Evaluation and Management of PVCs
The Good, the Bad, and the Ugly

Sahil Attawala, MD
Cardiac Electrophysiology
Princeton Baptist Medical Center
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To understand the range of symptoms and presentations of patients with frequent PVCs

To illustrate the importance of defining prognosis in these patients

To outline the therapeutic options to suppress or eliminate ventricular ectopy

To illustrate the role of ablative therapy in PVC management
“TRUE” STATEMENTS

“PVCs are benign…”

“PVCs are a sign of underlying heart disease…”

“PVCs can lead to heart failure…..”

“PVCs can be life threatening…”
“…Occasional pulse irregularities did not predict an adverse outcome.”

“However, frequent irregularities (1 in 10 beats) was associated with an ominous prognosis…often resulting in death within one year.”

-Chinese Physician Pien Ts’Io ~ 600 BC
PVCS ARE COMMON

**Table 2. Probability (%) of Observing a Given Number of Premature Ventricular Complexes During a Given Length of Observation in Subjects with Normal Hearts**

<table>
<thead>
<tr>
<th>No. of PVCs</th>
<th>0</th>
<th>≥1</th>
<th>&gt;5</th>
<th>&gt;10</th>
<th>&gt;50</th>
<th>&gt;100</th>
<th>&gt;500</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 hour (9-10 a.m.)</td>
<td>85</td>
<td>15</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3 hours (9 a.m.-12 noon)</td>
<td>78</td>
<td>22</td>
<td>9</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>6 hours (9 a.m.-3 p.m.)</td>
<td>74</td>
<td>26</td>
<td>14</td>
<td>8</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>8 hours (9 a.m.-5 p.m.)</td>
<td>72</td>
<td>28</td>
<td>15</td>
<td>10</td>
<td>3</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>12 hours (9 a.m.-9 p.m.)</td>
<td>64</td>
<td>36</td>
<td>22</td>
<td>15</td>
<td>5</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>24 hours</td>
<td>61</td>
<td>39</td>
<td>25</td>
<td>20</td>
<td>9</td>
<td>4</td>
<td>1</td>
</tr>
</tbody>
</table>

Abbreviation: PVC = premature ventricular complex.

**Average Number of PVC per 24 hours**

Kostis JB. Circulation 1981; 63: 1351-6
### Table 3. Effect of Age on the Probability (%) of Having More Than a Given Number of Premature Ventricular Complexes per 24 Hours in Subjects with Normal Hearts

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>n</th>
<th>&gt;0</th>
<th>&gt;50</th>
<th>&gt;100</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–29</td>
<td>6</td>
<td>16.7</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>30–39</td>
<td>11</td>
<td>18.2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>40–49</td>
<td>29</td>
<td>27.6</td>
<td>3.5</td>
<td>0</td>
</tr>
<tr>
<td>50–59</td>
<td>39</td>
<td>51.3</td>
<td>12.8</td>
<td>5.1</td>
</tr>
<tr>
<td>60–69</td>
<td>12</td>
<td>58.3</td>
<td>25.0</td>
<td>16.7</td>
</tr>
</tbody>
</table>

Abbreviation: PVC = premature ventricular complex.
PVCS ARE BENIGN...

Kennedy HL NEJM 1985
In pts with prior infarction, frequent or complex PVCs are associated with increased mortality

Hallstrom AP et al. JACC 1992; 20: 259-64
Ruberman W et al. NEJM 1977; 297: 750-7

Hypertension is associated with frequency and/or complexity of PVCs

Abdalla IS et al. Am J Cardiol 1987; 60: 1036-42.

In pts with LVH, frequent PVCs are associated with increased mortality

Bikkina M et al. JACC 1993; 22: 1111-16

...EXCEPT WHEN THEY ARE NOT BENIGN.
MORTALITY AND MORBIDITY IN PATIENTS RECEIVING ENCAINIDE, FLECAINIDE, OR PLACEBO

The Cardiac Arrhythmia Suppression Trial

PVCs may be associated with adverse outcomes in MI patients…but PVC treatment can cause more harm than good
PVCS AND “NORMAL” HEARTS

Benign Prognosis
✓ Circulation 1971; 44: 617-25
✓ Chest 1973; 64: 564-9
✓ Cardiology 1983; 70 (Suppl 1): 82-7
✓ Eur Heart J 1983; 4: 338-46
✓ Am J Cardiol 1992; 70: 748-51
✓ Jpn Circ J 1994; 58: 190-8
✓ J Intern Med 1999; 246: 363-72
✓ J Am Coll Cardiol 2001; 38: 364-70
✓ Heart 2009; 95: 1230-7

