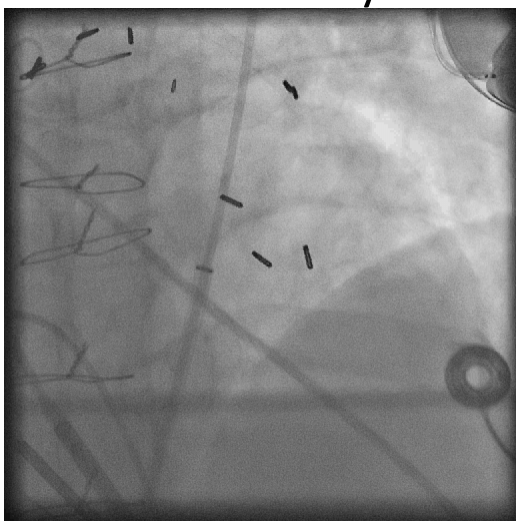
Making sure to stay away from the grafts and coronary arteries



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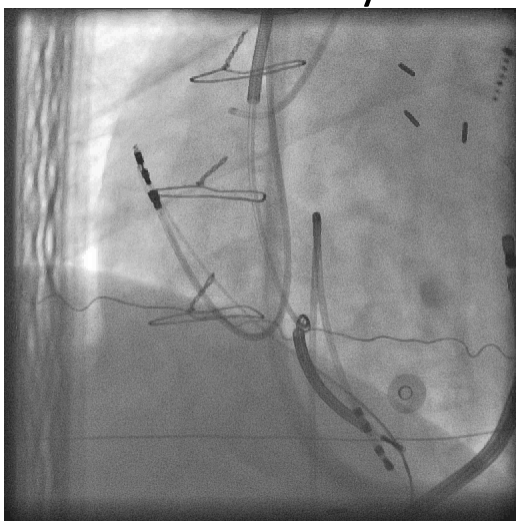
Making sure to stay away from the grafts and coronary arteries



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Making sure to stay away from the grafts and coronary arteries

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Making sure to stay away from potential collateral damage

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Hemodynamic support with epicardial ablation in a post CABG patient

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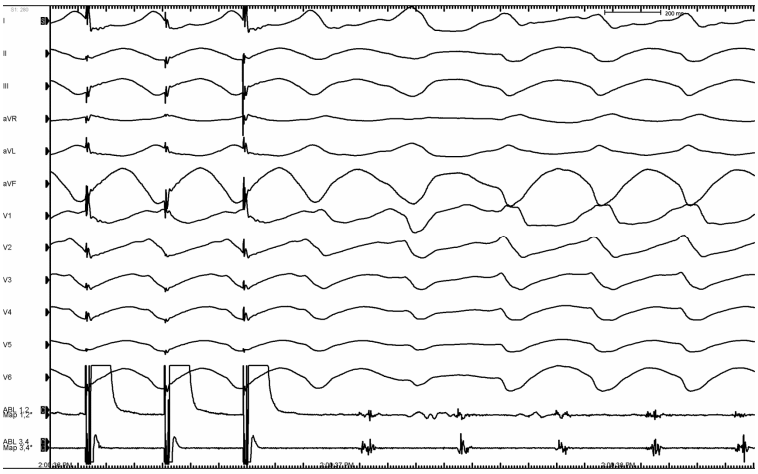
On epicardial surface of heart with impella



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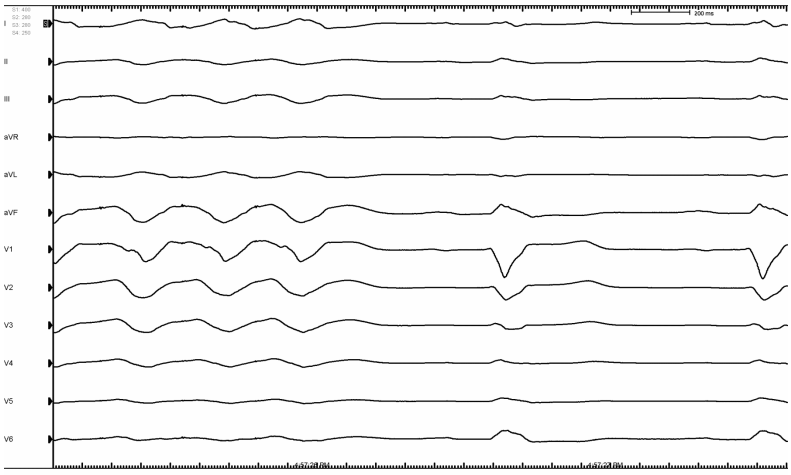
After extensive epicardial ablation and aposing endocardial surface ablation



SUCCESS!!!!!

