Ventricular tachycardia ablation in the absence of structural heart disease

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19ο Πανελλήνιο Καρδιολογικό Συνέδριο ΚΕΒΕ

Disclosure
I have no conflict of interest to declare in relation to this presentation
VT classification

1. Duration and hemodynamic consequences
   - Non sustained
   - Sustained → lasts ≥30 s, causes syncope or haemodynamic compromise requiring therapy

2. Morphology

3. Mechanism
   - Reentrant
   - Triggered activity
   - Automatic
4. Underlying substrate

- Structural abnormalities
  - Ischaemic
  - Dilated
  - HCM + other cardiomyopathies
- Channelopathies
  - Brugada
  - Long QT
  - Short QT syndrome
  - Early Repolarisation Syndrome
  - CPVT
- “Normal” heart
  (based on ECG, Echo, CAA)

Modified
Klein et al.
Circulation 1992
Age-related risk for sudden cardiac death


<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>No.</th>
<th>OTT/FT</th>
<th>Mean FU</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>Josephson</td>
<td>1983</td>
<td>30</td>
<td>OTT</td>
<td>30</td>
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<tr>
<td>Wellens²</td>
<td>1989</td>
<td>52</td>
<td>OTT 60%  FT 40%</td>
<td>96</td>
<td>1 dead carcinoma</td>
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<tr>
<td></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
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<td>3 anti-tachy devices</td>
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<tr>
<td>Shimomura³</td>
<td>1995</td>
<td>37</td>
<td>FT</td>
<td>72</td>
<td>1 SD post AT device</td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td>1 cryosurgery</td>
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<td></td>
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<td></td>
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<td>4 cath ablation</td>
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<tr>
<td>Petrac⁴</td>
<td>2002</td>
<td>20</td>
<td>17 OT 3 FT</td>
<td>56</td>
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<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>0 dead</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 failed ablations</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>2 recurrence</td>
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<tr>
<td>Pfammatter⁵</td>
<td>1999</td>
<td>98</td>
<td>LBBB 71 RBBB 27</td>
<td>47</td>
<td>0 dead</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>25 no drugs</td>
</tr>
<tr>
<td>Chang⁶</td>
<td>1997</td>
<td>61</td>
<td>RBBB 31 LBBB 30</td>
<td>29</td>
<td>1 dead non-CV</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>84% ablated</td>
</tr>
<tr>
<td>Shibate⁷</td>
<td>1997</td>
<td>13</td>
<td>RVOT</td>
<td>28</td>
<td>0 dead</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>All ablated</td>
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<tr>
<td>Chang⁸</td>
<td>1996</td>
<td>26</td>
<td>RBBB 14 LBBB 12</td>
<td>95</td>
<td>0 dead</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>14 ablated</td>
</tr>
</tbody>
</table>

Idiopathic VT prognosis

Evaluation of patients with ventricular tachycardia

- **VT classification** (morphology – duration – hemodynamic consequences)
- **History** (including family) and physical examination
- **12 lead ECG VT and SR**
- **Echocardiogram**
- **Exercise testing**
  - Ambulatory electrocardiography
  - **Cardiac MRI** - CT
- Imaging tests for ischemia (SPECT, stress echo, stress CMR, CTCAA)
- **Coronary angiogram**
- **Genetic testing**
- **Signal averaged ECG**
- Electrophysiology study (voltage mapping)
- **Drug challenges**
- **Right ventricular biopsy**
- **Blood test** (BNP has been proposed to distinguish idiopathic VT from ARVC)
Classification of ventricular tachycardia in the absence of structural heart disease

Ventricular Tachycardia

No Structural Heart Disease

Monomorphic

Polymorphic

Outflow Tract
Annular
Purkinje-related
Papillary Muscle

Long QT Syndrome
Short QT Syndrome
Brugada Syndrome
ER Syndrome

Ventricular tachycardia in the absence of structural heart disease
Killu et al.
BMJ 2018
Idiopathic VT sites of origin

- Outflow tract VT
- Mitral and tricuspid annular VT
- Papillary muscle VT
- Fascicular VT
ECG

LV free wall - RBBB
RV, septum - LBBB
Basal - positive precordial transition
Apical - negative precordial transition
Inferior axis - outflow tract
Superior axis – posterior
Epicardium
- Pseudodelta wave ≥ 34msec
- MDI ≥ 0.55