Adverse Outcomes
✓ Am Heart J 1981; 101: 135-42
✓ Am J Cardiol 1987; 60: 1036-42
✓ Am J Cardiol 2006; 97: 1351-7
✓ J Cardiol 2010; 56: 23-6
✓ Am J Cardiol 2011; 107: 151-5
SYMPTOMS AND PVCS

Asymptomatic

Nonspecific
(Fatigue, Weakness, Exercise intolerance)

Typical
(Palpitations, Irregular and/or “pounding” Heart Beat)

Overt
(Heart Failure, Syncope/Cardiac Arrest from PVC-induced VT/VF)
**Severity of Symptoms**

**Associations/Triggers?**

- Worse at night/Positional
- Stress
- Caffeine
- Exercise

**Medications**

- Alpha-, Beta-, Dopamine-receptor agonists
- Over the counter stimulants

**Illicit drug use**

- Amphetamines, cocaine
Medical History

Structural heart disease/Hx of Heart Surgery
  MI, CHF, Valvular Hrt Dz
  Congenital Hrt Dz
  Inherited Diseases
  Hypertrophic Cardiomyopathy
  ARVC
  Long QT, Brugada, CPVT
Pertinent Non-cardiac disease
  Sarcoid
  Endocrinopathy
  Pulmonary Hypertension
HISTORY AND EXAM

Family History

Sudden death
Early MI/CAD
Inheritable Cardiac Disease

Exam

Findings of CHF or valvular heart disease
Elevated JVP, Crackles, Edema
Irregularity, Murmurs, S3, S4
Goiter
Confirm Diagnosis: 12 Lead ECG
### Heart Rate Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Beats</td>
<td>109357</td>
</tr>
<tr>
<td>Min HR</td>
<td>42 BPM at 12:53:58 AM</td>
</tr>
<tr>
<td>Avg HR</td>
<td>77 BPM</td>
</tr>
<tr>
<td>Max HR</td>
<td>77 BPM at 1:42:59 AM</td>
</tr>
</tbody>
</table>

#### Heart Rate Variability

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASDNN</td>
<td>102.5 mse</td>
</tr>
<tr>
<td>SDNN</td>
<td>122.6 mse</td>
</tr>
<tr>
<td>SDANN</td>
<td>92.8 mse</td>
</tr>
<tr>
<td>RMSSD</td>
<td>87.9 mse</td>
</tr>
</tbody>
</table>

#### QT Analysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QT Min</td>
<td>273 msec</td>
</tr>
<tr>
<td>QT Avg</td>
<td>387 msec</td>
</tr>
<tr>
<td>QT Max</td>
<td>497 msec</td>
</tr>
<tr>
<td>QTc Min</td>
<td>350 msec</td>
</tr>
<tr>
<td>QTc Avg</td>
<td>453 msec</td>
</tr>
<tr>
<td>QTc Max</td>
<td>631 msec</td>
</tr>
<tr>
<td>QTc &gt; 450 msec</td>
<td>42%</td>
</tr>
</tbody>
</table>

#### ST Episode Analysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Ch1</th>
<th>Ch2</th>
<th>Ch3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Min ST Level</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Max ST Level</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ST Episodes</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

#### Pacer Analysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus Beats</td>
<td>-</td>
</tr>
<tr>
<td>Paced Beats</td>
<td>-</td>
</tr>
<tr>
<td>Atrial Paced</td>
<td>-</td>
</tr>
<tr>
<td>Ventricular Paced</td>
<td>-</td>
</tr>
<tr>
<td>Dual Paced Beats</td>
<td>-</td>
</tr>
<tr>
<td>Fusion Beats</td>
<td>-</td>
</tr>
</tbody>
</table>

### Ventricular Ectopy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total VE Beats</td>
<td>40004 (36.6%)</td>
</tr>
<tr>
<td>Vent Runs Beats</td>
<td>0</td>
</tr>
<tr>
<td>Longest Beats</td>
<td>0</td>
</tr>
<tr>
<td>Fastest Beats</td>
<td>0 BPM</td>
</tr>
<tr>
<td>Triplets</td>
<td>2 Events</td>
</tr>
<tr>
<td>Couplets</td>
<td>1522 Events</td>
</tr>
<tr>
<td>Single/Interp PVC</td>
<td>2321/13219</td>
</tr>
<tr>
<td>R on T</td>
<td>0</td>
</tr>
<tr>
<td>Single/Late VE's</td>
<td>41/0</td>
</tr>
<tr>
<td>BI/Trigeminy</td>
<td>20525/846 Beats</td>
</tr>
</tbody>
</table>

