MHIF Research in 2017:

- **206** Active studies
- **683** Patients enrolled

**MHIF RESEARCH HIGHLIGHTS**

**WAY TO GO**

first MHIF patient enrolled in Accucinch trial

**KUDOS to the team…**

*Drs. Mudy and Eckman and Sara Olson*

**CONGRATS** to Dr. Lin

MHIF enrolled the first patient in the world in the KPL trial, putting our center in the lead for all sites

**175**

Publications in 2017 sharing learnings from MHIF research
Ventricular Arrhythmias
Overview on management and emerging technologies

January 29th, 2018

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Abbott Northwestern Hospital

Disclosure

I have no actual or potential conflict of interest in relation to this presentation. I have no relevant financial relationships in relation to this presentation.
Objectives

Approach to management of ventricular arrhythmias in
- the structurally normal heart
- structural heart disease

Review representative case studies
Discuss new and emerging technologies for the treatment of ventricular tachycardia

Outline

Structurally normal heart
- PVCs
- Idiopathic VT

Structural heart disease
- VT in post-MI patients
- VT in other cardiomyopathies
Outline

Structurally normal heart
- PVCs
- Idiopathic VT

Structural heart disease
- VT in post-MI patients
- VT in other cardiomyopathies

PVC prevalence in the structurally normal heart

Review of holter monitors from healthy volunteers during phase I clinical trials for non-cardiac drugs

<table>
<thead>
<tr>
<th>Morphology Abnormality</th>
<th>Total n (%) (n = 1273)</th>
<th>Age 18-45 Years (n = 1165)</th>
<th>Age 46-65 Years (n = 108)</th>
<th>P Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature ventricular complexes (PVCs)-total</td>
<td>552 (43.4)</td>
<td>475 (40.6)</td>
<td>79 (73.1)</td>
<td>&lt;.0001*</td>
</tr>
<tr>
<td>PVCs in studies with 3-lead Holter ECG**</td>
<td>199 (50.1)</td>
<td>170 (47.2)</td>
<td>29 (78.4)</td>
<td>.0021*</td>
</tr>
<tr>
<td>PVCs in studies with 12-lead Holter ECG**</td>
<td>353 (46.2)</td>
<td>303 (47.6)</td>
<td>50 (70.6)</td>
<td>.0001**</td>
</tr>
<tr>
<td>Multiform PVC</td>
<td>68 (5.3)</td>
<td>54 (4.6)</td>
<td>14 (13)</td>
<td>.003*</td>
</tr>
</tbody>
</table>

- N = 1273
- 10 had 1% PVC burden (1000 PVCs / 24 hours)
- 7 had 2% PVC burden (2000 PVCs / 24 hours)
- 12.3% had multifocal PVCS

PVC location

Most common site in those without structural heart disease is in the outflow tract.

Outflow region includes RVOT, LVOT, Aortic cusps, mitral valve annulus, aortic/mitral continuity, superior/epicardial LV.

Outflow PVC on EKG

Strongly positive QRS in inferior leads.
Mechanism of outflow VT

Delayed afterdepolarizations
- Increased levels of cyclic adenosine monophosphate increase intracellular calcium

Adrenergic stimulation increases cAMP via a stimulatory G-protein
- Beta-blockers inhibit this effect

CCBs inhibit slow-inward calcium current

Are isolated outflow PVCs a benign finding?

In general, probably yes
- Can sometimes be seen due to ARVC, sarcoidosis
- Very rarely reported with malignant arrhythmias / SCD
Increasing PVC burden can lead to LV dysfunction

174 consecutive patients with frequent idiopathic PVCs
- 24 holter monitoring, TTE

**PVC burden > 24%** was independently associated with PVC-induced cardiomyopathy

<table>
<thead>
<tr>
<th>PVC burden (%)</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>100</td>
<td>46</td>
</tr>
<tr>
<td>21</td>
<td>90</td>
<td>99</td>
</tr>
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<td>27</td>
<td>70</td>
<td>88</td>
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<td>30</td>
<td>60</td>
<td>91</td>
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<tr>
<td>34</td>
<td>50</td>
<td>91</td>
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<td>35</td>
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<td>94</td>
</tr>
<tr>
<td>39</td>
<td>30</td>
<td>95</td>
</tr>
<tr>
<td>43</td>
<td>20</td>
<td>96</td>
</tr>
<tr>
<td>50</td>
<td>10</td>
<td>99</td>
</tr>
</tbody>
</table>

RXC = receiver-operator characteristic; other abbreviations as in Table 2.