LBBB or RBBB morphology
Inferior leads
Precordial transition
Lead I

*Lead I threedimensionality
  anterior - posterior
  left - right
Outflow tract VT

- 80-90% of idiopathic VT
- 30-60 years of age
- Women RVOT
- Men aortic cusps
- Palpitations
- Few with syncope → 10%
- Physical or emotional stress, menstrual cycles
- Most with ectopics or NSVT
- Fewer with exercise induced VT
- CMR may reveal mild structural abnormalities of the RV primarily involving the free wall (focal thinning, fatty infiltration, and wall motion abnormalities)
- “Excellent” prognosis
  - But some reports of SCD in literature
  - Some patients have very fast tachycardia
Outflow Tract VT

Non-Sustained  ←  Sustained VT

↑

Autonomic Nervous System (hormones – stress)

Isolated VE’s  ←  Non-Sustained  ←  Sustained VT
DDx of RVOT tachycardia

- Atriofascicular fibers
- AVRT using Rt-sided accessory pathway
- VT after repair of TOF
- ARVC
AC and idiopathic VT have different prognosis and treatment
- AC and idiopathic RVOT VT can both manifest on exercise
- In AC there is a link between exercise intensity and disease development and arrhythmic risk
- Echocardiogram can be normal in subtle AC
- Look at rest ECG carefully - ECG abnormalities can precede overt structural manifestations
- Look at VT morphology
  - CMR
  - Voltage mapping
    - most effective for early stage ARVC
  - Signal averaged ECG
  - BNP
- Keep your patients under f/u

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Useful characteristics to assist differentiating idiopathic from scar-related monomorphic RV VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristic</td>
<td>Idiopathic RV VT</td>
</tr>
<tr>
<td>Any sinus rhythm ECG abnormality</td>
<td>Rare</td>
</tr>
<tr>
<td>VT QRS morphology</td>
<td></td>
</tr>
<tr>
<td>QRS transition after V4</td>
<td>Rare</td>
</tr>
<tr>
<td>Notched down stroke in V1/2</td>
<td>Rare</td>
</tr>
<tr>
<td>Delay in nadir of V1</td>
<td>Rare</td>
</tr>
<tr>
<td>Multiple VT morphologies</td>
<td>Rare</td>
</tr>
<tr>
<td>Isoproterenol required for initiation at EP study</td>
<td>++</td>
</tr>
</tbody>
</table>

AC, arrhythmogenic cardiomyopathy; EP, electrophysiology; RV, right ventricular; VT, ventricular tachycardia.
Acute Management

- Vagal manoeuvres
- Adenosine iv
- Verapamil iv
- Cardioversion

Long-term management

- B blockers *effective 30-50%
- Ca channel blockers
- (Class Ic and amiodarone)

- Drug refractory, intolerant, etc then **ablation** is preferred choice
- Even after successful ablation in idiopathic VT keep the patients under a follow up.... recurrence or ARVC after all...
When to ablate?

- Symptom control
- Tachy-cardiomyopathy
- Malignant form of idiopathic VT
- Concern about crossover with subtle form of ARVC

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>B-R</td>
<td>1. In patients with frequent and symptomatic PVCs originating from the RVOT in an otherwise normal heart, catheter ablation is recommended in preference to metoprolol or propafenone.</td>
</tr>
<tr>
<td>I</td>
<td>B-NR</td>
<td>2. In patients with symptomatic VAs from the RVOT in an otherwise normal heart for whom antiarrhythmic medications are ineffective, not tolerated, or not the patient’s preference, catheter ablation is useful.</td>
</tr>
<tr>
<td>I</td>
<td>B-NR</td>
<td>3. In patients with symptomatic idiopathic sustained monomorphic VT, catheter ablation is useful.</td>
</tr>
<tr>
<td>IIa</td>
<td>B-NR</td>
<td>4. In patients with symptomatic VAs from the endocardial LVOT, including the SV, in an otherwise normal heart for whom antiarrhythmic medications are ineffective, not tolerated, or not the patient’s preference, catheter ablation can be useful.</td>
</tr>
<tr>
<td>IIa</td>
<td>B-NR</td>
<td>5. In patients with symptomatic VAs from the epicardial OT or LV summit in an otherwise normal heart for whom antiarrhythmic medications are ineffective, not tolerated, or not the patient’s preference, catheter ablation can be useful.</td>
</tr>
</tbody>
</table>
Cardiomyopathy vs tachy-cardiomyopathy

