Ventricular Tachycardia and Premature Ventricular Contractions

Gadi Silberman, MD
Epidemiology VT and SCD:

- Cardiovascular disease → leading cause of death in U.S.
- > 350,000 deaths/year
- Most SCD results from ventricular VT/VF
- In CICU: 8% have episodes of nonsustained VT, 2% develop sustained VT, and 5% are treated for VF
- Mortality in ischemic heart disease → 22% from SCD
- Nonischemic conditions associated with VT, VF, and SCD: genetic conditions, cardiomyopathies, and idiopathic VT in structurally normal hearts
Ventricular Arrhythmia Classification

**Life threatening**
- Abnormal structure heart
  - ICM, NICM
  - Sig valve disease
  - HCM, ARVCM, non compaction
  - Congenital heart disease
- Normal structure heart
  - Genetic syndromes (e.g. LQT, SCT, Brugada, CPVT)
  - Idiopathic VF

**Non Life threatening →** ~10% (20% in Japan)
- Normal structure heart
  - Outflow tract VT’s (RVOT, LVOT, aortic valve, pulmonic valve, peri His, epi)
  - LV VT fascicular form
    - Left posterior, left anterior, high septal
  - Other
    - MV or TV annulus
    - Papillary VT
    - Perivascular Epicardial

**Mono**

**Poly/VF**

**Mono/PolyVF**

**Poly/VF Bi-directional**

Modified from Prystowsky et al JACC 2012
ICD Shocks

- 30% of primary prevention patients receive a shock for sustained VT within 3 years of implant (Poole et al. NEJM 2008 and Moss et al. Circ 2004)
- 45% of secondary prevention patients receive a shock (AVID, NEJM 1997)
- Electrical storm:
  - Primary prevention → ~ 4% (MADIT-II substudy)
  - Secondary prevention → ~ 20% (AVID)
- Decrease QOL (AVID, SCD-HeFT)
- Increase in mortality (appropriate or inappropriate)

**Figure 1. Hazard Ratios for the Association of ICD Shock with the Risk of Death, According to Shock Type.**
Panel A shows the hazard ratios for the association of shock types with the risk of death, adjusted for baseline prognostic factors identified in the trial (age, sex, cause of heart failure, New York Heart Association class, time since the diagnosis of heart failure, left ventricular ejection fraction, distance covered on a 6-minute walk, systolic blood pressure, presence or absence of diabetes, use or nonuse of angiotensin-converting-enzyme inhibitors, use or nonuse of digoxin, presence or absence of mitral regurgitation, renal sufficiency or insufficiency, presence or absence of a history of substance abuse, baseline electrocardiographic intervals, and score on the Duke Activity Status Index²). Panel B shows the adjusted hazard ratios for the risk of death according to the number of appropriate or inappropriate shocks. App denotes appropriate defibrillator shock, CI confidence interval, and Inapp inappropriate defibrillator shock.
Complications of ICD/CRT implant

<table>
<thead>
<tr>
<th>Complication</th>
<th>ICD (n = 23,110)</th>
<th>CRT-D (n = 7,874)</th>
<th>p Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any complication†</td>
<td>11.0%</td>
<td>10.5%</td>
<td>NS</td>
</tr>
<tr>
<td>Mechanical complication of ICD system</td>
<td>4.8%</td>
<td>3.8%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>With lead or pocket revision</td>
<td>1.2%</td>
<td>1.8%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hematoma/hemorrhage</td>
<td>2.5%</td>
<td>3.4%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Infection associated with implant</td>
<td>1.4%</td>
<td>0.7%</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>1.0%</td>
<td>1.2%</td>
<td>NS</td>
</tr>
<tr>
<td>Death</td>
<td>0.9%</td>
<td>1.1%</td>
<td>0.07</td>
</tr>
<tr>
<td>Other cardiac complications</td>
<td>0.8%</td>
<td>0.7%</td>
<td>NS</td>
</tr>
<tr>
<td>Pericardial effusion/tamponade</td>
<td>0.3%</td>
<td>0.3%</td>
<td>NS</td>
</tr>
<tr>
<td>Acute renal failure with new hemodialysis</td>
<td>0.3%</td>
<td>0.3%</td>
<td>NS</td>
</tr>
</tbody>
</table>

Reynolds et al JACC 2006
• Wide complex tachycardia ≠ VT
  – WPW
  – SVT with aberrant conduction
  – Pacemaker mediated rhythms

• Although rare, not all VT is wide complex
  – Some forms of fascicular VT
Classification by Clinical Presentation

• **Hemodynamically stable**
  – Palpitations, fatigue, dyspnea ...

• **Hemodynamically unstable**
  – Profound pre-syncope
  – Syncope
  – Aborted sudden cardiac arrest (< 1 hr of symptom)
  – Sudden cardiac death (< 1 hr of symptom)
# Initial Assessment

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Assessment</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital signs</td>
<td>Hemodynamic stability</td>
<td>If hemodynamically unstable, treat with urgent DCCV or defibrillation</td>
</tr>
<tr>
<td></td>
<td>Tachycardia diagnosis</td>
<td>Differentiate VT from SVT with aberrancy; determine VT exit site</td>
</tr>
<tr>
<td>History</td>
<td>Symptoms (e.g., chest pain indicating ongoing ischemia)</td>
<td>Identify cause and triggers</td>
</tr>
<tr>
<td>Antiarrhythmics, digoxin, QTc-prolonging medications</td>
<td>Identify pharmacologic contribution to a proarrhythmic state</td>
<td></td>
</tr>
<tr>
<td>Family history</td>
<td>Family history of SCD</td>
<td>Determine risk of inherited predisposition to SCD</td>
</tr>
<tr>
<td>Physical examination</td>
<td>Canon A waves, Murmurs, sternotomy scar</td>
<td>Indicate AV dissociation</td>
</tr>
<tr>
<td>Laboratory tests</td>
<td>Electrolytes, creatinine, troponin, thyroid-stimulating hormone, toxicology assays</td>
<td>Indicate existing structural heart disease</td>
</tr>
<tr>
<td>Imaging</td>
<td>Chest roentgenography, echocardiography, Coronary angiography, Computed tomography, magnetic resonance imaging</td>
<td>Indicated in all patients with VT to assess for structural heart disease; Indicated if VT occurs secondary to ischemia; Indicated in special cases when particular cardiomyopathies are suspected</td>
</tr>
</tbody>
</table>

Schleifer and Sriwathsan, Cardiol Clin 31 (2013) 595–605
32 year old with recurrent runs of tachycardia and recent syncope. What antiarrhythmic would you choose?

Tracing obtained from Schleifer et al. Cardiol Clin 2013
Antiarrhythmic selection in wide complex tachycardia:

• Beware of long QT
  – Acquired
    • Drugs, ischemia
  – inherited

• Amiodarone no longer appropriate in WPW related wide complex arrhythmias (Class III)
<table>
<thead>
<tr>
<th>Antiarrhythmic</th>
<th>Dosing</th>
<th>Acute Adverse Reactions</th>
</tr>
</thead>
</table>
| Procainamide  | Load: 17 mg/kg  
                Maximum rate: 50 mg/min  
                Maintenance: 1–4 mg/min | Hypotension  
                Hold if QRS prolongs >50% |
| Lidocaine     | Load: 1–3 mg/kg  
                Rate: 20–50 mg/min  
                Maintenance: 1–4 mg/min | Reduce dose in heart failure  
                Monitor for neurotoxicity: delirium, seizures, or paresthesias |
| Amiodarone    | Load: 150 mg over 10 min if blood pressure is normal:  
                Maintenance: 1 mg/min for 6 h, then 0.5 mg/min for 18 h | Caution in cardiogenic shock  
                TdP is rare  
                Use with pacing if patient is severely bradycardic |
Ventricular arrhythmia classification:

- Pleomorphic VT
  - Normal EF
  - VDP-induced Cardiomyopathy
  - Purkinje mediated BBR-VT
  - Inherited Arrhythmias
    - CPVT
    - LQTS, SQTS
    - Brugada Syndrome

- Polymorphic VT / VF
  - VPD-induced VF
    - RVOT
    - Purkinje
    - Moderator band

- Monomorphic VT
  - Secondary
  - Idiopathic
    - RVOT/LVOT
    - TA/MA
    - Fascicular
    - Papillary muscle
    - Epicardial origin