**Mechanism of PVC induced CMY**

Not necessarily a tachycardia-induced phenomenon

- Impaired calcium handling
- Dyssynchrony leading to adverse ventricular remodeling
  - Epicardial origin appears to be the only location independently associated with CMY
  - Less likely to be associated with outflow origins

Carballeira, Marchlinski, et al. VPD QRS duration as a new marker of risk for the development of VPD-induced CMY. Heart Rhythm 2014.
QRS width of PVCs as a prognostic factor for PVC-induced CMY

45 patients undergoing ablation

Carballa, Marchlinski, et al. VPD QRS duration as a new marker of risk for the development of VPD-induced CMY. Heart Rhythm 2014.

QRS width of PVCs may also be a prognostic factor for PVC-induced CMY

294 patients undergoing ablation

Are PVCs a modifiable risk factor?

69 patients with NICM (35% +/- 9%) undergoing PVC outflow tract ablation
- Mean follow-up 11 +/- 6 months

Degree of LVEF improvement correlated with degree of decline in PVC burden
- 5 patients with successful ablation did not have LVEF improvement

PVC suppression can improve LVEF

### When to intervene?

**Symptoms**

**Cardiomyopathy**

Less common indications:
- Impairment of bi-ventricular pacing
- PVCs causing malignant arrhythmias (R on T)

Generally do not need to treat asymptomatic patients with < 10% **burden**
- But asymptomatic patients with > 10% **burden**
  - Not clear
  - Risk/benefit discussion of monitoring vs treatment
  - Monitoring: serial echocardiogram, Holter monitor

### PVC management

**Beta-blockers**

**Diltiazem**

**Verapamil**

- Efficacy limited, but low risk
Limited data for medical therapy

Double-blind RCT. 52 patients with PVCs (average burden 20% by 24 hr Holter) randomized to atenolol vs placebo for 1 month
- structurally normal hearts
- PVC appeared outflow by EKG (LBBB, inf axis)

Atenolol group
- decreased PVC count (average 24K to 16K)
- Decreased symptoms
- no change in QOL

Placebo
- No change in PVC count
- decreased symptoms
- no change in QOL


PVC management

Beta-blockers
- Diltiazem
- Verapamil
  - Efficacy limited, but low risk

Anti-arrhythmics
- Flecainide, amiodarone, sotalol
  - Possibly higher efficacy
  - Risk of pro-arrhythmia
  - Toxicities

ICDs generally not indicated
- Catheter ablation
Cather Ablation (CA) by the Guidelines

EHRA/HRS/APHRS Expert Consensus on Ventricular Arrhythmias 2014
- CA may be beneficial by improving symptoms or LV dysfunction in patients suffering from frequent non-sustained VAs (eg PVC burden 10%), IIA LOE B

ESC Guidelines for the Management of Patients with Ventricular Arrhythmias 2015
- CA of RVOT PVC recommended in patients with symptoms; failure of drug therapy; or decline in LV function due to PVC burden (I, LOE B)
- CA of LVOT or epicardial PVCs recommended in symptomatic patients after failure of one class IC agent or in patients not wanting long-term therapy. (IIa, LOE B)

Localization by EKG

Difficult to exactly differentiate due to number of structures (in close proximity) in the outflow area

http://www.bq.ic.ac.uk/Staff/khparker/homepage/BSc_lectures/2002/Heart_anatomy.jpg
Differentiating RVOT vs left-sided PVCs

V1 morphology


R/S transition of PVC occurring later than sinus transition excludes LVOT origin

LV summit

Most superior epicardial portion of the LV
Often difficult to reach by catheter
LV summit

Can sometimes access by:
- LCC or the endocardial aorto-mitral continuity
- Coronary sinus (great cardiac vein or anterior IV vein)
  - Proximity to coronary arteries
- Pericardial access
  - Obstruction by epicardial fat, coronary arteries

Yamada et al. Challenging RF catheter ablation of idiopathic ventricular arrhythmias originating from the LV summit near the LMA. Circ Arrhythm Electrophysiol 2016.