- Young
- “No” signs of structural heart disease
- >20,000 PVCs / 24h
- Monomorphic ectopy

Recommendations for catheter ablation of PVCs in patients with or without LV dysfunction

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>B-NR</td>
<td>1. In patients with cardiomyopathy suspected to be caused by frequent and predominate monomomorphic PVCs and for whom AADs are ineffective, not tolerated, or not preferred for long-term therapy, catheter ablation is recommended.</td>
</tr>
<tr>
<td>IIa</td>
<td>B-NR</td>
<td>2. In patients with SHD in whom frequent PVCs are suspected to be contributing to a cardiomyopathy and for whom AADs are ineffective, not tolerated, or not preferred for long-term therapy, catheter ablation can be useful.</td>
</tr>
<tr>
<td>IIa</td>
<td>B-NR</td>
<td>3. In patients with focally triggered VF refractory to AADs and triggered by a similar PVC, catheter ablation can be useful.</td>
</tr>
<tr>
<td>IIa</td>
<td>C-LD</td>
<td>4. In nonresponders to cardiac resynchronization therapy (CRT) with very frequent unifocal PVCs limiting optimal biventricular pacing despite pharmacological therapy, catheter ablation can be useful.</td>
</tr>
</tbody>
</table>
Malignant variant of idiopathic VT - Short-coupled variant of RVOT-VT

Abstract
Ventricular extrasystole originating from the right ventricular outflow tract or the left ventricular outflow tract are the most commonly encountered ventricular arrhythmias recorded in ostensibly healthy individuals with no evidence of heart disease. These ventricular arrhythmias have a distinctive electrocardiographic morphology. The morphology is so distinctive that it is common practice to accept the diagnosis of "idiopathic benign ventricular arrhythmias from the outflow tract" based on this unique morphology when the electrocardiogram during sinus rhythm and the echocardiogram are normal, sometimes removing the need to perform invasive tests in patients. Even if the outflow ventricular extrasystole ultimately triggers sustained ventricular arrhythmia, the resulting ventricular tachycardia (VT) will be a monomorphic VT originating from the outflow tract, which is known to be hemodynamically well tolerated. Thus, idiopathic ventricular arrhythmias originating from outflow tracts are universally considered benign. In 2005, we described a rare form of malignant polymorphic VT resulting in syncope or cardiac arrest. Here, we review the literature on this topic since the emergence of initial descriptions of this intriguing phenomenon.

Are they just missed ARVC cases?

History of syncope
Very fast VT (>230 beats/min)
Very frequent ectopy (>20,000 VPBs/day)
VPBs with a short coupling interval...
Identification of patients at risk

<table>
<thead>
<tr>
<th>Age at presentation (year)</th>
<th>Polymorphic VT</th>
<th>Monomorphic VT</th>
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<tr>
<td>Noda et al. 10)</td>
<td>39±10</td>
<td>43±14</td>
<td>NS</td>
</tr>
<tr>
<td>Igarashi et al. 18)</td>
<td>43±14</td>
<td>51±15</td>
<td>NS</td>
</tr>
<tr>
<td>Kurosaki et al. 16)</td>
<td>45±11</td>
<td>47±14</td>
<td>NS</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Female (%)</th>
<th></th>
<th></th>
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<tbody>
<tr>
<td>Noda et al. 10)</td>
<td>56</td>
<td>71</td>
<td>NS</td>
</tr>
<tr>
<td>Igarashi et al. 18)</td>
<td>61</td>
<td>43</td>
<td>NS</td>
</tr>
<tr>
<td>Kurosaki et al. 16)</td>
<td>86</td>
<td>65</td>
<td>NS</td>
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</table>

<table>
<thead>
<tr>
<th>Familial sudden death (%)</th>
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<tbody>
<tr>
<td>Noda et al. 10)</td>
<td>6</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Igarashi et al. 18)</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Kurosaki et al. 16)</td>
<td>0</td>
<td>1</td>
<td>NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Duration of symptoms (month)</th>
<th></th>
<th></th>
<th></th>
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<tbody>
<tr>
<td>Noda et al. 10)</td>
<td>80±103</td>
<td>69±79</td>
<td>NS</td>
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<tr>
<td>Kurosaki et al. 16)</td>
<td>120±118</td>
<td>81±105</td>
<td>NS</td>
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<table>
<thead>
<tr>
<th>History of syncope (%)</th>
<th></th>
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<th></th>
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</thead>
<tbody>
<tr>
<td>Noda et al. 10)</td>
<td>69</td>
<td>18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Igarashi et al. 18)</td>
<td>61</td>
<td>14</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Kurosaki et al. 16)</td>
<td>57</td>
<td>10</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

On the other hand, both benign vagal syncope and benign idiopathic RVOT arrhythmias are common...