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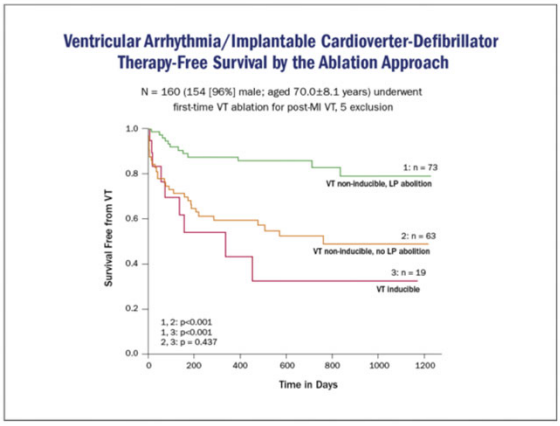

A total of 160 patients postinfarction undergoing first-time ablation procedures. Of the 159 patients surviving the procedure, 137 (86%) were inducible or in VT at baseline and 103 (65%) had baseline LP presence, of which 79 (77%) underwent successful LP abolition. Kaplan-Meier curves comparing freedom from VT recurrence according to groups. Patients who had the combined endpoint of VT noninducibility and LP abolition compared with inducible patients exhibited a significantly lower incidence of VT recurrence (16.4% vs. 47.4%; p < 0.001). Among noninducible patients, those with additional LP abolition also had a lower incidence of VT recurrence (16.4% vs. 46%; p < 0.001). After multivariate analysis, the combined endpoint of VT noninducibility and LP abolition (hazard ratio, 0.205; p < 0.001) was independently associated with VT recurrence and cardiac death (hazard ratio, 0.106; p = 0.001).

LP = late potential; MI = myocardial infarction; VT = ventricular tachycardia.

Modified from Silberbauer J, Oloriz T, Maccabelli G, et al. Noninducibility and late potential abolition: a novel combined prognostic procedural end point for catheter ablation of postinfarction ventricular tachycardia. Circ Arrhythm Electrophysiol 2014;7:424-35.

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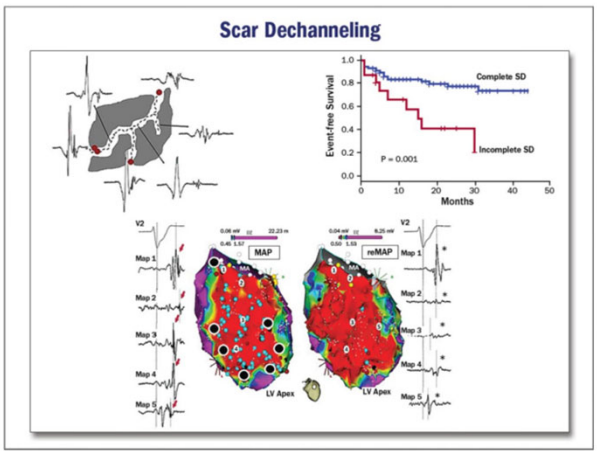

Inferior view of bipolar voltage electroanatomical substrate maps during sinus rhythm before (MAP) and after (reMAP) scar dechanneling in a patient with healed myocardial infarction. Electrograms recorded as conducting channel entrances are labeled with black dots and inner sites with blue dots. Examples of bipolar electrograms at entrances (1 and 5) and inner parts (2-4) are shown. Delayed components of the electrograms are highlighted with arrows. After scar de-channeling (SD), repeat electrogram recordings at the same sites demonstrated elimination of the delayed component (asterisks). Kaplan–Meier curve showed a higher 2-year probability of ventricular arrhythmia or sudden death event-free survival (80% versus 62%) according to the complete versus incomplete elimination of conducting channel electrograms.