### Supraventricular Ectopy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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</thead>
<tbody>
<tr>
<td>Total SVE Beats</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>Atrial Runs Beats</td>
<td>0</td>
</tr>
<tr>
<td>Longest Beats</td>
<td>0</td>
</tr>
<tr>
<td>Fastest Beats</td>
<td>0 BPM</td>
</tr>
<tr>
<td>Atrial PArs</td>
<td>0 Events</td>
</tr>
<tr>
<td>Drop/Late</td>
<td>0/0</td>
</tr>
<tr>
<td>Longest R-R</td>
<td>1.6 sec at 12:38:45 AM</td>
</tr>
<tr>
<td>Single PAC's</td>
<td>0</td>
</tr>
<tr>
<td>BI/Trigeminy</td>
<td>0/0 Beats</td>
</tr>
</tbody>
</table>

### Atrial Fibrillation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AFib Beats</td>
<td>0 (0.0%)</td>
</tr>
<tr>
<td>AFib Duration</td>
<td>0.0 min</td>
</tr>
</tbody>
</table>

### Interpretation
1. Basic Rhythm: Sinus. Rate 40 to 100/m. Average 70/m.
2. PVCs - mostly in the form of bigeminy - some interpolated - few couplets/salvos/No V tach. Total of 30% of beats scanned.
3. Rare PACs.
4. No ST-T wave changes.
5. Activity Log - No correlation.
Temporal relationship between PVCs and symptoms helps dictate management.  

12 lead ECG may be insufficient  
Continuous Monitoring is often needed  

24-72 hour HOLTER Monitor  
If symptoms are frequent  

30 day Event Recorder  
If symptoms are less frequent  

Implantable loop recorder (ILR)  
Rare, but significant events
NONINVASIVE IMAGING

Transthoracic Echocardiography

Assess ventricular function

Rule out valvular heart disease

Other: HCM, infiltrative CM

Exercise treadmill test

Especially, if exercise-induced

Elevated heart rate often suppresses “benign” PVCs

PVCs may be more prevalent during recovery

Dynamic ST changes or exercise induced multifocal PVCs or NSVT may indicate ischemic heart disease

Echo or nuclear imaging can be combined with exercise in patients with higher suspicion of CAD
NONINVASIVE IMAGING

Other:
Cardiac CT
Cardiac MRI
Positron-emission Tomography

Reserved when suspicion of specific, less common disorders is present
HCM
Cardiac Sarcoid
Infiltrative Cardiomyopathy
ARVC
PVCS WITH STRUCTURAL HEART DISEASE

Coronary Disease

PVC origin is commonly from ischemic tissue or scar

Reperfusion arrhythmias are common

Can increase likelihood of sustained VT/VF

Remember CAST!

Nonischemic Cardiomyopathy

Epicardial and basal LV origins are common

ARVC

PVCs are common with RV origin from regions of fibrofatty replacement of tissue

Frequent PVCs may interfere with biventricular pacing (CRT)
“MALIGNANT” PVCS
Ventricular outflow tracts are most common sites of origin

RVOT >>> Aortic Cusps/LVOT

Common Embryologic Origin

Other: Mitral/Tricuspid Annulus, Aortomitral continuity, papillary muscles, purkinje fibers

Triggered Activity

Catecholamine sensitive

Catheter ablation can be curative
Idiopathic PVCs: Anatomy

Points of Emphasis

• The RVOT runs anterior, leftward, and superior to the LVOT

• Tight Quarters: Many potential sites of origin requiring different approaches
First Evidence of Premature Ventricular Complex-Induced Cardiomyopathy: A Potentially Reversible Cause of Heart Failure

- 22 y/o Female with Fatigue/Palpitations
- Holter: 25-50,000 PVCs/24 hrs
- Echo: LVEF 43%, globally dilated LV
- Successful elimination of PVCs (Ablation)
- 6 months follow up: Normal LV, LVEF

Chugh SS et al. JCE 2000; 11: 328
PVC Burden > 24% associated with overtly diminished LV function

Bauman TS et al. Heart Rhythm 2010; 7: 865-69
Cardiomyopathy is reversible if PVC burden is substantially reduced