An otherwise benign episode of spontaneous vagal syncope can unpredictably trigger polymorphic RVOT VT in a prone patient...

In patients with VA conduction triggering of baroreceptor reflex activity can cause syncope...

[Graph showing RVOT polymorphic VT during an event of spontaneous vagal syncope triggered by venipuncture]
Prematurity Index
coupling interval of the first VT beat or isolated extrasystoles
/ preceding R-R interval during sinus rhythm

QT index
coupling of the first VT beat or isolated premature ventricular contraction / QT interval of the preceding sinus complex
For the **asymptomatic** patient, documentation of short-coupled extrasystoles probably calls for an aggressive approach with radiofrequency ablation (**duly admitting** that there is no data about the natural history of short-coupled extrasystoles).

Successful RVOT ectopics ablation after malignant episode … ICD still needed?

Of note, the five-year recurrence of malignant arrhythmias following successful ablation of **idiopathic VF** is as high as **38%**.
Preparing for ablation
ECG morphology and algorithms

Sites of successful ablation ≠ VT origin
- 40% RVOT
- 45% Aortic cusps
- 15% LVOT, PA, epicardium
Transition ≤ V2 LVOT
Transition ≥ V4 RVOT
Transition V3 either.... compare with SR
Septal - narrower QRS, synchronous ventricular activation
RVOT- Late transition ≥ V4 - Late transition + R waves notching in inferior leads = free wall superior RVOT
Lead V3 PVC/VT R/S transition

PVC/VT R/S transition later than SR?

Yes

RVOT

No

Measure V2 Transition Ratio

< 0.6

≥ 0.6

RVOT

LVOT

V2 Transition Ratio formula

\[ \frac{A}{(A+B)_{VT}} \div \frac{C}{(C+D)_{SR}} \]
If in the future noninvasive mapping could be achieved with MRI and if the technology was compatible with invasive mapping systems so that catheter positioning and noninvasive maps can be merged this would represent a new dimension of mapping technology and ablation strategy of arrhythmias.
VIVO for pre-procedure arrhythmia localization

ECG localization correction from restrictions of anatomical and body habitus variations

A DICOM image, CT or MRI, is segmented to create patient specific cardiac and torso models.

Select the arrhythmic beat of interest

3D photograph to identify the exact 12 lead ECG placement

A pre-procedure planning tool for patients with structurally normal hearts undergoing ablation treatment for idiopathic ventricular arrhythmias. In clinical trials, VIVO was shown to be 100% accurate in identifying a PVC or VT foci in the right, left, or septal region of the heart.
Pre-procedure CT or CMR to merge with EAM 3D map
Workflow

Stop ADDs 5 half life times
Isoprenaline if needed (might work at wash out)
Avoid sedation
Catheters – quad in RV – quad in CS – small curve ablation catheter
His catheter marks the commissure of NCC & RCC and in LAO marks the septum
CS catheter tip far around marks LCC and its body marks Lcx
Early on His catheter? Early on distal CS catheter?
Look at ECG - Decide if you start R or L (transaortic or transeptal)
Activation mapping
Pacemapping
Good spot
-  Bipolar≥ 30msec
-  Unipolar QS timing with bipolar
-  Good pacemap
-  Good contact (tact- sense, impedance drop, unipolar ST elevation (injury current like PPM implantation), ICE, contact force)
-  Safe spot
*Before you decide to ablate you should consider mapping other places....
*You might have to ablate at a distant spot to get the arrhythmia ... magic 13mm
Transaortic vs transeptal approach

- Presumed site of VT origin
- Aortic valve disease
- Peripheral arterial disease
- Transeptal limitations
- Operator experience
Catheters in the heart.....