Procedural success of catheter ablation

Retrospective, 1185 patients from 8 centers between 2004 - 2013

Average acute procedural success 84%

Long-term (mean 1.9 years) success without anti-arrhythmics: 71%
Long-term success with anti-arrhythmics: 85%

Only significant predictor of long-term success: RVOT location

Procedural complications of catheter ablation

![Pie chart showing distribution of major complications](chart.png)


Case #1

58 y/o F with dyspnea, palpitations
- Echocardiogram with LVEF 34% (previously normal)

![Holter monitor with 15,000 PVCs (over 24 hours)](holter.png)

A. Cardiac MRI
B. Ischemic evaluation
C. ICD
D. Medical therapy
E. Catheter ablation
F. Observation only
Case #1 continued

58 y/o F with dyspnea, palpitations
- Echocardiogram with LVEF 34%

Holter monitor with 15,000 PVCs (over 24 hours)

Initiated on sotalol with great response
Not interested in long-term medical therapy and presented for ablation
Localized to aortic-mitral continuity, underneath LCC
Has been doing well, repeat echocardiogram pending
Case #2

40 y/o healthy male presents with dyspnea. Echo is normal.

Outline

Structurally normal heart
- PVCs
- **Idiopathic VT**

Structural heart disease
- VT in post-MI patients
- VT in other cardiomyopathies

Spontaneously converts

How to manage?
- Verapamil
- Metoprolol
- Anti-arrhythmic
- Ischemic evaluation
- Cardiac MRI
- ICD
Left posterior fascicular VT

Approximately 90% of all fascicular VTs

Idiopathic VT

Sustained VT in the structurally normal heart
Accounts for approximately 10% of all sustained VT
- Outflow tract VT
- Papillary muscle VT
- Fascicular VT

Risk of SCD is low
Generally tolerated by the patient
Good prognosis
ICD for VT in structurally normal hearts?

Risk of sudden cardiac death is low

EHRA/HRS Ventricular Arrhythmias Consensus 2014
- In the absence of SHD, SMVT is generally associated with an excellent prognosis

AHA/HRS 2012 Device Consensus:
- ICD therapy is not indicated when VF or VT is amenable to surgical or catheter ablation (e.g. RV or LV outflow tract VT, idiopathic VT or fascicular VT in the absence of structural heart disease)

Fascicular VT

Verapamil-sensitive VT

Re-entry circuit involving calcium-dependent Purkinje fibers along septal side of LV

Often seen in young, healthy males

Often exercise-induced
Management of fascicular VT

CCB (verapamil)
- Breakthrough rate up to 20%
Lower efficacy for beta-blockers
Class I/III anti-arrhythmics

Catheter ablation of fascicular tachycardia

Long-term success rates of approximately 70%
Recurrence rate as high as 20%

Outline

Structurally normal heart
- PVCs
- Idiopathic VT

**Structural heart disease**
- VT in post-MI patients
- VT in other cardiomyopathies

VT in the structurally abnormal heart

As opposed to the prior discussion, carries elevated risk for SCD

ICDs are standard of care

Catheter ablation can help minimize ICD therapies and improve symptoms in the appropriately selected patient
Outline

Structurally normal heart
- PVCs
- Idiopathic VT

Structural heart disease
- VT in post-MI patients
- VT in other cardiomyopathies

Post-MI ventricular tissue

Endomyocardial scar provides a re-entrant substrate for VT circuits

Border zone tissue
- Bed of surviving myocardial tissue interspersed with fibrotic tissue
- Decreased cell-to-cell coupling
- Slow electrical conduction which predisposes to re-entrant circuits

Ideal VT circuit

Single entrance, central isthmus and exit site

Healthy Tissue

Dense Scar

Border Zone Tissue

Reality

Multiple pathways can exist, with multiple VT circuits
12 lead ECG depends on the exit site of the circuit

Outline

Structurally normal heart
- PVCs
- Idiopathic VT

Structural heart disease
- VT in post-MI patients
- VT in other cardiomyopathies
Other processes may lead to myocardial scar

- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- ARVC/D
- Cardiac sarcoidosis
- post-surgical scar

Exit site of re-entrant scar in post-MI VT is usually subendocardial

- Non-MI VT can often have mid-myocardial or epicardial circuits

This distinction has consequences for catheter ablation

https://www.slideshare.net/faminteractive/a-fib-treatment-strategies

Dilated cardiomyopathies

Exit site for dilated cardiomyopathies can often be mid-myocardial or epicardial

Although free wall of LV is thinned, endocardium is relatively preserved (epicardial contours are traced).

Endocardial ablation was unsuccessful. Epicardial ablation was successful.