LV = left ventricular; MA = mitral annulus; SD = scar dechanneling.

Modified from Berruezo A, Fernández-Armenta J, Andreu D, et al. Scar dechanneling: new method for scar-related left ventricular tachycardia substrate ablation. Circ Arrhythm Electrophysiol 2015;8:326-36.

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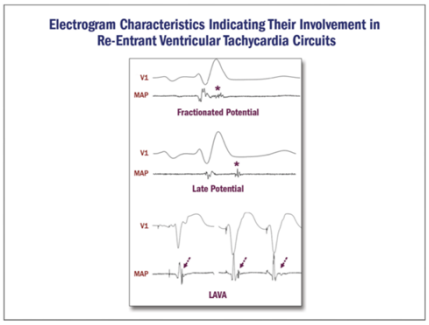
Mapping without induction of VT



Electrogram characteristics that indicate their involvement in re-entrant VT circuits: fractionated electrograms (top row, asterisk), late potentials (middle row, asterisk), and LAVA (bottom row). In the bottom row, ventricular pacing separates the local abnormal electrogram (dashed arrow) from the far-field electrogram with demonstration of local entrance block to the site in the third paced beat.

LAVA = local abnormal ventricular activity; MAP = mapping catheter; VT = ventricular tachycardia.

Reproduced with permission from Dukkipati SR, Choudry S, Koruth JS, et al. Catheter ablation of ventricular tachycardia in structurally normal hearts: indications, strategies, and outcomes-Part I. J Am Coll Cardiol 2017;70:2909-23.

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Case 8 Brief History

- 20 year old College Basketball Player with recurrent palpitations while playing basketball. Verapamil suppressed his tachycardia but impaired his performance. He was referred for VT Ablation for verapamil sensitive idiopathic ventricular tachycardia

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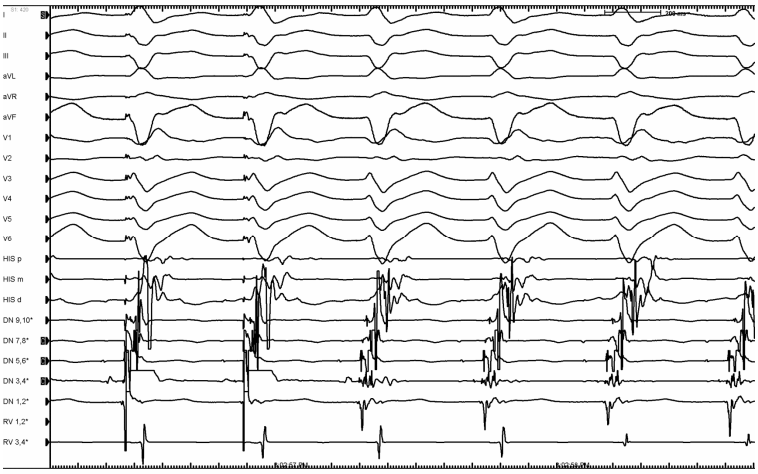
Tachycardia with entrainment



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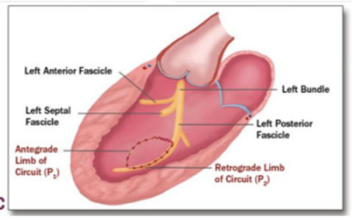
Automaticity with ablation



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Brief History

- 57 year old man with Waldestrom's Macroglobulinemia and Non Hodgkin's lymphoma for which he was on acyclovir and recently started on ibrutinib 140 mg daily who is admitted with recurrent syncope. While in ER he has numerous polymorphic VT/ VF arrests.