Then wait.....
• Wait for spontaneous tachycardia or ectopy
• Isoprenaline infusion
• Pacing
• Atropine

• Principle: earliest spot is the origin of the tachycardia/ectopy
Mapping of RVOT

Figure 1: Representation of the Right Ventricular Outflow Tract Free Wall and Septal Right Ventricular Outflow Tract

The right ventricular outflow tract (RVOT) is divided into nine sites. Sites 1, 4, and 7 are the posterior sites, and 3, 6, and 9 are the anterior sites. The right side shows 12-lead electrocardiogram pace maps from sites 1-3 along the RVOT free wall (top), right and septal RVOT (bottom, right). PA = posterior anterior; PV = pulmonic valve; RAO = right anterior oblique; RVOT = right ventricular outflow tract; TV = tricuspid valve.
Good spot

- Early bipolar
- QS unipolar timing with bipolar
- ST elevation
Late site
Early Site
Pace Mapping
Septal RVOT VT
St Luke's Clinic Thessaloniki
Ablation & termination

[Graph showing various electrical activity waves labeled I, II, III, aVR, aVL, aVF, V1, V2, V3, V4, V5, V6, ABL, and Stim 1. The graph indicates a time period of 30000 msec.]
Ablation with acceleration
Pulmonary artery PVC

Para-Hisian PVC - cryoablation catheter in the right aortic sinus
45% aortic cusps!
Let’s map the aorta!
- Check for peripheral arterial disease
- Check AoV
- How to cross the AoV
- Where to loop your ablation catheter

- Retrograde dissection – ok ...
- Antegrade dissection ....
V1 morphology with pace map from aortic cusps
RCC – QS or Rs
LCC – M or W
NCC – atrial or parahisian capture
• Coronary angiogram
• Aortogram
• ICE
• TOE
• Contrast through ablation catheter
• Diagnostic catheter in ostium for protection
• Continuous injections
• 5-8 mm
• Cryoaablation
• 3D mapping
• Low watts
• Ablate under the valve when at the commissure
Injecting contrast through an irrigated ablation catheter

*You can’t see the whole course of the artery - mainly locate the os
The left ventricular summit (LVS) is a triangular region of the epicardial left ventricle. The apex of the triangle is formed by the bifurcation of the left anterior descending and left circumflex (LCx) arteries, while the base is formed by an arc connecting the first septal perforator branch with the LCx (white dotted line). The great cardiac vein bisects the LVS, separating it into two regions (blue and yellow dotted lines). The proximal portion (blue dotted lines) is less accessible for ablation due to the proximity of the coronary arteries as well as the presence of a thicker layer of epicardial fat. The right side depicts 12-lead ECG of typical morphology of ventricular ectopic beat originating from the LVS. Adapted from: Santangeli et al. 2015. Used with permission from Wolters Kluwer Health.
Alternative treatment

- Ablation via the GCV
- Epicardial ablation via subxiphoid access
- Alcohol ablation via Lxc branch
- Surgical cryoablation
- Simultaneous unipolar ablation
- Bipolar ablation
- Other...
Branch of the circumflex artery potentially suitable for transcoronary ethanol ablation for VTs arising from the AMC.

Ventricular tachycardia arising from the aortomitral continuity in structural heart disease: Characteristics and therapeutical considerations for an anatomically challenging area of origin

Steven et al.
Circulation
Focal VT and PVCs originating from the Purkinje system - Papillary muscles - Mitral valve
Mitral valve VAs

- Rare
- Benign
- Mitral valve is a posterior structure - Positive concordance in precordial leads
A. Left Ventricular VA with Inferior Axis (n = 25)

1. QRS ≤ 130 msec
   - Yes (n = 3)
     3 Fascicular VA (100%)
   - No (n = 22)
     Positive Precordial Concordance
       - Yes (n = 15)
         13 Mitral Annular VA (87%)
         2 Papillary VA (29%)
       - No (n = 7)
         5 Papillary VA (71%)
         2 Mitral Annular VA (17%)
Posterior MV VT
St Luke's Clinic Thessaloniki
- Posteromedial – RBBB, Superior axis, precordial transition < V4 (more frequent)
- Anterolateral – RBBB, Inferior axis, precordial transition > V4
* Posterior vs anterior is an artificial distinction as it is a confluence of muscles
- Potential risk for LBBB or AV block during ablation.
- For VPCs from the PM, real-time imaging with intracardiac echo is very useful.
- The contact and stability of the catheter on the PM can be challenging, and sometimes multiple RF lesions are required to eliminate the VPCs.
Papillary Muscle Ventricular Tachycardia Or Ectopy: Diagnostics, Catheter Ablation And The Role Of Intracardiac Echocardiography
Josef Kautzner et al.
Arrhythmia & Electrophysiology Review 2019
Papillary Muscle Ventricular Tachycardia Or Ectopy: Diagnostics, Catheter Ablation And The Role Of Intracardiac Echocardiography
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Figure 3: Site of Successful Ablation at the Ectopic Focus at the Tip of the Lateral Head of the Anterolateral Papillary Muscle

The right panel shows the corresponding electrogram at the site.