- Ventricular Premature Depolarizations

Pleomorphic VT

Slow Sinusoidal VT
- Hyperkalemia
- Drug toxicity
- End stage heart disease

Karitsis et al. JACC 2012
Stevenson WG. Heart Rhythm 2014
<table>
<thead>
<tr>
<th>Classification by Electrocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nonsustained VT</strong></td>
</tr>
<tr>
<td>Three or more beats in duration, terminating spontaneously in less than 30 s.</td>
</tr>
<tr>
<td>VT is a cardiac arrhythmia of three or more consecutive complexes in duration emanating from the ventricles at a rate of greater than 100 bpm (cycle length less than 600 ms)</td>
</tr>
<tr>
<td><strong>Monomorphic</strong></td>
</tr>
<tr>
<td>Nonsustained VT with a single QRS morphology.</td>
</tr>
<tr>
<td><strong>Polymorphic</strong></td>
</tr>
<tr>
<td>Nonsustained VT with a changing QRS morphology at cycle length between 600 and 180 ms.</td>
</tr>
<tr>
<td><strong>Sustained VT</strong></td>
</tr>
<tr>
<td>VT greater than 30 s in duration and/or requiring termination due to hemodynamic compromise in less than 30 s.</td>
</tr>
<tr>
<td><strong>Monomorphic</strong></td>
</tr>
<tr>
<td>Sustained VT with a stable single QRS morphology.</td>
</tr>
<tr>
<td><strong>Polymorphic</strong></td>
</tr>
<tr>
<td>Sustained VT with a changing or multiform QRS morphology at cycle length between 600 and 180 ms.</td>
</tr>
<tr>
<td><strong>Bundle-branch re-entrant tachycardia</strong></td>
</tr>
<tr>
<td>VT due to re-entry involving the His-Purkinje system, usually with LBBB morphology; this usually occurs in the setting of cardiomyopathy.</td>
</tr>
<tr>
<td><strong>Bidirectional VT</strong></td>
</tr>
<tr>
<td>VT with a beat-to-beat alternans in the QRS frontal plane axis, often associated with digitalis toxicity.</td>
</tr>
<tr>
<td><strong>Torsades de pointes</strong></td>
</tr>
<tr>
<td>Characterized by VT associated with a long QT or QTc, and electrocardiographically characterized by twisting of the peaks of the QRS complexes around the isoelectric line during the arrhythmia:</td>
</tr>
<tr>
<td>- “Typical,” initiated following “short-long-short” coupling intervals.</td>
</tr>
<tr>
<td>- Short coupled variant initiated by normal-short coupling.</td>
</tr>
<tr>
<td><strong>Ventricular flutter</strong></td>
</tr>
<tr>
<td>A regular (cycle length variability 30 ms or less) ventricular arrhythmia approximately 300 bpm (cycle length—200 ms) with a monomorphic appearance; no isoelectric interval between successive QRS complexes.</td>
</tr>
<tr>
<td><strong>Ventricular fibrillation</strong></td>
</tr>
<tr>
<td>Rapid, usually more than 300 bpm/200 ms (cycle length 180 ms or less), grossly irregular ventricular rhythm with marked variability in QRS cycle length, morphology, and amplitude.</td>
</tr>
</tbody>
</table>
Tachycardia Mechanisms

Example:

- Idioventricular rhythms, parasystole
- Scar related VT, Fascicular VT
- Long QT
- Digitalis related, Some idiopathic VT (outflow tract)
VT Classification

- Underlying substrate ➔ may not be known
- Clinical stability
- Morphology
- Mechanism
PVCs

• Are PVCs dangerous... can they cause sudden cardiac death? cardiomyopathy (CM)?
• How frequent do PVCs need to occur to cause a CM?
• What came first PVCs or cardiomyopathy?
VF triggered by PVCs

• “idiopathic VF” initiated by monomorphic PVCs in some patients
• PVCs typically originate in the Purkinje, rarely in myocardium (RVOT)
• Ablation is effective at eliminating recurrence, > 5 year followup

Stevenson WG. Heart Rhythm 2014
Knecht et al. JACC 2009
Are PVCs dangerous...can they cause cardiomyopathy?

- Ablation of PVCs can improve ventricular function
- It may take 4-45 months for ventricular function to improve

Bogun et al. Heart Rhythm 2007
Yokokawa et al. Heart Rhythm 2013
How frequent do PVCs need to occur to potentially lead to cardiomyopathy (CM)?

- Typically > 20% PVC burden is needed to cause a CM, but, can be with > 10% burden
- < 10% PVC and asymptomatic, typically no CM
- > 10% should follow as may develop CM
- Certain PVC morphologies can predict a PVC induced CM
- Assessment of EF can be challenge with frequent PVCs
  - Do not use the PVC or post PVC beat, if bigeminy, may need to average the EF (???)
  - Consider PVC suppression with meds
  - Consider cardiac MRI to look for delayed enhancement (scar)
  - Consider invasive unipolar mapping

Bogun et al. Heart Rhythm 2007
Yokokawa et al. Heart Rhythm 2013
Sadek et al. Trends in Cvs Med 2014
What came first PVCs or cardiomyopathy?

Bogun et al. Heart Rhythm 2007
Yokokawa et al. Heart Rhythm 2013
Voltage Mapping
Patient with irreversible CM
NICM, LVEF <35%

Campos et al. JACC 2012
Patient with reversible PVC induced CM, LVEF 35% at time of ablation

Campos et al. JACC 2012
LV unipolar voltage abnormality accounting for > 32% of LV endocardial surface area in the absence of myocardial scar as defined by bipolar voltage abnormality or cardiac MRI suggests irreversibility of LV dysfunction and appears to represent microscopic fibrosis.
VT in “normal hearts”
~10%
Outflow Tract Type VTs

- cAMP mediated, catecholamine sensitive
- Adenosine \(\rightarrow\) can terminate
- most common forms have LBBB/inferior morphology
- Need to exclude cardiomyopathy, esp ARVC M
- Imaging, voltage map...
Anatomical Relationships of the Outflow Tracts

Issa and Miller 2010
Pyrstowsky et al jacc 2012
Outflow Tract Type VTs

Hoffmayer et al. Curr Probl in Cardiol 2013
Pyrstowsky et al. JACC 2012
Outflow Tract Anatomy (continued)

antero-septum (left)

free-wall

postero-septum (right)

Lead I

Apex of TV

Posterior aspect of RVOT
Septal aspect of RVOT
Anterior aspect of RVOT
Upper RVOT border
Midway line
Lower RVOT border
ECG Features: RVOT v. LVOT/Aortic Cusps

TABLE 2. ECG classification of RVOT-VT versus LVOT/aortic cusp VT

<table>
<thead>
<tr>
<th>RVOT-VT</th>
<th>LVOT/Aortic Cusp VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Later precordial transition (V3 or later)</td>
<td>Earlier precordial transition (by V3)</td>
</tr>
<tr>
<td>With V3 transition:</td>
<td>With V3 transition:</td>
</tr>
<tr>
<td>VT transition later than sinus rhythm</td>
<td>VT transition earlier than sinus</td>
</tr>
<tr>
<td>V2 transition ratio &lt;0.60</td>
<td>V2 transition ratio ≥0.60</td>
</tr>
<tr>
<td>Narrower R-wave duration and greater</td>
<td>Broader R-wave duration and greater R/</td>
</tr>
<tr>
<td>R/S-wave amplitude ratio in V1 and V2</td>
<td>S-wave amplitude ratio in V1 and V2</td>
</tr>
<tr>
<td></td>
<td>Notch (qrS) in V1 or V2</td>
</tr>
</tbody>
</table>

LVOT 20-30%
ECG Features/Localization

Park et al. PACE 2012
Outflow Tract Type VTs: Management

- **Acute**
  - Avoid CCB/BB if heart “structure” unknown
  - Vagal maneuvers
  - Adenosine (6-24mg), verapamil (diltiazem), BB

- **Chronic**
  - CCB (diltiazem or verapamil), BB
  - 20-50% efficacy
  - AAD: IC (flecainide), IA, III (amiodarone)

- **Ablation** → cure rates >80%
  - Coronary artery or valve injury possible in certain locations

- **Monitoring response to therapy:**
  - Symptoms, ECG monitor, exercise test
  - NOT EPS

Issa and Miller 2010
Second coupling interval of nonsustained ventricular tachycardia to distinguish malignant from benign outflow tract ventricular tachycardias.

