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

Edwin. T. Zishiri, M.D.

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

I have no conflicts of interest to disclose.
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.

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.
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.

Baseline ecg
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

Mapping to find a good site to ablate.
Ablation

PVC was forever silenced!

Activation Map and ablation lesion
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.

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.
Clinical ECG

Baseline ecg with PVCs in lab
Good site to ablate?

Aortic cusps
Aortic cusps

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

Ablation
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.

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.
12 lead of PVC

Intracardiac echo
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.

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.
Clinical PVCs

Slurring of the initial portion of the QRS and delayed activation

- Pseudo delta $\geq 34$ ms
- Intrinscoind deflection time $\geq 85$ ms
- Shortest RS complex in precordial leads $\geq 121$ ms
- Maximum deflection index $\geq 0.55$
- QS or rS in lead 1

Went straight for mapping epicardial surface of heart
Too close for comfort

Ablation
Small marginal branch sacrificed

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.
ECG on presentation

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?
Ablation

LV map and Ablation lesions
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.

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.
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.

Baseline ecg
Induction of tachycardia
500-290-240

Tachycardia CL
What do you think about this site for ablation

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
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

How about here

47

48
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

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

Pacing During VT

Concealed Entrainment

Manifest Entrainment

PPI–VTCL (±30 msec) or S-QRS–EG-QRS (±20 msec)

Yes

No

(S-QRS/VTCL) X 100

<30% 31-50% 51-70% >70%

Exit Central Proximal Inner Loop Adjacent Bystander

Termination with ablation

Perfect pacemap of VT

RV map and ablation points
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.

Not inducible at end with triple extrastimuli
(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.

Induction of tachycardia

BP 40/0 patient needs emergent cardioversion
Now what?

Criteria for epicardial VT circuit

Slurring of the initial portion of the QRS and delayed activation
- Pseudo delta $\geq 34$ ms
- Intrinsicsoid deflection time $\geq 85$ ms
- Shortest RS complex in precordial leads $\geq 121$ ms
- Maximum deflection index $\geq 0.55$
- QS or rS in lead 1

Decide to go epicardial

Making sure to stay away from the grafts and coronary arteries
Making sure to stay away from the grafts and coronary arteries

Making sure to stay away from the grafts and coronary arteries
Making sure to stay away from potential collateral damage

Hemodynamic support with epicardial ablation in a post CABG patient
On epicardial surface of heart with impella

After extensive epicardial ablation and aposing endocardial surface ablation

SUCCESS!!!!!
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 ablation. Kaplan–Meier curves comparing freedom from VT recurrence according to groups. Patients who had the combined endpoint of VT noninducibility and LP ablation compared with inducible patients exhibited a significantly lower incidence of VT recurrence (16.8% vs. 47.4%; p < 0.001). Among noninducible patients, those with additional LP ablation 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 ablation (hazard ratio, 0.205, p < 0.001) was independently associated with VT recurrence and cardiac death (hazard ratio, 0.104, p = 0.001).

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


Scar Dechanneling

Inferior view of bipolar voltage electroanatomical substrate maps during sinus rhythm before (MAP) and after (pMAA) scar dechanneling in a patient with healed myocardial infarction. Electrophysograms 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 (5D), repeated electrogram recordings at the same sites demonstrated elimination of the delayed component (asterisk). Kaplan–Meier curve showed a higher 2-year probability of ventricular sympathetic or sudden death event-free survival (88% versus 62%) according to the complete versus incomplete elimination of conducting channel electrograms.

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

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
Tachycardia with entrainment

Automaticity with ablation
Brief History

- 57 year old man with Waldemström’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.
History continued

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

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

- Now let's review the guidelines.
Considerations for NSVT in “Normal hearts”

<table>
<thead>
<tr>
<th>NSVT clinical presentation</th>
<th>ECG</th>
<th>Risk of sudden cardiac death</th>
<th>Alternative diagnostic considerations</th>
<th>Treatment</th>
<th>Treatment to be considered</th>
<th>Key references</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical NSVT</td>
<td>BBB, left atrial fibrillation, atrial flutter, or atrial tachycardia</td>
<td>Very rare</td>
<td>Standard</td>
<td>Differentiate from AICD</td>
<td>Beta-blocker, amiodarone, ICD with or without mortality, ICD</td>
<td>Catheter ablation, ICD</td>
</tr>
<tr>
<td>Typical NSVT</td>
<td>BBB, left atrial fibrillation, atrial flutter, or atrial tachycardia</td>
<td>Very rare</td>
<td>Standard</td>
<td>Differentiate from AICD</td>
<td>Beta-blocker, amiodarone, ICD with or without mortality, ICD</td>
<td>Catheter ablation, ICD</td>
</tr>
<tr>
<td>Uniphasic ventricular tachycardia, Other Form VT</td>
<td>BBB, left atrial fibrillation, atrial flutter, or atrial tachycardia</td>
<td>Very rare</td>
<td>Standard</td>
<td>Differentiate from AICD</td>
<td>Beta-blocker, amiodarone, ICD with or without mortality, ICD</td>
<td>Catheter ablation, ICD</td>
</tr>
<tr>
<td>Exercise</td>
<td>Multiple</td>
<td>Very rare</td>
<td>Standard</td>
<td>Differentiate from AICD</td>
<td>Beta-blocker, amiodarone, ICD with or without mortality, ICD</td>
<td>Catheter ablation, ICD</td>
</tr>
<tr>
<td>Athlete</td>
<td>Multiple</td>
<td>Very rare</td>
<td>Standard</td>
<td>Differentiate from AICD</td>
<td>Beta-blocker, amiodarone, ICD with or without mortality, ICD</td>
<td>Catheter ablation, ICD</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy, Ventricular Polymorphic VT</td>
<td>Multiple</td>
<td>Very rare</td>
<td>Standard</td>
<td>Differentiate from AICD</td>
<td>Beta-blocker, amiodarone, ICD with or without mortality, ICD</td>
<td>Catheter ablation, ICD</td>
</tr>
</tbody>
</table>