Figure 4: Recording During Delivery of Radiofrequency Current at the Site on the Anterolateral Papillary Muscle

Note the brief acceleration of ectopy and immediate disappearance during the application of the radiofrequency current (radiofrequency on marks the beginning of delivery).
Idiopathic Ventricular Fibrillation

IVF accounts for less than 10% of all VF episodes but plays a major role in the context of unexplained cardiac arrest in otherwise healthy individuals.

A resuscitated cardiac arrest victim, preferably with documentation of VF, in whom known cardiac, respiratory, metabolic, and toxicological etiologies have been excluded through clinical evaluation. To help differentiate this arrhythmogenic disorder from pause-dependent polymorphic VT/VF or torsades de pointes, some authors have put forth the term short-coupled IVF (SCIVF).

<table>
<thead>
<tr>
<th>Gene</th>
<th>Locus</th>
<th>Mutation</th>
<th>Affected Ion Channel or Receptor</th>
<th>Electrophysiological Feature</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>DPP6</td>
<td>7q36</td>
<td>c.1-340C&gt;T</td>
<td>$I_{to,f}$</td>
<td>Gain of function</td>
<td>19-21</td>
</tr>
<tr>
<td></td>
<td>7q36</td>
<td>pH332R (DPP6-T)</td>
<td>$I_{to,f}$</td>
<td>Gain of function</td>
<td>22</td>
</tr>
<tr>
<td>CALM1</td>
<td>14q32.11</td>
<td>c.268T&gt;C: p.F90L</td>
<td>Calmodulin 1</td>
<td>Unknown</td>
<td>28</td>
</tr>
<tr>
<td>RYR2</td>
<td>1q43</td>
<td>c.6224T&gt;C: p.Ile2075Thr, exon 41, c.13781A&gt;G: p.Lys4594Arg, exon 94</td>
<td>Ryanodine receptor 2</td>
<td>Unknown</td>
<td>29</td>
</tr>
</tbody>
</table>

Genetic mutations in familial idiopathic ventricular fibrillation

(A) Various 12-lead electrocardiogram morphologies of PVCs originating in the left Purkinje system in the same patient. (B) Holter results showing a self-terminating episode of VF triggered by short-coupled PVC and demonstrating the R-on-T phenomenon.
### Treatment of idiopathic ventricular fibrillation

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class</th>
<th>Level</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICD implantation is recommended in survivors of idiopathic VF.</td>
<td>I</td>
<td>B</td>
<td>154, 583</td>
</tr>
<tr>
<td>Catheter ablation of PVCs triggering recurrent VF leading to ICD interventions is recommended when performed by experienced operators.</td>
<td>I</td>
<td>B</td>
<td></td>
</tr>
<tr>
<td>Catheter ablation of PVCs leading to electrical storm is recommended when performed by experienced operators.</td>
<td>I</td>
<td>B</td>
<td></td>
</tr>
</tbody>
</table>

2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death.
Short-coupled TdP

- Rare variant of TdP
- Unknown aetiology
- Extremely short-coupled interval of the first PVC (300 ms) initiating the tachycardia
- Young patients presenting with unclear syncope and a positive family history for SCD.
- In most cases, TdP deteriorates into VF
- There may be a link to an autonomic nervous system imbalance
- ICD
- Verapamil iv for storm
- Ablation

### Treatment of short-coupled torsade de pointes

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>Class (^a)</th>
<th>Level (^b)</th>
<th>Ref. (^c)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICD is recommended in patients with conclusive diagnosis of short-coupled TdP.</td>
<td>I</td>
<td>B</td>
<td>589</td>
</tr>
<tr>
<td>Intravenous verapamil to acutely suppress/prevent an electrical storm or recurrent ICD discharges should be considered.</td>
<td>IIa</td>
<td>B</td>
<td>590, 591</td>
</tr>
<tr>
<td>Catheter ablation for long-term suppression/prevention of an electrical storm or recurrent ICD discharges should be considered.</td>
<td>IIa</td>
<td>B</td>
<td>586</td>
</tr>
</tbody>
</table>
Fascicular Tachycardia