<table>
<thead>
<tr>
<th>Age, y</th>
<th>Sex</th>
<th>Presenting Symptom</th>
<th>Origin of CMP</th>
<th>Cardiac Medications</th>
<th>PVC Origin in RVOT</th>
<th>RFA Success</th>
<th>Initial Holter, PVCs/24 h</th>
<th>F/U Holter, PVCs/24 h</th>
<th>Initial EF, %</th>
<th>F/U EF, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>37</td>
<td>F</td>
<td>Dyspnea</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Anterior</td>
<td>Yes</td>
<td>5502</td>
<td>44</td>
<td>38</td>
<td>65</td>
</tr>
<tr>
<td>51</td>
<td>M</td>
<td>None</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>PosteroSeptal</td>
<td>Yes</td>
<td>26491</td>
<td>1893</td>
<td>45</td>
<td>60</td>
</tr>
<tr>
<td>80</td>
<td>M</td>
<td>Presyncope</td>
<td>Idiopathic</td>
<td>None</td>
<td>AnteroSeptal</td>
<td>Yes</td>
<td>35664</td>
<td>1100</td>
<td>35</td>
<td>55</td>
</tr>
<tr>
<td>51</td>
<td>M</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>None</td>
<td>Anterior</td>
<td>Yes</td>
<td>9791</td>
<td>5</td>
<td>35</td>
<td>60</td>
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<tr>
<td>71</td>
<td>F</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>PosteroSeptal</td>
<td>Yes</td>
<td>23352</td>
<td>117</td>
<td>43</td>
<td>65</td>
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<tr>
<td>62</td>
<td>M</td>
<td>Presyncope</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>PosteroLateral</td>
<td>Yes</td>
<td>...*</td>
<td>332</td>
<td>45</td>
<td>65</td>
</tr>
<tr>
<td>47</td>
<td>M</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>None</td>
<td>PosteroLateral</td>
<td>Yes</td>
<td>16362</td>
<td>55</td>
<td>45</td>
<td>65</td>
</tr>
<tr>
<td>68</td>
<td>F</td>
<td>Palpitations</td>
<td>Idiopathic</td>
<td>β-Blockers</td>
<td>Posterior</td>
<td>No</td>
<td>5626</td>
<td>12883</td>
<td>30</td>
<td>35</td>
</tr>
</tbody>
</table>

Yarlagadda RK et al CIRC 2005; 112: 1092
PVC INDUCED CARDIOMYOPATHY

Asymptomatic patients are more likely to develop cardiomyopathy

Yokokawa M et al. Heart Rhythm 2012: 9:92-95

PVC Burden greater than 15-20% have been associated with CM…but it has also been seen with lower burdens (4%).

Lee et al. Circ AE 2012; 5: 229-36
Yarlagadda RK. CIRC 2005; 112:1092

Subtle cardiac dysfunction likely exists in most patients with frequent PVCs and preserved EF…reversible after elimination.

Wijnmaalen PA et al. Heart 2010; 96:1275

Wide PVC duration, epicardial origin, and shorter coupling intervals also associated with cardiomyopathy

Yokokawa M et al. Heart Rhythm 2012: 9: 1460
Moulton KP et al CIRC 1990; 81: 1245
TREATMENT: LIFESTYLE CHANGES

Typical Advice

Avoid caffeine

Avoid smoking

Avoid excessive alcohol

Avoid triggers if present

Data is lacking to support behavior modification…but a reasonable start while further work-up initiated
MEDICAL THERAPY: BETA BLOCKERS

Mainstay of conventional medical therapy in symptomatic patients

Particularly useful and indicated in patients with heart failure or CAD

Beware the side effects…esp in young pts.

Fatigue, depressed mood, sexual dysfunction
MEDICAL THERAPY: CALCIUM-CHANNEL BLOCKERS

Commonly used in young patients without structural heart disease

Common Side Effect: Constipation

Avoid in patients with significant LV systolic dysfunction
Remember CAST!

Sotalol

Class III AAD

Beta blocker and Potassium Channel Blocker

Monitor QT +/- hospitalization

Amiodarone

"Class III AAD "

Diverse effects on various ion channels

QT prolongation is of less concern

Effective, but side effect concerns (short and long-term)

Thyroid, Liver, Lungs, Skin, Eyes
Patient sedated in fasting state

Multiple, steerable catheters are placed via femoral access sites

High burden of PVCs desirable to allow for precise mapping

Activation Mapping

Pace-mapping

Ablation is performed at sites with “earliest” activation and/or “perfect” pacemaps
Ablation most effective modality for PVC reduction...  

...but drugs aren’t terrible.

Ablation improves LV function... ...and is often curative!

~ Success rate 70-90 %
~ Complication rate 1-3 %
~ Duration 2-6 hrs

Zang M et al. Heart 2014; 100: 787
“He that is good with a Hammer tends to think everything is a nail.”

-- Maslow’s Maxim: Law of the Instrument
Ablation should be offered to patients with a substantial PVC burden (>20-25%) and diminishing EF for presumed PVC-induced cardiomyopathy. If asymptomatic with normal EF, it is reasonable to follow with annual TTE.

Ablation can be considered in patients with symptomatic PVCs that are refractory to CCB or BB.

PVCs that induce malignant arrhythmias (PMVT, TdP, VF) and are refractory to AAD, should be targeted with ablation.
PVCs are common…and are often benign

Assessing PVC severity and burden is essential to guiding management

Level of treatment aggression should be guided by symptoms and/or presence of LV dysfunction

Medical therapy is reasonably effective but side effects are common, especially in young pts.

Catheter ablation is effective and curative for most PVCs
COMMENTS/QUESTIONS