- Outflow tract VTs are rarely associated with malignant outcome.
- Case control study compared patients without structural heart disease who had documented OT-NSVT on ECG.
  - ECG parameters were compared between patients with syncope, aborted sudden cardiac death, or ventricular fibrillation (malignant group, n = 36) and patients without syncope (benign group, n = 40).
- Baseline characteristics were similar between groups
- Analysis of NSVT
  - First coupling interval (CI) of NSVT was comparable between the 2 groups (458 ± 87 ms vs 485 ± 95 ms, P = .212).
  - Second CI of NSVT beats was significantly shorter in the malignant group (313 ± 58 ms vs 385 ± 83 ms, P < .0001).
- 48-month follow-up
  - the benign group had a significantly lower recurrence of clinical VT than the malignant group (P = .046).
  - The malignant group frequently had more than 1 focus of VT, whereas the benign group showed only a single focus (1.82 vs 1.09, P = .023).

Kim et al. Heart Rhythm 2014
Idiopathic Left VT/Fascicular VT/Verapamil Sensitive VT

1. Left posterior fascicular VT
   i. Proximal type (mid-septum)
   ii. Distal type (apical-inferior septum)
2. Left anterior fascicular VT
   i. Proximal type (mid-septum)
   ii. Distal type (antero-lateral wall)
3. Left upper septal fascicular VT (upper septum)

RBBB/LAD (90-95%)
RBBB/RAD (5-10%)
Narrow QRS/normal axis

Issa and Miller 2010
Response of recurrent sustained ventricular tachycardia to verapamil

BERNARD BELHASSEN, HESCHI H ROTMENSCH, SHLOMO LANIADO
From the Department of Cardiology, Municipal Governmental Medical Center, Ichilov Hospital, Tel-Aviv, Israel

SUMMARY A 28-year-old man is described with no demonstrable organic heart disease and recurrent paroxysmal attacks of sustained ventricular tachycardia. Lignocaine and ajmaline failed to terminate the first attack but a bolus injection of verapamil succeeded. This drug was subsequently successful on six more occasions. During electrophysiological study of the eighth attack, slow intravenous administration of verapamil significantly reduced the rate of the tachycardia and prevented its subsequent reinitiation by pacing.

Br Heart J 1981; 46: 679–82
Idiopathic Left VT / Fascicular VT/
Verapamil Sensitive VT

• Reentry mechanism: area of slow conduction is in Purkinje fibers
• 10-15% of idiopathic VTs, most common form of idiopathic LV VTs
• Clinical presentation:
  – VT rate usually 150-200bpm, paroxysmal but may be incessant
  – Excellent prognosis
• Initial evaluation \(\rightarrow\) r/o structural heart disease
• Acute treatment \(\rightarrow\) IV verapamil (not sensitive to adenosine)
• Chronic treatment \(\rightarrow\)
  • verapamil useful in mild cases
  • Ablation: >90% acute success rate

Tsuchiya et al Circ 1999
Issa and Miller 2010
The others...are rare

20-30%

> 70%

Table II.
Classification of the Idiopathic VT According to the Site of Origin

<table>
<thead>
<tr>
<th>I. LV Origin VT</th>
<th>C. Fascicular VT</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Supraventricular</td>
<td>1. Left posterior fascicle</td>
</tr>
<tr>
<td>a. ASOV</td>
<td>2. Left anterior fascicle</td>
</tr>
<tr>
<td>(1) LCC</td>
<td>3. Left upper septal fascicle</td>
</tr>
<tr>
<td>(2) RCC</td>
<td></td>
</tr>
<tr>
<td>(3) NCC</td>
<td></td>
</tr>
<tr>
<td>(4) LCC/RCC junction</td>
<td></td>
</tr>
<tr>
<td>2. Infraventricular</td>
<td>A. RVOT</td>
</tr>
<tr>
<td>a. AMC</td>
<td>B. Parahisian</td>
</tr>
<tr>
<td>b. Septo-parahisian</td>
<td>C. TA</td>
</tr>
<tr>
<td>3. Epicardial</td>
<td>D. PA</td>
</tr>
<tr>
<td>a. AIV/CGV</td>
<td>E. VT arising from the other</td>
</tr>
<tr>
<td>B. VT arising from other LV sites</td>
<td>sites</td>
</tr>
<tr>
<td>1. MA</td>
<td>1. PPM</td>
</tr>
<tr>
<td>2. PPM</td>
<td>III. Epicardial origin VT</td>
</tr>
<tr>
<td>a. Anterolateral PPM</td>
<td></td>
</tr>
<tr>
<td>b. Posteromedial PPM</td>
<td></td>
</tr>
<tr>
<td>3. Crux</td>
<td></td>
</tr>
</tbody>
</table>

Park et al. PACE 2012
Prior to defining an outflow tract VT as benign, need to exclude structural heart disease and assess the induction coupling intervals

- Is baseline ECG normal?
- ECG 2\(^{nd}\) coupling interval short? (pap muscle VT?)
- (SAECG)
- Is there a myopathic process? → imaging
  - Cath, echo, MRI
  - MRI may miss small scar
- Voltage map
Life threatening VT’s
VT in cardiomyopathy/ structural heart disease

- ICM
- NICM/IDCM
- ARVCM
- Sarcoid
- Chagas
- Hypertrophic CM
- (Channelopathies)
## Secondary Prevention Trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>N</th>
<th>Age (yr)</th>
<th>Women (%)</th>
<th>NYHA &gt;II (%)</th>
<th>Mean Ejection Fraction</th>
<th>Mean Follow-up (mo)</th>
<th>Annual Rate in Controls</th>
<th>Relative Risk Reduction</th>
<th>Absolute Risk Reduction</th>
<th>Number Needed to Treat (36 mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVID (1999)</td>
<td>1016</td>
<td>65</td>
<td>20</td>
<td>8</td>
<td>0.35</td>
<td>18</td>
<td>12</td>
<td>31</td>
<td>8.2</td>
<td>9</td>
</tr>
<tr>
<td>CIDS (2000)</td>
<td>659</td>
<td>64</td>
<td>16</td>
<td>11</td>
<td>0.34</td>
<td>36</td>
<td>10</td>
<td>20</td>
<td>4.3</td>
<td>23</td>
</tr>
<tr>
<td>CASH (2000)</td>
<td>228</td>
<td>58</td>
<td>20</td>
<td>19</td>
<td>0.45</td>
<td>57</td>
<td>9</td>
<td>23</td>
<td>8.1</td>
<td>20</td>
</tr>
<tr>
<td>MADIT-I (1996)</td>
<td>196</td>
<td>63</td>
<td>8</td>
<td>—</td>
<td>0.26</td>
<td>27</td>
<td>17</td>
<td>54</td>
<td>22.8</td>
<td>3</td>
</tr>
<tr>
<td>MUSTT* (1999)</td>
<td>704</td>
<td>65</td>
<td>10</td>
<td>24</td>
<td>0.28</td>
<td>39</td>
<td>13</td>
<td>51</td>
<td>23</td>
<td>5</td>
</tr>
</tbody>
</table>

*Inducible at EP Study Trial

Modified from Ellenbogen et al 2011
## Primary Prevention Trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>N</th>
<th>Age (yr)</th>
<th>Women (%)</th>
<th>NYHA &gt;II (%)</th>
<th>Mean Ejection Fraction</th>
<th>Mean Follow-up (mo)</th>
<th>Annual Relative Rate in Controls</th>
<th>Relative Risk Reduction</th>
<th>Absolute Risk Reduction</th>
<th>Number Needed to Treat (36 mo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADIT-II (2002)</td>
<td>1232</td>
<td>64</td>
<td>16</td>
<td>29</td>
<td>0.23</td>
<td>20</td>
<td>10</td>
<td>31</td>
<td>5.4</td>
<td>10</td>
</tr>
<tr>
<td>AMIOVIRT (2003)</td>
<td>103</td>
<td>52</td>
<td>30</td>
<td>20</td>
<td>0.23</td>
<td>24</td>
<td>4</td>
<td>13</td>
<td>1.7</td>
<td>39</td>
</tr>
<tr>
<td>CAT (2002)</td>
<td>104</td>
<td>52</td>
<td>20</td>
<td>35</td>
<td>0.24</td>
<td>23</td>
<td>4</td>
<td>17</td>
<td>5.4</td>
<td>12</td>
</tr>
<tr>
<td>COMPANION* (2004)</td>
<td>903</td>
<td>67</td>
<td>32</td>
<td>100</td>
<td>0.22</td>
<td>15</td>
<td>19</td>
<td>36</td>
<td>7.3</td>
<td>5</td>
</tr>
<tr>
<td>SCD-HeFT* (2005)</td>
<td>1676</td>
<td>60</td>
<td>23</td>
<td>30</td>
<td>0.25</td>
<td>46</td>
<td>7</td>
<td>23</td>
<td>6.8</td>
<td>23</td>
</tr>
<tr>
<td>DEFINITE (2005)</td>
<td>458</td>
<td>58</td>
<td>29</td>
<td>21</td>
<td>0.21</td>
<td>29</td>
<td>7</td>
<td>35</td>
<td>5.2</td>
<td>24</td>
</tr>
</tbody>
</table>