- Different pathophysiology - mechanism to outflow tract VT (*reentry)
- Left ventricular origin
- Structurally normal hearts
- 15 to 40 year olds (unusual after 55)
- Male 60%
- More occur at rest
- Usually paroxysmal
- Can rarely cause TCM
- 10% of idiopathic/normal heart VT (or 1%)
- Benign course-prognosis excellent
- Verapamil sensitive
- Can be induced also from atrium
- RF ablation 90% cure
Management of fascicular tachycardia

• Acute
  – Verapamil infusion
  – Cardioversion

• Medical management
  – Verapamil orally
  – B-blockers, class I & III AADs

• Ablation
  – Very successful

Recommendations for catheter ablation of bundle branch reentrant VT and for catheter ablation of fascicular VT

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>B-NR</td>
<td>1. In patients with bundle branch reentrant VT, catheter ablation is useful for reducing the risk of recurrent VT.</td>
</tr>
<tr>
<td>I</td>
<td>B-NR</td>
<td>2. In patients with idiopathic left fascicular reentrant VT for whom medications are ineffective, not tolerated, or not the patient’s preference, catheter ablation is useful.</td>
</tr>
<tr>
<td>I</td>
<td>B-NR</td>
<td>3. In larger pediatric patients (≥15 kg) with idiopathic left fascicular reentrant VT in whom medical treatment is ineffective or not tolerated, catheter ablation is useful.</td>
</tr>
<tr>
<td>I</td>
<td>B-NR</td>
<td>4. In patients with focal fascicular VT with or without SHD, catheter ablation is useful.</td>
</tr>
<tr>
<td>I</td>
<td>B-NR</td>
<td>5. In patients with postinfarction reentrant Purkinje fiber-mediated VT, catheter ablation is useful.</td>
</tr>
</tbody>
</table>
Fascicular Tachycardia (Belhassen's VT)

(A) In left posterior fascicle (LPF)-VT, activation propagates antegrade in the midseptum where abnormal Purkinje tissue distributes and conducts retrogradely near the LPF. In the past, LPF was considered as part of the circuit, but recent reports suggest the LPF is a bystander. (B) In left anterior fascicle (LAF)-VT, activation propagates antegrade in the midseptum and conducts retrogradely near the LAF. (C) In USVT, activation propagates antegrade in the LPF or LAF and then conducts retrogradely in the midseptum.
Twelve-lead electrocardiogram recorded during idiopathic left ventricular tachycardia. The QRS morphology is right bundle superior, although the right bundle pattern does not match that seen in right bundle branch block (monophasic R wave). Nonetheless, the QRS complexes are relatively narrow and normal in appearance, which often leads to confusion for SVT with aberrancy.
**Ablation during tachycardia**
- Ablation at Purkinje potential site
- Ablation at pre-Purkinje potential site

**Ablation during sinus rhythm**

*Mechanical block during mapping (60%)*

*Non inducible before mapping (15-30%)*

Target site during sinus rhythm:
“earliest late potential“- earliest retrograde Purkinje Potential

- Pace mapping
- Electro anatomic mapping

*ablate apical to avoid LBBB or CHB*
Complications

Leg - use echo for access

RVOT perforation (very thin) - don’t push perpendicular – look at the shape of your catheter at fluro- contact force

His - RB - LB injury - mark them - use cryo - ablate from RCC

Stroke - heparine

AoV - ICE - loop your catheter - go transeptal if valve is not good

CS dissection (select roght tools, be gentle)

Aortic dissection (select patients, loop your catheter low)

Coronary arteries injury (avoid extensive septal ablation, coronary angiogram > 5-8mm, CS marks Lcx, ICE, 3D mapping)
Conclusions

- Catheter ablation is a safe and effective treatment option for outflow tract ventricular arrhythmias.
- The 12-lead ECG is critical in accurately predicting the site of origin of OT VAs.
- Ablation success rates are highest when VA is spontaneously seen or reproducibly induced at the time of procedure, which allows for both activation and pace mapping.
- Ablation of OT VAs can be sometimes challenging due to anatomic constraints. Different strategies can be utilised to overcome these difficulties.
- Remember you are chasing an “innocent” arrhythmia, in often very young people, with a normal hearts. You might consider to try another day, better prepared.