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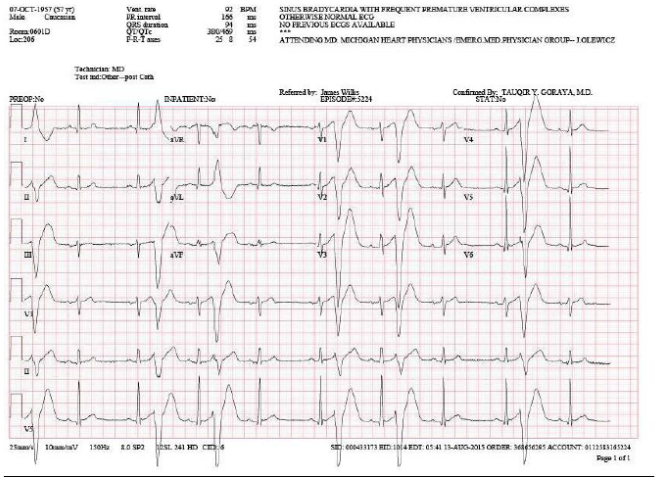
Initial ECG in ER



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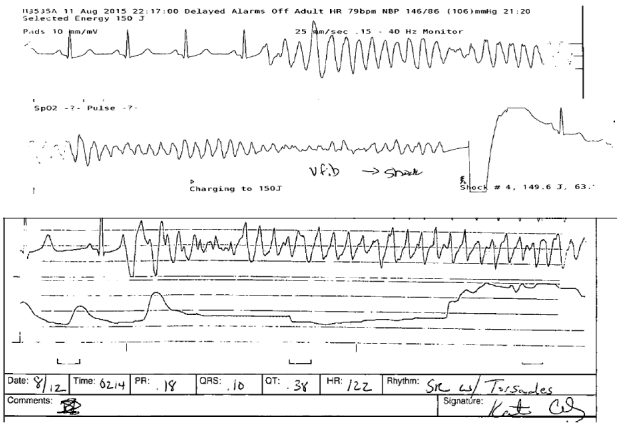
Numerous times throughout the hospitalization



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History continued

- Cath: Normal right dominant coronary arteries.
- LV gram _ EF 15%.
- What shall we do?

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Clinical Course

- Numerous external shocks through out the night.
- Improved marginally with isoproterenol.
- Taken to the EP lab where we ablated PVC focus from the moderator band of the right ventricle

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- Now lets review the guidelines.

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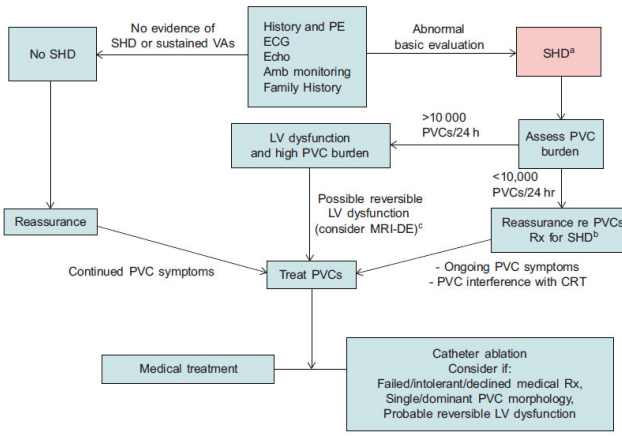

Figure 1 Management of PVCs. a. See table for definitions of structural heart disease; b. Medical therapy + ICD; c. Absence of high scar burden suggests reversibility. CRT = cardiac resynchronisation therapy; ICD = implantable cardioverter-defibrillator; LV = left ventricular; MRI-DE = magnetic resonance imaging with delayed enhancement; PE = physical examination; PVC = premature ventricular complexes; Rx = therapy; SHD = structural heart disease; VAs = ventricular arrhythmias.

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Considerations for NSVT in " Normal hearts"