Modified from Ellenbogen et al 2011
ICD Shocks

- 30% of primary prevention patients receive a shock for sustained VT within 3 years of implant (Poole et al. NEJM 2008 and Moss et al. Circ 2004)
- 45% of secondary prevention patients receive a shock (AVID, NEJM 1997)
- Electrical storm:
  - Primary prevention → ~ 4% (MADIT-II substudy)
  - Secondary prevention → ~ 20% (AVID)
- Decrease QOL (AVID, SCD-HeFT)
- Increase in mortality (appropriate or inappropriate)

Poole et al. NEJM 2008
Antiarrhythmic Drugs Therapy for Ventricular Arrhythmias

**Beta-blockers**
- Safe
- Limited efficacy

**Sotalol**
- More effective than other Beta-BIs
- QT prolongation – polymorphic VT
- Renal excretion

**Others**
- Dofetilide – poor efficacy, not approved for VT
- Mexiletine – limited efficacy, GI and neuro side effects
- Quinidine – limited efficacy, QT, GI side effects
- Flecainide – proarrhythmia, negative inotropic effects

**Amiodarone**
- Most effective
- Long-term toxicities (20% of pts)
- Bradyarrhythmias

**Combinations**
- Amio + Mex
- Quinidine + Mex
- Others
Medical therapy vs. VT ablation

- Ablation vs. medical therapy
  - OR of VT recurrence following ablation v. meds (pooled results)
    0.37 CI 0.24-0.57 p < 0.05

- VT recurrence following a more traditional VT ablation approach is common 32-37% (up to ~ 50%)
  - regardless of timing i.e. primary or secondary prevention

- Studies to date used mostly an activation/entrainment mapping approach or limited substrate ablation approach

- Complication rate: 5-7%

Santegeli et al Card Electophy Clin 2011
VT ablation in structural heart disease

- Ischemic heart disease is best studied
- Reduction or elimination of VT in about 70%
- 25-50% have a recurrence
- Mortality ~3%
- Endpoints:
  - Ablation of clinical VT
  - Ablation of all inducible VTs by PES
  - Substrate mapping/ablation targets
Challenges in VT ablation
Traditional entrainment mapping guided VT ablation

• Limitations:
  – Hemodynamic instability
  – Inducibility of non-clinical VT
  – Recurrence rates
Progression of disease (NICM)

Increase in UNI scar area: +38 cm²

Liuba and Marchilinski Circ J. 2013
NICM

% Abnormal electrograms

VT induced

No VT induced

Epi

Endo

P=NS

P=NS

Liuba and Marchilinski Circ J. 2013
Epicardial/intramyocardial substrate

- NICM > ICM
- Particular issue for patients with ARVCM
- Identification pre-procedure
- ECG
- Imaging: MRI, CT
- Identification at EP study
- Voltage mapping:
  - Unipolar EGMs (LV < 8.3mV RV < 5.5mV)
  - Bipolar epi EGMs (<1.0 with associated morph features)
- ICE
Substrate Mapping

- **Voltage**
  - Bipolar
  - Unipolar
  - Unexcitable tissue

- **Local signals**
  - Fractionated, multicomponent, high frequency, low amplitude
  - Late in sinus rhythm
  - Early in VT
  - Pacing maneuvers may be required to confirm
NICM

ENDO Bip

ENDO Uni

EPI Bip

Liuba and Marchilinski Circ J. 2013
ARVCM

ENDO Bip

ENDO Uni

EPI Bip

Liuba and Marchilinski Circ J. 2013
Local abnormal ventricular activities (LAVAs)
• Voltage alone is not enough to define substrate due to pericardial fat
• Need to look at split signals, late signals, fractionation and timing

Liuba and Marchilinski Circ J. 2013
LAVA guided ablation

Jaïs et al. Circ 2012
LAVA guided ablation

Jaïs et al. Circ 2012
Mapping and ablation of scar based VT mitral annulus (NICM)
Limited substrate ablation

Extensive substrate ablation ("homogenization" of scar)

Santegeli et al Card Electrophys Clin 2011
Modified from Natale 2013
Endo – Epi Homogenization
Bundle Branch Reentry
Complications of VT/PVC ablation

- Perforation /tamponade
- Depending on location $\rightarrow$ AV block
- Coronary injury
- Valve injury
- Thromboembolism
Clinical Trials

- STAR-VT: No prior shock, late potential ablation
- PARTITA: Prophylactic
- Rescue VT
- Prevent VT: Ganglia
- VTACH II: Ablation instead of ICD
- VTACH
- VANISH
- STRATUM
- SMASH
- THERMOCOOL (Not Randomized)

Legend:
- Planned or ongoing
- Trials with large, non-cardiac ablation components
- Complete
- Proposed trial
- Size of bubble roughly reflects study size

Sadek and Marchlinskini. Trends in Cvs Med 2014
Adjunct Techniques

- Epicardial access and ablation
- Mechanical circulatory support
- Cardiac sympathetic denervation
Epicardial Access

Lim et al. Heart Rhythm 2014
Epicardial access and ablation safety considerations

Lim et al. Heart Rhythm 2014
Epicardial access and ablation safety considerations

<table>
<thead>
<tr>
<th>Potential complication</th>
<th>Incidence</th>
<th>Preventive measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inadvertent RV puncture</td>
<td>4.5%–17% (without significant bleeding)²,³</td>
<td>1. Ensure no continual aspiration of blood</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Ensure contrast injection results in a thin outline of the pericardium around the cardiac silhouette</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Ensure guidewire that is passed through is unrestricted, wraps around the left and the right cardiac silhouette and crosses multiple heart chambers in the LAO projection</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Ensure no premature ventricular complexes are observed</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Do not insert sheath until confirmed in the pericardial space</td>
</tr>
<tr>
<td>Pericardial bleed</td>
<td>10%–30% (mild, self-limiting)¹,²</td>
<td>1. Avoid anticoagulation before epicardial access</td>
</tr>
<tr>
<td></td>
<td>4.5% (intrapericardial bleeding &gt; 80 mL)²</td>
<td>2. Examine aspirated pericardial fluid</td>
</tr>
<tr>
<td></td>
<td>0.6% (delayed tamponade)²</td>
<td>3. Consider surgical repair if hemostasis not achieved</td>
</tr>
<tr>
<td>Injury to subdiaphragmatic vessels and abdominal viscera</td>
<td>0.5%⁹</td>
<td>1. Beware in cases of congestive heart failure and hepatomegaly</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Ensure adequate palpation of the level of the xiphoid process</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Apply gentle downward and leftward pressure in the epigastrium to steer the liver away from the course of the needle</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Adjust needle angle from the left lateral projection to avoid the diaphragm</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Maintain trajectory of the needle with no sideways movements</td>
</tr>
<tr>
<td>Coronary vessel damage</td>
<td>0.6% (coronary artery stenosis)²</td>
<td>1. Beware in cases of abnormally dilated RV (acute marginal artery)</td>
</tr>
<tr>
<td></td>
<td>0.6% (myocardial infarction)²</td>
<td>2. Monitor pericardial aspirate</td>
</tr>
<tr>
<td>Phrenic nerve injury</td>
<td>0% (during epicardial puncture)²¹</td>
<td>3. Perform coronary angiography before ablation and if suspected vessel damage</td>
</tr>
<tr>
<td></td>
<td>Maneuvers to avoid injury during ablation⁹</td>
<td>1. Perform high-output pacing for phrenic nerve capture</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Consider methods to interpose the ablation site and the phrenic nerve</td>
</tr>
<tr>
<td>Pneumopericardium</td>
<td>Uncommon</td>
<td>1. Results in decreased defibrillator threshold</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Minimize introduction of air during sheath insertion and catheter exchange</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Evacuate any air in the pericardium</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Ensure adequate placement of defibrillator patches, electrocardiographic patches, and electroanatomic mapping patches</td>
</tr>
<tr>
<td>Pericarditis (postprocedural)</td>
<td>Mild symptomatic pericarditis in almost all patients²</td>
<td>1. Remove the catheter and sheath at the end of the procedure when no further bleeding is observed</td>
</tr>
<tr>
<td></td>
<td>0.6% (severe pericardial reaction)⁶</td>
<td>2. Administer anti-inflammatory agents</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Consider steroid injection into the pericardial space</td>
</tr>
<tr>
<td>Pleuritis (postprocedural)</td>
<td>Not uncommon</td>
<td>1. Administer anti-inflammatory agents</td>
</tr>
</tbody>
</table>