ARVC/D

Replacement of myocardium by fibrofatty tissue

Begins subepicardially and moves towards endocardial surface

Sustained VT in 1/3 of patients

HCM

Slow conduction due to myocyte disarray

VT uncommon in the general HCM population

Highest risk in those with LV aneurysms

- Approximately 30% of HCM patients with LV aneurysms and primary prevention ICDs experience monomorphic VT

Endocardial ablation may be limited due to thick myocardium
Treatment of VT in structural heart disease

ICD therapy

Medications

Ablation

ICD Therapy

Meta-analysis of three RCTs for ICD vs AAD (AVID, CASH, CIDS)

Medical Therapy of VT in SHD

Aside from beta-blockers, no RCT evidence that AAD improves survival (primary or secondary prevention)
- Role is for arrhythmia/symptom control

BB: good safety profile
- adrenergic-receptor blockade
- slowing of sinus rate
- possible inhibition of excess calcium release
- can enhance anti-arrhythmic efficacy

CCB:
- Can be helpful in outflow VT, fascicular VT
- Otherwise, no role

AAD Therapy of VT in SHD

Amiodarone
- Up to 34% reduction in appropriate ICD therapies
- Up to 70% reduction in inappropriate ICD therapies

Sotalol
- 15-44% reduction in recurrent VT

Medical Therapy of VT in SHD

No mortality benefit to anti-arrhythmic therapy for VT
- Amiodarone may increase mortality
- Toxicities/adverse events from amiodarone as high as 30%

Ranolazine:
- Primarily late Na blockade
- may reduce VT/shocks, but clinical data is minimal

Mexiletine

Quinidine
Catheter Ablation

VERY brief overview of catheter ablation in EP lab

Creating a 3-dimensional shell

Voltage Mapping

Color-coded map based upon voltage amplitude of the electrogram recorded by the mapping catheter
- Normal myocardium: > 1.5 mV (purple)
- Border zone tissue: 0.5 – 1.5 mV
- Dense scar: < 0.5 mV (red)

Ablation of the clinical VT vs substrate modification

Di Biase, Natale et al. VISTA trial. JACC 2015.
Catheter ablation in VT with SHD

When should we consider CA?

Is there a role for CA in lieu of AAD?
- SMASH-VT
- VTACH
- Substrate Modification Study
- VANISH

---

SMASH-VT

Multi-center RCT, 128 pts with history of prior MI with VT/VF (either clinical; syncope with inducible VT; or after appropriate ICD shock)
- ICD alone
- ICD + CA (substrate modification)

Primary Endpoint: Survival free from any appropriate ICD therapy

Excluded pts on class I/III AADs

SMASH-VT

65% reduction in rate of appropriate ICD therapy in CA group
- 21 patients received therapy in the control group (33%) and 8 in the ablation group (12%) (P=0.007)
- No significant difference in mortality

VTACH

European multi-center RCT. 110 pts with h/o MI, EF <= 50%, first episode of stable VT
- ICD alone
- CA, followed by ICD (both clinical VT ablation and substrate modification)

Primary Endpoint: time to first VT/VF recurrence
35% of patients in each group on amiodarone
VTACH

freedom from VT: 47%
- HR 0.61; (95% CI 0.37–0.99); p 0.045
- Guided by change in those with LVEF > 30% (though smaller # of events in LVEF < 30%


Substrate Modification Study

Multi-center RCT, 111 pts post-MI, LVEF <=40%, hemodynamically unstable VT presenting with VT/VF or syncope with induced VT
- ICD only
- CA, followed by ICD (both clinical VT ablation and substrate modification)

Primary endpoint: time to first VT/VF recurrence
~30% on AAD

Substrate Modification Study

No difference in time to first VT/VF recurrence (49% CA + ICD; 52.4% ICD)

However, in those with CA: > 50% reduction in total ICD interventions over the follow-up period

<table>
<thead>
<tr>
<th></th>
<th>Ablation (n=56)</th>
<th>ICD Only (n=57)</th>
<th>Hazard Ratio (95% CI)</th>
<th>Hazard Ratio (95% CI)†</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous VT/VF</td>
<td>2.8±6.0</td>
<td>8.1±19.1</td>
<td>0.43 (0.22-0.85)</td>
<td>...</td>
<td>0.015</td>
</tr>
<tr>
<td>Spontaneous VT/VF with ATP or shock</td>
<td>2.8±8.4</td>
<td>12.9±34.6</td>
<td>0.34 (0.18-0.65)</td>
<td>...</td>
<td>0.001</td>
</tr>
<tr>
<td>VT/VF episode with ATP or shock</td>
<td>1.8±4.8</td>
<td>7.0±18.6</td>
<td>0.33 (0.15-0.69)</td>
<td>...</td>
<td>0.004</td>
</tr>
</tbody>
</table>