Table 3 Non-sustained ventricular tachycardia with apparent normal heart

NSVT clinical presentation	ECG	Risk of sudden cardiac death	Diagnostic evaluation	Alternative diagnostic considerations	Treatment	Treatment to be considered	Key references
Typical RVOT	LBBB, inf axis, axis transition V3 – V4	Very rare	Standard	Differentiate from ARVC	Beta-blocker, verapamil, IC drugs with symptoms	Catheter ablation	Latif et al.[62]
Typical LVOT	Inferior axis, transition, V3	Very rare	Standard	RVOT VT	Beta-blocker, verapamil, IC drugs with symptoms	Catheter ablation	Latif et al.[62]
Idiopathic reentrant LV tachycardia	RBBB, LS axis	Very rare	Standard EP testing	Ischaemic heart disease, CM	Verapamil if symptomatic	Catheter ablation	Latif et al.[62]
Other focal VT	Multiple morphologies, monomorphic	Uncommon	Exercise testing or catecholamine stimulation	Ischaemic heart disease, CM	Beta-blocker for the arrhythmia	Catheter ablation	Latif et al.[62]
Exercise	Multiple	Increased risk when NSVT in recovery	Ischaemic heart disease, cardiomyopathy	CPVT	Underlying disease	Beta-blockers, flecainide	Jouven et al.[65], Frolkis et al.[66]
Athlete	Multiple	If it disappears with increased exercise low risk	Evaluate for latent HCM or ischaemic heart disease	HCM	No treatment training can continue	None	Biffi et al.[67,68]
Hypertension valvular disease	Multiple morphology	As without arrhythmia	Consider ischaemic heart disease	Ischaemic heart disease, CM	Treat HTN	Beta-blocker	
Polymorphic VT	Polymorphic	High	Evaluated for CAD, CPVT, inherited arrhythmia syndromes	Purkinje fibre triggering focus	Underlying disease	Revascularization, ICD, beta-blocker, catheter ablation	Zipes et al.[60]
TdP VT	Long QT, TdP	High	Medications, congenital LQTS	Medications, K^+, Mg^{++}, Ca^{++}	Stop medications, correct electrolytes	ICD, beta-blocker	Sauer and Newton-Cheh[69]

ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; CM = cardiomyopathy; HTN = hypertension; CPVT = catecholaminergic polymorphic ventricular tachycardia; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; LS = left-superior; LV = left ventricular; LVOT = left ventricular outflow tract; NSVT = non-sustained ventricular tachycardia; RBBB = right bundle branch block; RVOT = right ventricular outflow tract; TdP = torsade de pointes; VT = ventricular tachycardia.

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Considerations for NSVT in the presence of structural heart disease

Table 4 Non-sustained ventricular tachycardia in structural heart disease

Clinical setting	Risk of sudden cardiac death	Arrhythmia specialist evaluation	Diagnostic evaluation	Diagnostics to be considered	Treatment	Treatment to be considered	Key references
ACS within 48 h	No increased risk	No	Coronary artery disease	Monitoring	Beta-blockers		Hohnloser et al.[70]
ACS after 48 h	Risk increased	Yes	Consider EPS if moderate LV dysfunction	Continued evaluation for repetitive arrhythmias	Beta-blockers	ICD	Zipes et al.[60]
Previous MI, EF 31 – 40	Increased risk	Yes	EPS		ICD withinducible VT/VF	ICD, see relevant guidelines	Zipes et al.[60]
Previous MI, EF ≤ 30 Chronic heart failure, EF ≤ 30	Increased risk	Yes	Non-driven by arrhythmia		ICD	Antiarrhythmic medical therapy or ablation with symptoms	Zipes et al.[60]
Syncope with chronic CAD, EF ~40	Increased risk	Yes	EP testing, ischaemia testing	Monitoring	ICD with inducible VT/VF	Additional antiarrhytmic therapy or ablation	Zipes et al.[60]
Non-ischaemic dilated CM	Uncertain	Yes	Uncertain	EP testing	Uncertain	ICD, see relevant guidelines	Zipes et al.[60]
HCM	Increased risk	Yes	Echo, MRI	MRI-DE	Beta-blocker, ICD		Zipes et al.[60]
LQTS	Increased risk	Yes	Genetic screening		Beta-blocker	ICD	Zipes et al.[60]
Short QT syndrome	Increased risk	Yes	Provocative testing				
Brugada syndrome	Increased risk	Yes	Provocative testing	Genetic screening	With syncope or cardiac arrest: ICD	Quinidine	Aliot et al.[3]
ER syndrome	Increased risk	Yes					

CAD = coronary artery disease; CM = cardiomyopathy; EF = ejection fraction; EP = electrophysiology; EPS = electrophysiological study; ER = early repolarisation; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter defibrillator; LV = left ventricular; MI = myocardial infarction; VF = ventricular fibrillation; VT = ventricular tachycardia.