Lim et al. Heart Rhythm 2014
Epicardial access and ablation is feasible post cardiac surgery

Mechanical Circulatory Support: multicenter experience

- Impella or Tandem Heart v. IABP
- Non IABP group:
- Could map more and terminate more VTs by ablation, less rescue shocks but trend to more complications (NS)
- LVEF < 15% strong predictor of mortality

Reddy et al. Circ Arrhythm and Ep 2014
Electrical Storm

IV Amiodarone + IV Lidocaine + IV Esmolol
(if blood pressure permits)

IV Propofol or General Anesthesia

Left Stellate Ganglion Blockade

Hemodynamic Support

Catheter Ablation

Left or bilateral cardiac sympathectomy

Modified from Schleifer et al. Cardiol Clin 2013
Cardiac sympathetic denervation for treatment of refractory ventricular arrhythmias or electrical storm

Veseghi et al. Heart Rhythm 2014

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
<th>Bilateral CSD (N = 27)</th>
<th>Left CSD (N = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>23 (85%)</td>
<td>12 (86%)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>57 ± 14</td>
<td>63 ± 11</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICM</td>
<td>4 (15%)</td>
<td>5 (36%)</td>
</tr>
<tr>
<td>NICM</td>
<td>15 (56%)</td>
<td>7 (50%)</td>
</tr>
<tr>
<td>HCM</td>
<td>1 (3.7%)</td>
<td>2 (14%)</td>
</tr>
<tr>
<td>Sarcoid</td>
<td>2 (7.4%)</td>
<td></td>
</tr>
<tr>
<td>Chagasie</td>
<td>1 (3.7%)</td>
<td></td>
</tr>
<tr>
<td>ARVC</td>
<td>1 (3.7%)</td>
<td></td>
</tr>
<tr>
<td>Valvular</td>
<td>1 (3.7%)</td>
<td></td>
</tr>
<tr>
<td>TGA (+infarct)</td>
<td>1 (3.7%)</td>
<td></td>
</tr>
<tr>
<td>None (idiopathic VF)</td>
<td>1 (3.7%)</td>
<td></td>
</tr>
<tr>
<td>Type of VT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PMVT/VF</td>
<td>6 (22%)</td>
<td>2 (14%)</td>
</tr>
<tr>
<td>MMWTS</td>
<td>21 (78%)</td>
<td>12 (86%)</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>32 ± 13</td>
<td>29 ± 13</td>
</tr>
<tr>
<td>Pulmonary artery pressure (mm Hg)</td>
<td>38 ± 12.5</td>
<td>39 ± 9.8</td>
</tr>
<tr>
<td>Median no. of VT ablations</td>
<td>2 (0–5)</td>
<td>2 (0–4)</td>
</tr>
</tbody>
</table>

Number of ICD Shocks

[Graph showing pre-procedure and post-procedure number of ICD shocks for Left CTS and Bilateral CTS]

ICD Shock Free Survival - Bilateral vs. Left CSD

[Graph showing ICD shock free survival for bilateral and left CSD]

Veseghi et al. Heart Rhythm 2014
Approach to sustained VT in patients with cardiomyopathy/structural heart disease

1. Identify reversible precipitating factors
2. Optimize medications (BB/AAD) and device programming
3. Consider ablation: monomorphic VTs are favored, acceptable procedure risk, LVEF, comorbidities, epicardial/endocardial/combined, mechanical support, patient preference

Modified from Stevenson WG. Heart Rhythm 2014
Final Thoughts

• It is critical to understand the underlying substrate for the VT/PVC→ cardiomyopathy/scar
  – Growing role of advanced imaging modalities: MRI, invasive voltage mapping
• Moderate to high density PVCs can be responsible for cardiomyopathy
• Catheter ablation is effective at treating VT and PVCs in “normal” hearts
Final Thoughts

• VT is common in patients with cardiomyopathies/structural heart disease
• ICD, CRT devices are proven and effective in reducing mortality
• The continually evolving substrate is in part responsible for the difficulty in achieving long term cure with single VT ablation procedures
• Catheter ablation is effective in reducing arrhythmia burden, however, there is significant room for improvement...
## Table 1: Specific Management of NSVT

<table>
<thead>
<tr>
<th>Clinical Setting</th>
<th>Investigations</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic NSVT</td>
<td>Differentiate from ARVC</td>
<td>Beta-blockers, calcium-channel blockers; RF ablation if inducible sustained VT, progressively reduced LVEF, or symptoms</td>
</tr>
<tr>
<td>Arrhythmogenic ventricular cardiomyopathy</td>
<td>Value of EPS not established; NSVT indicates intermediate arrhythmic risk (&lt;2% per year)</td>
<td>Not established; perhaps amiodarone or sotalol; ICD frequently considered</td>
</tr>
<tr>
<td>Hypertension, valve disease</td>
<td>No need for specific management</td>
<td>Optimal antihypertensive therapy including beta-blockers</td>
</tr>
<tr>
<td>Non-STE ACS, NSVT &gt;48 h after admission</td>
<td>Meticulous ischemia testing</td>
<td>Revascularization and optimal medical therapy</td>
</tr>
<tr>
<td>Acute MI, NSVT &gt;13–24 h until pre-discharge</td>
<td>Routine for acute MI</td>
<td>Revascularization and optimal medical therapy</td>
</tr>
<tr>
<td>Previous MI with LVEF of 31%–40%</td>
<td>Ischemia testing; EPS†</td>
<td>Revascularization and optimal medical therapy; if EP-inducible monomorphic VT or VF;† ICD§</td>
</tr>
<tr>
<td>Previous MI with LVEF &lt;30% or LVEF &lt;35% and NYHA functional class II/III</td>
<td>Ischemia testing; no EPS†</td>
<td>Revascularization and optimal medical therapy; ICD§</td>
</tr>
<tr>
<td>Asymptomatic CAD with EF &gt;40%</td>
<td>Ischemia testing</td>
<td>Revascularization and optimal medical therapy; no need for specific NSVT therapy</td>
</tr>
<tr>
<td>Syncope in CAD with EF &gt;40%</td>
<td>Ischemia testing; EP testing†</td>
<td>Revascularization and optimal medical therapy; if EP-inducible monomorphic VT or VF;† ICD</td>
</tr>
<tr>
<td>Nonischemic dilated cardiomyopathy</td>
<td>Value of EP testing not established</td>
<td>Optimal CCF therapy (medical and CRT if indicated); ablation for bundle branch re-entry; ICD for syncope or LVEF ≤35% and NYHA functional class II/III</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Evaluate additional risk factors: previous cardiac arrest, unexplained syncope, massive LV hypertrophy (≥30 mm), hypotensive or attenuated blood pressure response to upright exercise</td>
<td>Beta-blockers; ICD, especially with frequent and prolonged (&gt;10 beats) episodes of NSVT</td>
</tr>
<tr>
<td>Congenital heart disease (usually repaired Fallot)</td>
<td>EP testing</td>
<td>Predictive value of NSVT not established; consider corrective surgery; if VT inducible, ablation and ICD</td>
</tr>
<tr>
<td>Long QT syndrome</td>
<td>Genotype analysis useful</td>
<td>Beta-blockers; if syncope despite beta-blockers, ICD</td>
</tr>
<tr>
<td>Catecholaminergic polymorphic VT</td>
<td>Value of EP testing not established</td>
<td>Beta-blockers and perhaps calcium-channel blockers; if cardiac arrest, ICD</td>
</tr>
<tr>
<td>Brugada syndrome</td>
<td>Value of EP testing disputed</td>
<td>Possibly quinidine (more data needed); If cardiac arrest, ICD</td>
</tr>
</tbody>
</table>
NSVT
Monomorphic VT
- No Structural Heart Disease / Idiopath
- Scar-related
- Purkinje-related

Polymorphic VT
- Acute myocardial ischemia
- Ventricular scar, hypertrophy, failure
- Genetic sudden death syndromes
  - Long QT, short QT
  - Brugada
  - CPVT

Slow Sinusoidal VT
- Hyperkalemia
- Drug toxicity
- End stage heart disease
Primary Prevention ICD in Patients With LV Dysfunction

• 21-29 % relative reduction in mortality with ICD
  – NNT of 14 to 18 over 36 months.
  – 65% reduction in arrhythmic events

• Benefit in addition to:
  – 6.1% ARR with ACE inhibitors
  – 4.4% ARR with β-blockers