Three RCTs demonstrating that catheter ablation may be of benefit in those with post-MI ventricular tachycardia

These trials were not designed to assess efficacy of CA in comparison to use/escalation of anti-arrhythmic medications.
VANISH

Multi-center RCT, 259 pts with ICM s/p ICD p/w VT despite AAD
- CA plus continuation of current AAD regimen
- Escalation of AADs
  - Start amiodarone (if non-amiodarone AAD)
  - Increase amiodarone (if < 300mg daily)
  - Add mexiletine (if amio >= 300mg daily)

PE: composite of death, VT storm or appropriate ICD shock
mean follow-up 28 months


[Graph showing survival outcomes for Ablation and Escalated therapy]

Primary endpoint (composite death, VT storm, ICD shock) reduced by 28% with CA
- driven by lower rates of VT storm and appropriate ICD shocks

### Complications

<table>
<thead>
<tr>
<th>Event*</th>
<th>AAD Group (n=127) No. (%)</th>
<th>Catheter Ablation Group (n=122) No. (%)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vascular injury†</strong></td>
<td></td>
<td></td>
<td>0.25</td>
</tr>
<tr>
<td>Major Bleeding</td>
<td>1 (0.8)</td>
<td>3 (2.3)</td>
<td>0.62</td>
</tr>
<tr>
<td>Cardiac Perforation</td>
<td>1 (0.8)</td>
<td>2 (1.5)</td>
<td>1.00</td>
</tr>
<tr>
<td>Endocarditis</td>
<td>1 (0.8)</td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td>Heart Block</td>
<td>1 (0.8)*</td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td><strong>Arrhythmogenic Drug Related</strong>&lt;br&gt;Death</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary toxicity</td>
<td>2 (1.6)</td>
<td></td>
<td>0.24</td>
</tr>
<tr>
<td>Liver toxicity/multiorgan failure</td>
<td>1 (0.8)</td>
<td></td>
<td>0.49</td>
</tr>
<tr>
<td>Pulmonary Infiltrate</td>
<td>2 (1.6)**</td>
<td></td>
<td>0.24</td>
</tr>
<tr>
<td>Shortness of Breath</td>
<td>3 (2.4)</td>
<td>1 (0.8)</td>
<td>0.36</td>
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<tr>
<td>Heart Failure Admission</td>
<td>1 (0.8)</td>
<td>3 (2.3)</td>
<td>0.62</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>5 (3.9)</td>
<td>3 (2.3)</td>
<td>0.49</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>5 (3.9)*</td>
<td>2 (1.5)</td>
<td>0.27</td>
</tr>
<tr>
<td>Hepatic Dysfunction</td>
<td>6 (4.7)</td>
<td></td>
<td>0.013</td>
</tr>
<tr>
<td>Tremor/Ataxia</td>
<td>6 (4.7)</td>
<td></td>
<td>0.013</td>
</tr>
<tr>
<td>Drug Therapy Change</td>
<td>6 (4.7)</td>
<td></td>
<td>0.013</td>
</tr>
<tr>
<td>Other adverse events no. (%)</td>
<td>6 (4.7)**</td>
<td>4 (3.0)</td>
<td>0.53</td>
</tr>
<tr>
<td><strong>TOTAL PATIENTS</strong></td>
<td>39 (30.7)</td>
<td>20 (15.2)</td>
<td>0.0031</td>
</tr>
<tr>
<td><strong>TOTAL EVENTS</strong></td>
<td>51</td>
<td>22</td>
<td>0.0033</td>
</tr>
</tbody>
</table>

**VANISH Trial supplementary appendix**

### CA and mortality benefit

None of these trials show CA provides a mortality benefit
CA and mortality benefit

Retrospective, 2061 pts with SHD undergoing VT ablation
Combined transplant and mortality rate 15% at one year
One year freedom from VT: 70% (72% ischemic; 68% NICM)
  - Higher VT recurrence: Lower LVEF, advanced NYHA, multiple VT morphologies


Outcome of catheter ablation

1 yr recurrence ranging 23% - 49% (many pts remaining on AADs)

Marchlinski et al. Long-Term Success of Irrigated RF CA of Sustained VT. JACC 2016.
Outcome of catheter ablation

Improved success when substrate modification is performed in addition to ablation of clinical VT


Outcomes: ICM vs Dilated cardiomyopathy

Better outcomes with post-MI VT

Dinov et al. HELP-VT. Circulation 2014.
Why less success in dilated cardiomyopathy?