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Sustained monomorphic VT

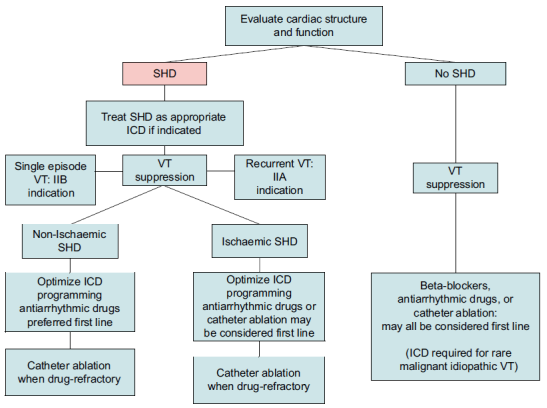

Figure 5 Sustained monomorphic ventricular tachycardia evaluation and management. ICD = implantable cardioverter-defibrillator; SHD = structural heart disease; VT = ventricular tachycardia.

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Polymorphic Ventricular tachycardia and Ventricular fibrillation

Sustained polymorphic ventricular tachycardia/ ventricular fibrillation

Expert consensus recommendations on sustained polymorphic VT/VF
1. Patients with polymorphic VT or VF should be thoroughly evaluated for the presence of SHD, inherited arrhythmia syndromes, early repolarization, coronary artery spasm, and pro-arrhythmic effects of medications using: a. Twelve-lead ECG during the arrhythmia (when feasible) and during normal rhythm. I LOE C b. Echocardiography. I LOE B c. Coronary arteriography. I LOE B
2. Specific antiarrhythmic therapies, e.g. quinidine in patients with idiopathic VF, sodium channel blocker therapy in patients with long QT syndrome (LQTS) III, intensive autonomic inhibition in patients with catecholaminergic VTs, or quinidine in BrS, should be considered in close cooperation with a specialist in these diseases to reduce the risk of recurrence as an adjunct to—and rarely as an alternative to—defibrillator therapy in survivors of polymorphic VAs. Detailed guidance can be found in the APHRS/EHRA/HRS document on inherited arrhythmia syndromes. IIa LOE B
3. For patients with VT/VF storm, reversible factors such as electrolyte abnormalities, pro-arrhythmic drugs, ischaemia, and decompensated chronic heart failure should be corrected. I LOE C
4. Pharmacological suppression of VT/VF storm with beta-adrenergic blockers, amiodarone, and/or lidocaine should be considered in all patients. IIa LOE C
5. For patients with VT/VF storm in whom pharmacological suppression has not been effective and who are unstable, neuraxial modulation, mechanical ventilation, catheter ablation, and/or anaesthesia may be considered. IIb LOE C
6. Catheter ablation of VTs or a triggering focus of VF should be considered in patients with VT/VF storm when adequate experience is available. IIa LOE C
7. For patients with VT/VF storm and significant SHD, implantation of a LV assist device (LVAD) or heart transplant evaluation should be considered and discussed early after the initial event. IIa LOE C

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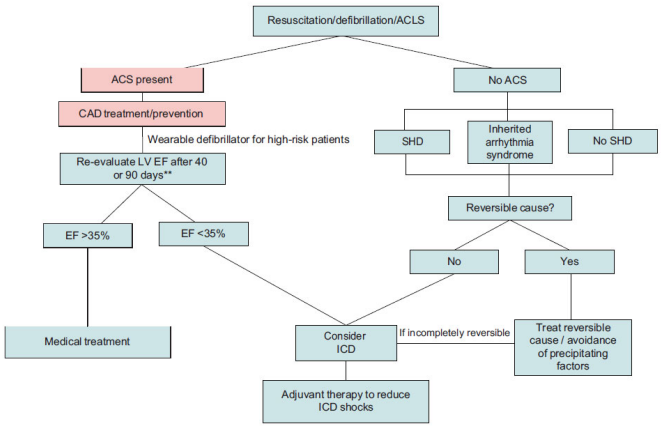
Patients resuscitated from VT/VF