Al-Katib AHJ 2005
### Electrocardiographic Features of Idiopathic Ventricular Tachycardias

<table>
<thead>
<tr>
<th>Localization of VT</th>
<th>BBB</th>
<th>Axis</th>
<th>Precordial Transition</th>
<th>V₁</th>
<th>V₆</th>
<th>I</th>
<th>Other ECG Features</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left ventricle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASOVT LCC</td>
<td>LBBB</td>
<td>Inferior</td>
<td>≤ V₃</td>
<td>rS, RS</td>
<td>R</td>
<td>rS</td>
<td>Notched M or W in V₁, QS or RS in lead I. Early transition, broad R in V₂. Positive in V₃. Notched on the downward deflection in V₁ or W pattern.</td>
</tr>
<tr>
<td>RCC</td>
<td>LBBB</td>
<td>Inferior</td>
<td>≤ V₃</td>
<td>rS, RS</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>LCCC/RCC junction</td>
<td>LBBB</td>
<td>Inferior</td>
<td>V₃</td>
<td>gS</td>
<td>R</td>
<td>R/Re'</td>
<td></td>
</tr>
<tr>
<td>LVOT Septal sites LAMC</td>
<td>LBBB</td>
<td>Inferior</td>
<td>Early</td>
<td>QS/Qs</td>
<td>Rs</td>
<td>Rs</td>
<td>Ratio of QS in II and III = 1. Positive precordial concordance and no S in V₃.</td>
</tr>
<tr>
<td>MA</td>
<td>RBBB</td>
<td>Inferior</td>
<td>None</td>
<td>qS</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Posterior</td>
<td>RBBB</td>
<td>Superior</td>
<td>Early</td>
<td>R</td>
<td>Rs</td>
<td>Rs</td>
<td>Late inferior lead notching; wider QRS, late S wave. Absence of notching in inferior leads.</td>
</tr>
<tr>
<td>Epicardial AVJ/SVC junction</td>
<td>LBBB</td>
<td>Inferior</td>
<td>Early</td>
<td>rS/QS</td>
<td>Rs</td>
<td>Rs</td>
<td>Precedural pattern break with abrupt loss of R waves in V₂. MDI &gt; 0.55. Positive concordance V₂-V₃. MDI &gt; 0.55; slurred intraventricular deflection.</td>
</tr>
<tr>
<td>Crux</td>
<td>LBBB</td>
<td>Inferior</td>
<td>Early</td>
<td>Variable</td>
<td>R</td>
<td>Rs</td>
<td></td>
</tr>
<tr>
<td>Papillary muscle</td>
<td>RBBB</td>
<td>Superior</td>
<td>Variable</td>
<td>rR</td>
<td>R</td>
<td>R</td>
<td>Late R to S precordial transition. qR pattern in lead aVL and rS pattern in lead V₆.</td>
</tr>
<tr>
<td>Posteroventricular</td>
<td>RBBB</td>
<td>Inferior</td>
<td>Variable</td>
<td>rR</td>
<td>R</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Fascicular</td>
<td>RBBB</td>
<td>Left superior</td>
<td>Early</td>
<td>rR</td>
<td>R</td>
<td>S</td>
<td>Loss of late precordial R waves with more apical axis. Similar to posterior fascicular apart from axis. Narrow QRS complex with VA dissociation.</td>
</tr>
<tr>
<td>Left posterior</td>
<td>RBBB</td>
<td>Right</td>
<td>None</td>
<td>rR</td>
<td>Q</td>
<td>G</td>
<td></td>
</tr>
<tr>
<td>Left anterior</td>
<td>RBBB</td>
<td>Right</td>
<td>None</td>
<td>rR</td>
<td>Q</td>
<td>G</td>
<td></td>
</tr>
<tr>
<td>Upper septal</td>
<td>LBBB</td>
<td>Normal or right</td>
<td>V₂</td>
<td>rS</td>
<td>Rs</td>
<td>Rs</td>
<td></td>
</tr>
<tr>
<td><strong>Right ventricle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RVOT Anterior, septal</td>
<td>LBBB</td>
<td>Inferior</td>
<td>≤ V₃</td>
<td>rS</td>
<td>R</td>
<td>rS</td>
<td>Early transition with lead I negative, isoelectric, or multiphasic. Late transition; broad late notched inferior leads and lead I positive isoelectric or positive aVL, large R amplitude in I, V₃ and V₆, taller inferior R.</td>
</tr>
<tr>
<td>Posterior, free wall</td>
<td>LBBB</td>
<td>Inferior</td>
<td>≥ V₃</td>
<td>rS</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
<tr>
<td>Parahilarisia</td>
<td>LBBB</td>
<td>Inferior</td>
<td>≥ V₃</td>
<td>qS</td>
<td>R</td>
<td>R</td>
<td></td>
</tr>
</tbody>
</table>

### Localization of VT

<table>
<thead>
<tr>
<th>Localization of VT</th>
<th>BBB</th>
<th>Axis</th>
<th>Precordial Transition</th>
<th>V₁</th>
<th>V₆</th>
<th>I</th>
<th>Other ECG Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA</td>
<td>LBBB</td>
<td>Inferior</td>
<td>&gt; V₃</td>
<td>rS/Qs</td>
<td>R</td>
<td>rS/Qs</td>
<td>1 &gt; aV₁/aV₆ ratio of the Q-wave amplitude, 1 &lt; R/S ratio in lead V₂.</td>
</tr>
<tr>
<td>TA</td>
<td>LBBB</td>
<td>Inferior</td>
<td>&lt; V₃</td>
<td>Qs</td>
<td>R</td>
<td>R/r</td>
<td>Positive, isoelectric, or multiphasic in aVL. Notching in limb leads; discordant forces in inferior leads if inferior.</td>
</tr>
<tr>
<td>Free-wall</td>
<td>LBBB</td>
<td>Variable</td>
<td>V₄-V₅</td>
<td>rS</td>
<td>R</td>
<td>R/r</td>
<td></td>
</tr>
</tbody>
</table>
Types and Etiologies of Sustained Ventricular Tachycardias

Monomorphic VT
- No Structural Heart Disease / Idiopathic
- Scar-related
- Purkinje-related

Polymorphic VT
- Acute myocardial ischemia
- Ventricular scar, hypertrophy, failure
- Genetic sudden death syndromes
  - Long QT, short QT
  - Brugada
  - CPVT

Slow Sinusoidal VT
- Hyperkalemia
- Drug toxicity
- End stage heart disease

---

William G. Stevenson, MD, FHRS

(Hart Rhythm 2013;10:1919–1926) © 2013 Heart Rhythm Society. All rights reserved.
### Table 1: Specific Management of NSVT

<table>
<thead>
<tr>
<th>Clinical Setting</th>
<th>Investigations</th>
<th>Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic NSVT</td>
<td>Differentiate from ARVC</td>
<td>Beta-blockers, calcium-channel blockers; RF ablation if inducible sustained VT, progressively reduced LVEF, or symptoms</td>
</tr>
<tr>
<td>Arrhythmogenic ventricular cardiomyopathy</td>
<td>Value of EPS not established; NSVT Indicates Intermediate arrhythmic risk (&lt;2% per year)</td>
<td>Not established; perhaps amiodarone or sotalol; ICD frequently considered</td>
</tr>
<tr>
<td>Hypertension, valve disease</td>
<td>No need for specific management</td>
<td>Optimal antihypertensive therapy including beta-blockers</td>
</tr>
<tr>
<td>Non-ST ACS, NSVT &gt;48 h after admission</td>
<td>Meticulous ischemia testing</td>
<td>Revascularization and optimal medical therapy*</td>
</tr>
<tr>
<td>Acute MI, NSVT &gt;13-24 h until pre-discharge</td>
<td>Routine for acute MI</td>
<td>Revascularization and optimal medical therapy</td>
</tr>
<tr>
<td>Previous MI with LVEF of 31%-40%</td>
<td>Ischemia testing; EPS†</td>
<td>Revascularization and optimal medical therapy; if EP-inducible monomorphic VT or VF; ICD§</td>
</tr>
<tr>
<td>Previous MI with LVEF &lt;30% or LVEF &lt;=35% and NYHA functional class II/III</td>
<td>Ischemia testing; no EPS†</td>
<td>Revascularization and optimal medical therapy; ICD§</td>
</tr>
<tr>
<td>Asymptomatic CAD with EF &gt;40%</td>
<td>Ischemia testing</td>
<td>Revascularization and optimal medical therapy; no need for specific NSVT therapy</td>
</tr>
<tr>
<td>Syncope in CAD with EF &gt;40%</td>
<td>Ischemia testing; EP testing†</td>
<td>Revascularization and optimal medical therapy; if EP-inducible monomorphic VT or VF; ICD§</td>
</tr>
<tr>
<td>Nonischemic dilated cardiomyopathy</td>
<td>Value of EP testing not established</td>
<td>Optimal CCF therapy (medical and CRT if indicated); ablation for bundle branch re-entry; ICD for syncope CAD functional class II/III especially with frequent and episodes of NSVT</td>
</tr>
</tbody>
</table>