AICD = automatic internal cardioverter defibrillator; BBB = bi-directional block; VT = ventricular tachycardia; NSVT = non-sustained ventricular tachycardia; VT = ventricular tachycardia; SVT = supraventricular tachycardia; ICD = implantable cardioverter defibrillator; CRT = cardiac resynchronization therapy; LV = left ventricle; MRI = magnetic resonance imaging; EP = electrophysiology; PVC = premature ventricular complexes; CRT = cardiac resynchronization therapy; ICD = implantable cardioverter defibrillator; LV = left ventricle; MRI = magnetic resonance imaging; delayed enhancement; PE = physical examination; PVC = premature ventricular complexes; CRT = cardiac resynchronization therapy; SHD = structural heart disease; VAs = ventricular arrhythmias.
Considerations for NSVT in the presence of structural heart disease

Table 4. Non-sustained ventricular tachycardia in structural heart disease

<table>
<thead>
<tr>
<th>Clinical setting</th>
<th>Risk of sudden cardiac death</th>
<th>Arrhythmia specialties evaluation</th>
<th>Diagnostic evaluation</th>
<th>Diagnostics to be considered</th>
<th>Treatment</th>
<th>Treatment to be considered</th>
<th>Key references</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACS within 4 h</td>
<td>No increased risk</td>
<td>No</td>
<td>Monitoring</td>
<td>Beta-blockers</td>
<td>ICD</td>
<td></td>
<td>Zipes et al.</td>
</tr>
<tr>
<td>ACS after 48 h</td>
<td>Increased risk</td>
<td>Yes</td>
<td>Consider EPS if moderate LV dysfunction</td>
<td>Beta-blockers</td>
<td>ICD</td>
<td></td>
<td>Zipes et al.</td>
</tr>
<tr>
<td>Previous HFr EF c. 30</td>
<td>Increased risk</td>
<td>Yes</td>
<td>Continued evaluation for repetitive arrhythmias</td>
<td>ICD, see relevant guidelines</td>
<td>Antiarhythmic therapy or ablation with ICD</td>
<td>Zipes et al.</td>
<td></td>
</tr>
<tr>
<td>Syncope (not chronic)</td>
<td>Increased risk</td>
<td>Yes</td>
<td>EP testing, ischaemia testing</td>
<td>Non-invasive EP testing</td>
<td>ICD with Inducible VT, VT/ VF</td>
<td>Additional antiarrhythmic therapy or ablation with ICD</td>
<td>Zipes et al.</td>
</tr>
<tr>
<td>Non-ischemic dilated CHF</td>
<td>Increased risk</td>
<td>Yes</td>
<td>Uncertain</td>
<td>EP testing, CRT-D</td>
<td>Beta-blocker, ICD, CRT-D</td>
<td>ICD, see relevant guidelines</td>
<td>Zipes et al.</td>
</tr>
<tr>
<td>3D DSE</td>
<td>Increased risk</td>
<td>Yes</td>
<td>Genetic screening</td>
<td>Genetic screening</td>
<td>Beta-blocker, CRT-D</td>
<td>ICD, see relevant guidelines</td>
<td>Zipes et al.</td>
</tr>
<tr>
<td>Short QT syndrome</td>
<td>Increased risk</td>
<td>Yes</td>
<td>Non-invasive EP testing</td>
<td>Non-invasive EP testing</td>
<td>Beta-blocker, CRT-D</td>
<td>ICD, see relevant guidelines</td>
<td>Zipes et al.</td>
</tr>
<tr>
<td>Short QT syndrome</td>
<td>Increased risk</td>
<td>Yes</td>
<td>Genetic screening</td>
<td>Genetic screening</td>
<td>Beta-blocker, CRT-D</td>
<td>ICD, see relevant guidelines</td>
<td>Zipes et al.</td>
</tr>
</tbody>
</table>
| e176

Sustained monomorphic VT

Figure 1. Sustained monomorphic ventricular tachycardia evaluation and management. ICD = implantable cardioverter-defibrillator; SHD = structural heart disease; VT = ventricular tachycardia.
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 angiography. 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 VT, 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 VTs. Detailed guidance can be found in the APHRS/EHRA/PHRS document on inherited arrhythmia syndromes. Ila LOE B
3. For patients with VT/VF storm, reversible factors such as electrolyte abnormalities, pro-arrhythmic drugs, ischemia, 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. Ilia 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 anesthesia 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

---

Patients resuscitated from VT/VF

---

Pedersen et al. EHRA/HRS/APHRS Expert Consensus on Ventricular Arrhythmias
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 infants 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 WVT, VT storms, 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. Peri-procedural 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]

Pedersen et al. EHRA/HRS/APHRS Expert Consensus on Ventricular Arrhythmias

Critical pathways leading to electric instability and ventricular arrhythmias.

Shared Risk Factors:
- Age
- HTN
- Diabetes
- Smoking
- Obesiy
- Kidney disease
- Inflammation
- Genetics

Vulnerable Substrate

Triggers:
- Hypoxia
- Ischemia
- Inflammatory stress
- Electrolyte flux
- Acute hemodynamic stress

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

Copyright © American Heart Association, Inc. All rights reserved.
Evidence for efficacy of early ablation for ventricular tachycardia

Please don’t just escalate AAD Rx before considering ablation
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.
References and suggested reading

Thank you!