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A few words on congenital heart disease patients

Ventricular arrhythmias in congenital heart disease

Expert consensus recommendations on VAs in CHD
(1) Electrophysiological testing is indicated in adults with unexplained syncope and 'high-risk' CHD substrates associated with primary VAs or poorly tolerated atrial tachyarrhythmias, such as tetralogy of Fallot, transposition of the great arteries with atrial switch surgery, or significant systemic or single ventricular dysfunction. I LOE C.
(2) In patients with CHD who have an implanted defibrillator and recurrent MMVT, VT storm, or multiple appropriate shocks, additional therapy including ATP, treatment with antiarrhythmic agents, and/or catheter ablation is indicated as adjunctive therapy to reduce the arrhythmia episodes. These therapies should be decided and initiated in an adequately trained centre. I LOE C
(3) In patients with CHD and sustained VAs who require surgical haemodynamic interventions, pre-operative electrophysiological testing and intra-operative ablation should be considered when adequate expertise is available. IIa LOE C
(4) Patients with good ventricular function, who are asymptomatic, have normal or near-normal ventricular haemodynamics and low-risk subtypes of CHD may reasonably be followed without advanced therapy and invasive evaluation despite the presence of moderately frequent and/or complex ventricular ectopy. IIb LOE C
(5) Catheter ablation may be appropriate for patients with CHD who have newly recognized or progressive ventricular dysfunction and a high burden of monomorphic ventricular ectopy. IIb LOE C

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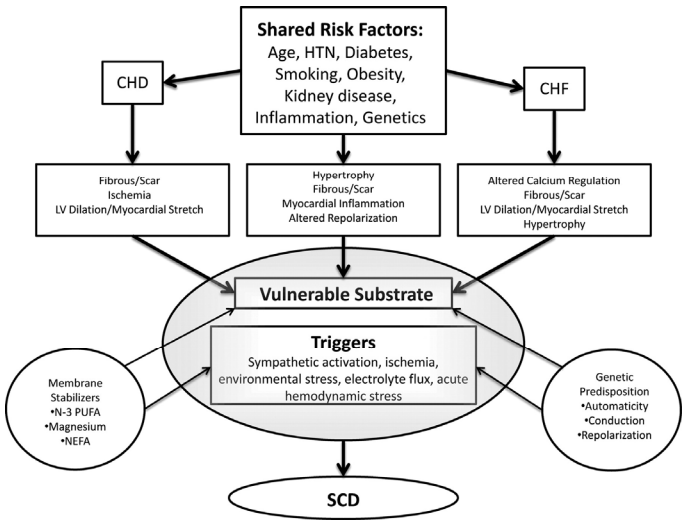
Critical pathways leading to electric instability and ventricular arrhythmias.



Deo R , and Albert C M *Circulation*. 2019;125:620-637

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Evidence for efficacy of early ablation for ventricular tachycardia

Table 2. Selected Clinical Trials of VT Ablation.*

Trial	Design	Study Population (Comparison)	No. of Patients	Months of Follow-up†	Outcome
SMASH VT,[49] 2007	RCT	Patients with ICM (ICD plus VT ablation vs. ICD alone)	128	22.5±5.5	Ablation superior (incidence of ICD therapy, 12% in ablation group vs. 33% in control group at 2 yr)
VTACH,[50] 2010	RCT	Patients with ICM (ICD plus VT ablation vs. ICD alone for stable VT)	107	22.5±9.0	Ablation superior (median time to recurrence of VT or VF, 18.6 mo in ablation group vs. 5.9 mo in control group)
VANISH,[48] 2016	RCT	Patients with ICM (VT ablation vs. escalation of antiarrhythmic-drug therapy for drug-refractory VT)	259	27.9±17.1	Ablation superior (primary composite end point of death, VT storm, or appropriate ICD shock, 59.1% in ablation group vs. 68.5% in control group)
Multicenter Thermocool VT Ablation Trial,[26] 2008	Observational	Patients with ICM	231	12	Catheter ablation of VT is a reasonable option for clinical management (freedom from recurrent VT, 53% at 6 mo)
IVTCC,[47] 2015	Retrospective	Patients with ICM or NICM	2061	12	Freedom from VT recurrence, 70% at 1 yr; transplantation-free survival, 90% for patients without recurrence vs. 71% for those with recurrence