*Katritsii et al.
Nonsustained Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Congenital heart disease (usually repaired Fallot)</th>
<th>EP testing</th>
<th>Program value of NSVT not established; consider corrective surgery; if VT inducible, ablation and ICD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long QT syndrome</td>
<td>Genotype analysis useful</td>
<td>Beta-blockers, if syncope despite beta-blockers, ICD</td>
</tr>
<tr>
<td>catecholaminergic polymorphic VT</td>
<td>Value of EP testing not established</td>
<td>Beta-blockers and perhaps calcium-channel blockers; if cardiac arrest, ICD</td>
</tr>
<tr>
<td>Brugada syndrome</td>
<td>Value of EP testing disputed</td>
<td>Possibly quinidine (more data needed); if cardiac arrest, ICD</td>
</tr>
</tbody>
</table>
PVCs in Idiopathic VF (normal appearing hearts)

Knecht et al. JACC 2009
Diseases/syndromes in which PVCs from the Purkinje system or RV outflow tract that initiate VF or polymorphic VT have been ablated

- Idiopathic VF
- Brugada syndrome
- Long QT syndrome
- Early or late after myocardial infarction
- Cardiac amyloidosis
- Cardiomyopathy
- Myocarditis
Figure 5  Recordings from a patient with prior anteroseptal infarct and recurrent VT, illustrating findings during substrate mapping. A: An LV voltage map (left anterior projection) with purple color indicating a bipolar voltage > 1.5 mV, and a large low-voltage scar (red, yellow, green, blue area). B: The intracardiac ultrasound imaging corresponding to the planar slide of the ultrasound beam seen in panel A. C: The last beat of pace mapping from the ablation catheter at a site with a long S-QRS interval of 180 ms in the low-voltage area. After termination of LV pace mapping, an LP is seen with the RV paced beat at the right side of the panel (arrow). Abl = ablation; d = distal; LP = late potential; LV = left ventricular; p = proximal; RV = right ventricular; VT = ventricular tachycardia.
Figure 3. Role of local abnormal ventricular activities (LAVAs) in the induction of ventricular tachycardia (VT) and the influence of radiofrequency energy on LAVAs. Before radiofrequency energy delivery (RF): A, At first sight, the local ventricular electrogram during baseline paced rhythm looks simple. However, in the terminal portion of this simple-looking signal, a very high-frequency component (LAVA) can be identified. B, Programmed electric stimulation from the right ventricle (RV) unmask the LAVA potential by increasing the delay from the far-field signal. The delay observed during RV pacing suggests poor coupling of the muscle bundle generating the LAVA signal. The delay is maximal with S3, which is associated not only with a change in the polarity of LAVA but also with the induction of VT. Post-RF energy delivery: C, After delivery of RF energy, there is a remarkable delay (see A) between the far-field ventricular signal and LAVAs during baseline paced rhythm. D, Repeat programmed electric stimulation from the RV results in the absence of LAVA signals after the far-field ventricular potential during S2 and S3 (open arrows). The absence of LAVAs is associated with an inability to induce the VT. Although ablation has rendered VT noninducible, further RF energy application is indicated to completely eliminate the LAVAs.
Figure 3: Endo and Epi Voltage Map With Wall Thinning Segmentation in Patient With ICM

(A) Endo bipolar voltage map shows low voltage with the presence of LAVA (light blue dots) at the basal posterolateral left ventricle. A peak-to-peak bipolar amplitude <1.5 mV is defined as low voltage, and <0.5 mV is defined as dense scar. (B) Endo unipolar voltage map is shown. A unipolar amplitude of <8.3 mV is defined as abnormal voltage. (C) Epi bipolar voltage map merged with the multidetector computed tomography model shows the location of low-voltage area and LAVA corresponding to the wall thinning segmentation (white area). Endo ablation successfully eliminated the posterolateral LAVA (in blue box), which had the following characteristics: low bipolar amplitude; location within the wall thinning segmentation; opposite Endo low unipolar voltage; and presence of LAVA at the opposite endocardium. On the contrary, Epi LAVA located mid-anteriorly (in red box) could not be eliminated by Endo ablation and needed to be ablated epicardially. They were of higher bipolar voltage (>1.5 mV), located outside the wall thinning segmentation, and opposite endocardium with higher unipolar amplitude. Abbreviations as in Figure 1.

Komatsu et al.  
Endocardial Ablation for Epicardial VT Substrate

JACC Vol. 63, No. 14, 2014
April 15, 2014:1416-26
Voltage Channels

Figure 4  VT Isthmus Not Associated With Channel

Bipolar voltage map (inferior view) of a patient with inferior scar (left panel). With adjustment of voltage cutoff, a channel is identified that does not harbor the identified isthmus (right panel, red tag). Entrainment from the isthmus site before ablation is shown in the bottom panel. Only 1 radiofrequency lesion was delivered in this case, denoted by the red tag, terminating a recurrent clinical VT and rendering it noninducible. The patient has had no VT after 6 months of follow-up.
Channels could be identified in 88% of patients with postinfarction VT by adjusting the voltage limits of bipolar maps; however, the specificity of those channels in predicting the location of VT isthmus sites was only 30%. The presence of ILPs inside the voltage channel significantly increased the specificity for identifying the clinical circuit. When VT is not inducible or hemodynamically tolerated, voltage channels containing ILPs could be targeted as a proxy for VT isthmus sites. This ablation strategy needs to be evaluated prospectively.
# Voltage Channels

## Objectives
The goal of this study was to determine the relationship of the ventricular tachycardia (VT) isthmus to channels of preserved voltage on an electroanatomic voltage map in postinfarction cardiomyopathy.

## Background
Substrate mapping in patients with postinfarction cardiomyopathy and VT may involve lowering the voltage cutoff that defines the scar (<1.5 mV) to identify “channels” of relative higher voltage within the scar. However, the prevalence of channels within the scar identified by using electroanatomic mapping and the relationship to the protected VT isthmus identified by entrainment mapping is unknown.

## Methods
Detailed bipolar endocardial voltage maps (398 ± 152 points) from 24 patients (mean age 69 ± 9 years) with postinfarction cardiomyopathy (ejection fraction 33 ± 9%) and tolerated VT were reviewed. Endocardial scar was defined according to voltage <1.5 mV. Isolated late potentials (ILPs) were identified and tagged on the electroanatomic voltage map. The baseline voltage cutoffs were then adjusted until all channels were identified. The VT isthmus was identified using entrainment mapping.

## Results
Inferior and anterior/lateral infarction was present by voltage mapping in 18 and 6 patients, respectively (scar area 44 ± 24 cm²). By adjusting voltage cutoffs, 37 channels were identified in 21 (88%) of 24 patients. The presence of ILPs within a channel was seen in 11 (46%) of 24 patients and 17 (46%) of 37 channels. A VT isthmus site was contained within a channel in only 11 of 24 patients or 11 of 37 channels. No difference in voltage characteristics was identified between clinical and nonclinical channels. Voltage channels with ILPs harbored the clinical isthmus with a sensitivity and specificity of 78% and 85%, respectively.