Location of scar


Dilated cardiomyopathy outcomes

Better outcomes with epicardial ablation

Complications

Major complications ~7%
- Cardiac perforation 1.5%
- Vascular injury 2.3%
- Death 3%
- Stroke/TIA 0.5%
- Coronary artery injury 0.2%

Hemodynamic decompensation 11%

Uncontrollable VT 2.6%

30 day mortality after VT ablation 5%
1 year mortality after VT ablation 18%
- Recurrent VT
- Refractory heart failure

Summary of catheter ablation

CA provides symptomatic benefit
CA can minimize ICD therapies

No prospective data regarding AAD vs CA as first-line treatment, but should always be considered
- May be first line treatment (in addition to ICD) in the select patient
- May provide an amiodarone-free treatment modality

Higher efficacy for post-MI ventricular tachycardia
- Increased use of epicardial techniques

CA does carry non-trivial risk
- Patients should be chosen carefully
- Technology continues to improve safety/efficacy
  - Improved 3-dimensional mapping software/hardware
  - Improvement in catheter technology (irrigation, contact force)
  - Pre-procedural planning with cardiac imaging
Case #3

77 y/o M with distant inferior MI presenting with palpitations

 Converted after IV amiodarone bolus

Cardiac imaging: LVEF 40% with inferior transmural scar
LHC without significant disease

Case #3 continued

ICD implanted

Post-operatively, continued to have intermittent sustained episodes of monomorphic VT

Active and “young” 77 y/o

Taken for catheter ablation
- Voltage mapping with inferior scar
- Clinical VT was successfully ablated, with additional substrate modification performed

No AADs used

No recurrence over 3 months of follow-up
Emerging Technologies

Sparse data
Safety profile unclear
Efficacy unclear
Trials underway
All of these therapies attempt to deliver deeper lesions

Transcoronary ethanol ablation

Used in some centers after failed endocardial and epicardial ablation
Requires appropriate coronary anatomy
Most useful for deep, intramural VT (eg septal)
High incidence (> 30%) of CHB in septal VT
Transcorony ethanol ablation

Basal septal scar. Failed endocardial ablation (ablated from both LV and RV septum)

Occlusive balloon in first septal perforator with ethanol injected

Dashed circle at area of exit site of VT


Bipolar RF ablation

Two ablation catheters placed on opposing sides of tissue to produce a larger (and, hopefully, transmural) lesion

? Increased risk for perforation, LV dysfunction

RCT, currently recruiting

Intramural needle ablation catheter

Needle electrode advanced into myocardium
- **Intramural RF lesion created**

? Increased risk for perforation, LV dysfunction

RCT, currently recruiting

---

**Stereotactic radioablation**

Used in the management of solid tumors

Delivers **precise** and high-dose radiation with minimal damage to adjacent tissue

Completely “**non-invasive**” methodology applied to treatment of VT:
- 25-35 Gy delivered in a single fraction, recapitulating effects of catheter RF ablation
- Off-label use
- Results in **late myocardial fibrosis** and electrically inert tissue without damage to nearby tissue

5 patients with structural heart disease s/p ICD presenting with refractory VT
High-risk patients with limited options

---


**Methods**


---

**Identify Arrhythmogenic Scar Substrate**
Create a contoured target volume

**Develop Plan**

---

**Treat**
Stereotactic radioablation

During 3 months prior to treatment, combined history of 6577 VT episodes

6 week "blanking period": 680 episodes of VT

After blanking period: 4 episodes of VT over 46 months

Stereotactic radioablation

No decrease in mean LVEF

Mild inflammatory lung changes at 3 months, which had resolved at 1 year
Take-home points

PVCs
- Outflow tract PVCs are generally benign
- Consider intervention when patient is symptomatic or LVEF is reduced
- Burden generally > 10-20%

Idiopathic VT
- Generally excellent prognosis
- Can respond well to medications or catheter ablation
- ICD generally not indicated

VT in structural heart disease
- ICD is standard of care
- Catheter ablation has the ability to minimize ICD therapy and, in some patients, minimize AAD use
- CA in ICM: generally better outcome due to endocardial scar
- Risk is non-trivial and patients must be properly selected

Thank you