* ICD denotes implantable cardioverter–defibrillator, ICM ischemic cardiomyopathy, IVTCC International VT Ablation Center Collaborative Group, NICM nonischemic cardiomyopathy, RCT randomized, controlled trial, SMASH-VT Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia, VANISH Ventricular Tachycardia Ablation versus Escalated Antiarrhythmic Drug Therapy, VF ventricular fibrillation, and VTACH Ventricular Tachycardia Ablation in Coronary Heart Disease.
† Plus–minus values are means ±SD.

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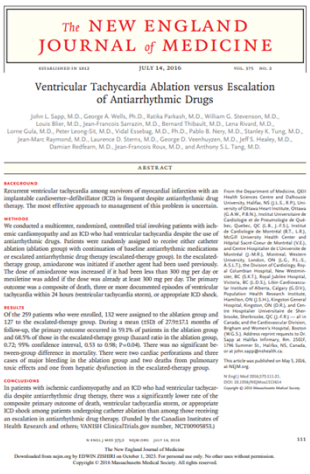
Please don't just escalate AAD Rx before considering ablation



The NEW ENGLAND JOURNAL of MEDICINE

JULY 14, 2016

Ventricular Tachycardia Ablation versus Escalation of Antiarrhythmic Drugs

ABSTRACT

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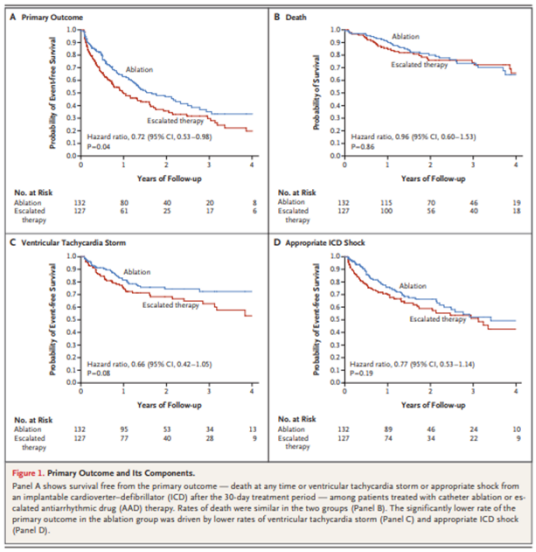

Figure 1. Primary Outcome and Its Components.

Panel A shows survival free from the primary outcome — death at any time or ventricular tachycardia storm or appropriate shock from an implantable cardioverter–defibrillator (ICD) after the 30-day treatment period — among patients treated with catheter ablation or escalated antiarrhythmic drug (AAD) therapy. Rates of death were similar in the two groups (Panel B). The significantly lower rate of the primary outcome in the ablation group was driven by lower rates of ventricular tachycardia storm (Panel C) and appropriate ICD shock (Panel D).

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Summary

- 1. Ventricular arrhythmias present in a variety of ways… some can be truly benign, some deceptively " benign" and others clearly malignant.
- 2. Structural heart disease especially coronary artery disease is a common underlying cause though there are many purely electrical variants.
- 3. PVCs can lead to cardiomyopathy and heart failure and at times cause sudden cardiac death.
- 4. When you have any doubt about whether your patient may benefit from ventricular tachycardia advanced therapies (medication/ catheter ablation) do not hesitate to refer them for evaluation.
- 5. Management of VT is the ultimate team sport and individual cases may require close collaboration between clinical cardiologists, advanced heart failure cardiologist, structural and interventional cardiologists, advanced imaging cardiologists, cardiac surgery, vascular surgery and EP.
- 6.(1-800-VEE-TACH)- We would like to become one of the world's preeminent centers for treatment of ventricular arrhythmias. We have all the necessary ingredients and in my view the future is ours for the taking.

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References and suggested reading

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ESC European Society of Cardiology

Europace (2019) **21**, 1143–1144
doi:10.1093/europace/euz132

CONSENSUS STATEMENT

2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias

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Thank you!

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