## Conclusions
Channels were identified in 88% of patients with VT by adjusting the voltage limits of bipolar maps; however, the specificity of those channels in predicting the location of VT isthmus sites was only 30%. The presence of ILPs inside the voltage channel significantly increases the specificity for identifying the clinical VT isthmus. *(J Am Coll Cardiol 2013;61:2088-95) © 2013 by the American College of Cardiology Foundation*
Mechanical Circulatory Support: multicenter experience

Table 2. Comparison of Procedural Variables Between Patients With Different Percutaneous Left Ventricular Assist Devices During Ventricular Tachycardia Ablation

<table>
<thead>
<tr>
<th>Variable</th>
<th>IABP (N=22)</th>
<th>Non-IABP Combined (N=44)</th>
<th>Non-IABP Subgroups</th>
<th>P Value (IABP vs Non-IABP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of VTs induced</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>3.3±1.5</td>
<td>3.1±1.9</td>
<td>2.5±1.7</td>
<td>3.2±1.8</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td>3 (1–7)</td>
<td>3 (0–7)</td>
<td>2 (0–7)</td>
<td>4 (2–7)</td>
</tr>
<tr>
<td>No. of nonclinical VTs induced</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>1.2±1.1</td>
<td>1.0±1.5</td>
<td>0.64±1.1</td>
<td>1.7±1.9</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td>1 (0–4)</td>
<td>0.5 (0–6)</td>
<td>0 (0–5)</td>
<td>1 (0–6)</td>
</tr>
<tr>
<td>No. of VTs ablated</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>1.8±1.0</td>
<td>2.4±1.3</td>
<td>1.9±1.1</td>
<td>3.0±1.2</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td>1 (1–4)</td>
<td>2 (1–6)</td>
<td>1 (1–5)</td>
<td>3 (1–6)</td>
</tr>
<tr>
<td>No. of unstable VTs mapped and ablated</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>0.32±0.48*</td>
<td>1.05±0.78*</td>
<td>1.12±0.83</td>
<td>0.95±0.70</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td>0 (0–1)*</td>
<td>1 (0–3)*</td>
<td>1 (0–3)</td>
<td>1 (0–2)</td>
</tr>
<tr>
<td>No. of VTs RF terminated</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>0.91±0.81*</td>
<td>1.59±1.00*</td>
<td>1.16±0.85</td>
<td>2.16±0.90</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td>1 (0–4)*</td>
<td>1 (0–4)*</td>
<td>1 (0–3)</td>
<td>2 (1–4)</td>
</tr>
<tr>
<td>Entrainment/activation mapping, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>13 (59)*</td>
<td>36 (82)*</td>
<td>20 (80)</td>
<td>16 (84)</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>External shocking</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean±SD</td>
<td>3.0±1.5*</td>
<td>1.9±2.2*</td>
<td>1.6±2.8</td>
<td>2.3±1.2</td>
</tr>
<tr>
<td>Median (min–max)</td>
<td>3 (0–6)*</td>
<td>2 (0–13)*</td>
<td>1 (0–13)</td>
<td>2 (0–15)</td>
</tr>
<tr>
<td>Fluoroscopy time, min, median (Q1–Q3)</td>
<td>63 (52–72)</td>
<td>63 (53–73)</td>
<td>68 (54–74)</td>
<td>58 (48–70)</td>
</tr>
<tr>
<td>RF time, min, median (Q1–Q3)</td>
<td>36 (27–45)</td>
<td>31 (18–41)</td>
<td>36 (13–50)</td>
<td>28 (19–37)</td>
</tr>
<tr>
<td>Procedure time, min, median (Q1–Q3)</td>
<td>302 (242–345)</td>
<td>339 (260–417)</td>
<td>291 (247–346)</td>
<td>379 (328–433)</td>
</tr>
<tr>
<td>Ventilation time, h, median (Q1–Q3)</td>
<td>16 (12–25)</td>
<td>12 (6–24)</td>
<td>6 (6–29)</td>
<td>20 (12–24)</td>
</tr>
</tbody>
</table>

IABP indicates intra-aortic balloon pump; RF: Radiofrequency; and VT, ventricular tachycardia. *Statistically significant.
# Mechanical Circulatory Support: multicenter experience

## Table 4. Predictors of Long-Term Mortality (Mean Follow-Up 12 Months) After Ventricular Tachycardia Ablation Using a Percutaneous Left Ventricular Assist Device

<table>
<thead>
<tr>
<th></th>
<th>Alive at Last Follow-Up (N=46)</th>
<th>Dead by Last Follow-Up (N=20)</th>
<th>P Value</th>
<th>Univariate Analysis</th>
<th>Multivariate Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean±SD</td>
<td>65±12</td>
<td>71±10</td>
<td>0.052</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior VT ablation, %</td>
<td>11 (24)</td>
<td>11 (55)</td>
<td>0.014</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRT-D device, %</td>
<td>13 (28)*</td>
<td>13 (65)*</td>
<td>0.005*</td>
<td></td>
<td>12.9*</td>
</tr>
<tr>
<td>Atrial fibrillation, %</td>
<td>15 (33)</td>
<td>12 (60)</td>
<td>0.038</td>
<td></td>
<td>1.3–126.4*</td>
</tr>
<tr>
<td>Amiodarone, %</td>
<td>25 (54)</td>
<td>18 (90)</td>
<td>0.005</td>
<td></td>
<td>0.2–54.2</td>
</tr>
<tr>
<td>LVEF, mean±SD</td>
<td>32±13</td>
<td>17±7</td>
<td>&lt;0.001</td>
<td></td>
<td>0.028</td>
</tr>
<tr>
<td>LVEF≤15%</td>
<td>6 (13)</td>
<td>11 (55)</td>
<td>&lt;0.001</td>
<td></td>
<td>0.023</td>
</tr>
<tr>
<td>In ICU before the ablation, %</td>
<td>16 (35)*</td>
<td>16 (80)*</td>
<td>0.001*</td>
<td></td>
<td>1.5–298.8*</td>
</tr>
<tr>
<td>No. of days in the hospital, mean±SD</td>
<td>6.1±4.8</td>
<td>11.9±8.5</td>
<td>0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of rescue shocks, mean±SD</td>
<td>1.96±1.5</td>
<td>3.05±3.0</td>
<td>0.050</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CI indicates confidence interval; CRT-D, cardiac resynchronization therapy-defibrillator; ICU, intensive care unit; LVEF, left ventricular ejection fraction; and VT, ventricular tachycardia.

*Statistically significant.

Reddy et al. Circ Arrhythm and Ep 2014
Percutaneous Left Ventricular Assist Devices in Ventricular Tachycardia Ablation
Multicenter Experience

Yeruva Madhu Reddy, MD; Larry Chinitz, MD; Moussa Mansour, MD; T. Jared Bunch, MD; Srijoy Mahapatra, MD; Vijay Swarup, MD; Luigi Di Biase, MD, PhD; Sudharani Bommana, MS; Donita Atkins, BS; Roderick Tung, MD; Kalyanam Shivkumar, MD, PhD; J. David Burkhardt, MD; Jeremy Ruskin, MD; Andrea Natale, MD; Dhanunjaya Lakkireddy, MD

**Background**—Data on relative safety, efficacy, and role of different percutaneous left ventricular assist devices for hemodynamic support during the ventricular tachycardia (VT) ablation procedure are limited.

**Methods and Results**—We performed a multicenter, observational study from a prospective registry including all consecutive patients (N=66) undergoing VT ablation with a percutaneous left ventricular assist devices in 6 centers in the United States. Patients with intra-aortic balloon pump (IABP group; N=22) were compared with patients with either an Impella or a TandemHeart device (non-IABP group; N=44). There were no significant differences in the baseline characteristics between both the groups. In non-IABP group (1) more patients could undergo entrainment/activation mapping (82% versus 59%; *P*=0.046), (2) more number of unstable VTs could be mapped and ablated per patient (1.05±0.78 versus 0.32±0.48; *P*<0.001), (3) more number of VTs could be terminated by ablation (1.59±1.0 versus 0.91±0.81; *P*=0.007), and (4) fewer VTs were terminated with rescue shocks (1.9±2.2 versus 3.0±1.5; *P*=0.049) when compared with IABP group. Complications of the procedure trended to be more in the non-IABP group when compared with those in the IABP group (32% versus 14%; *P*=0.143). Intermediate term outcomes (mortality and VT recurrence) during 12±5-month follow-up were not different between both groups. Left ventricular ejection fraction ≤15% was a strong and independent predictor of in-hospital mortality (53% versus 4%; *P*<0.001).

**Conclusions**—Impella and TandemHeart use in VT ablation facilitates extensive activation mapping of several unstable VTs and requires fewer rescue shocks during the procedure when compared with using IABP. *(Circ Arrhythm Electrophysiol. 2014;7:244-250.)*
Epicardial ECG characteristics

- **SHD**
  - Pseudo delta in RBBB VT’s > =34ms from QRS onset to rapid deflection
  - Delayed intrinsicoid deflection >=85ms QRS onset to peak R in V2
  - Shortest RS >= 120ms QRS onset to nadir of first S wave any precordial lead
  - NICM 4 step for epicard VT (basal sup and lat LV), inferior q, pseudo delta >=75ms. MDI > =0.59, q in I in VT.

- **No SHD**
  - Q’s in lat or inferior leads during VT
  - MDI >0.55 (shortest time to maximal + or – in any precordial lead divided QRSd
  - Pattren break loss of R from V1→V2 than prominent R in V3 /reverse pattern for MCV

Park et al. PACE 2012
Epicardial