PVC’s / PAC’s
What Do They Mean?
What Should You Do?

Jeffrey H. Neuhauser, D.O.,F.A.C.C.
BHNI Primary Care Care Symposium
February 27, 2015
Financial disclosures

Paid speaker for Pfizer
Learning Objectives

• Understand the pathophysiology of PVC’s / PAC’s

• Understand the role for appropriate diagnostic testing

• Differentiate the low risk from the high risk pt

• Understand management options
PVC’s - ECG

- QRS duration >120 ms
- Bizarre morphology that does not resemble typical RBBB/LBBB pattern
- T wave in the opposite direction of the QRS vector
- A fully compensatory pause
PVC’s - Mechanisms

- Reentry - previous MI or myocardial fibrosis

*Figure 16B-19A.* Theoretical illustration of a reentry circuit. The three necessary components are: (1) two separate pathways (labeled x and y); (2) unidirectional block in one of these pathways (represented by the lined area in pathway y); and (3) slow conduction along the other pathway.
PVC’s - Mechanisms

- Abnormal Automaticity - electrolyte imbalance, acute ischemia. Results from lowering diastolic transmembrane voltage.
PVC’s - Mechanisms

- Triggered Activity - digoxin toxicity, hypokalemia, cardiomyopathy, ischemia, infarction. Due to after depolarizations which reach membrane threshold potential.
PVC’s - Prevalence

- 1% of routine ECG’s of 30-60 sec duration
- 80% incidence on 24 hr holter monitors of healthy pts
- More frequent in males & African Americans
- Prevalence increases with age, metabolic abnormalities, & organic heart disease
PVC’s in Apparently Healthy Pts

• Atherosclerosis Risk in Communities (ARIC) study of subjects with no known heart disease followed for 10 yrs

• PVC’ were associated with a 2x increase in the incidence of CAD & SCD

AJC 2006; 98(12):1609
AJC 2011; 107(2):151-55
Figure 1. Kaplan-Meier survival curves for subjects with (top curve) versus those without (bottom curve) ventricular premature complexes.
PVC’s in Apparently Healthy Pts

- Study of >45K veterans
- 3.8% incidence of PVC’s on ECG
- PVC’s associated with increased all cause mortality (39% vs 22%) & increased CV mortality (20% vs 8%)

Ann Noninvasive Electro 2007; 12(2):121
PVC’s in Apparently Healthy Pts

- Meta analysis of 8 observational studies involving >106K pts
- PVC’s were associated with a 2.1x increase in cardiac mortality & 2.6x increase in SCD
- Pts were not screened for underlying heart disease

AJC 2013 Oct;112(8):1263-70
PVC’s - H&P

- Few or no symptoms in most pts
- Palpitations or dizziness are the most common symptoms
- Irregular pulse on exam
- Variable intensity of S1 & cannon A waves
- Ask about syncope, near syncope, family history of early unexpected death, seizure disorder, or drowning
- Must differentiate the high risk from the low risk pt!
PVC’s - Diagnostic Evaluation

• ECG
• Electrolytes, digoxin level
• 24 hr Holter monitor
• Echo
• Exercise Treadmill Testing +/- imaging
• Cardiac catheterization
• Electrophysiologic study
Identifying the High Risk Pt

- Frequent PVC’s (esp multifocal) & NSVT

- Decreased LV systolic function (LVEF \( \leq 35\% \))
  LVEF 35-40% is a gray zone

- Prior MI

- Ischemic burden

- LV hypertrophy with septal wall thickness \( \geq 3 \) cm

- Intraventricular conduction delay on ECG
PVC’s - Treatment Principles

- PVC’s should only be treated if they cause significant symptoms.
- The risk depends upon the presence of underlying structural heart disease.
- Suppressing PVC’s does not reduce mortality in high risk pts.
- Beta blockers often 1st line therapy.
- If pt has a structurally normal heart, the risk for SCD is low & catheter ablation can be considered for significant symptoms.
- Sotalol, dofetilide, & amiodarone can be used for continued symptoms in pts with CAD or CM.
- ICD’s are indicated for prevention of SCD in high risk pts.
Results of the Cardiac Arrhythmia Suppression Trial (CAST) in patients with ventricular premature beats after myocardial infarction. Patients receiving encainide or flecainide had, when compared to those receiving placebo, a significantly lower rate of avoiding a cardiac event (death or resuscitated cardiac arrest) (left panel, p = 0.001) and a lower overall survival (right panel, p = 0.0006). The cause of death was arrhythmia or cardiac arrest.

ICD Indications

• Secondary prevention - 1) pts with prior sustained VT/VF not associated with a prior MI or a reversible cause
  2) Spontaneous VT in pts with structural heart disease or channelopathy

• Primary Prevention - 1) >40days post MI with LVEF <=30%
  2) Cardiomyopathy, NYHA II/III, LVEF<=35%
  of at least 3 months duration
ICD Contraindications

- VT due to a completely reversible cause
- Pt with expected survival <1 yr
- Pts with significant psychiatric disorders
- Pts with refractory NYHA IV HF not candidates for transplantation
- Incessant VT/VF
- Idiopathic VT (no structural heart disease)
Identifying the High Risk Pt
“SHOCKED”

• Study of >45K pts that received an ICD for primary prevention of SCD
• Included ischemic & nonischemic CM pts
• Mean F/U 4 yrs
• Predictors of all cause mortality were identified

JACC 2012 Oct;60(17):1647-55
### Table 4: HRs and CIs for Abbreviated Model Covariates

<table>
<thead>
<tr>
<th>Covariate</th>
<th>HR</th>
<th>95% CI</th>
<th>p Value</th>
<th>Wald Chi-Square</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic kidney disease</td>
<td>2.33</td>
<td>2.20–2.47</td>
<td>&lt;0.0001</td>
<td>831.7</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td></td>
<td></td>
<td></td>
<td>465.9</td>
</tr>
<tr>
<td>&lt;75</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥75</td>
<td>1.70</td>
<td>1.62–1.79</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>1.70</td>
<td>1.61–1.80</td>
<td>&lt;0.0001</td>
<td>355.2</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.43</td>
<td>1.36–1.50</td>
<td>&lt;0.0001</td>
<td>190.6</td>
</tr>
<tr>
<td>NYHA functional class</td>
<td></td>
<td></td>
<td></td>
<td>149.3</td>
</tr>
<tr>
<td>I or II</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>1.35</td>
<td>1.29–1.42</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>LVEF</td>
<td></td>
<td></td>
<td></td>
<td>83.2</td>
</tr>
<tr>
<td>&gt;20%</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤20%</td>
<td>1.26</td>
<td>1.20–1.33</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>1.26</td>
<td>1.19–1.33</td>
<td>&lt;0.0001</td>
<td>69.9</td>
</tr>
</tbody>
</table>

JACC 2012 Oct 60(17):1647-55
Identifying the High Risk Pt “SHOCKED”

- Seventy five yrs age - 1.7x
- Heart failure - NYHA III - 1.35x
- Out of rhythm from AF - 1.26x
- COPD - 1.7x
- Kidney disease, chronic - 2.33x
- EF - <=20% - 1.26x
- DM - 1.43x

JACC 2012 Oct;60(17):1647-55
PVC Pt #1

- 55 yr old male with palpitations & near syncope
- H/O MI 5 yrs ago with PCI of the LAD
- HPTN, hypercholesterolemia, tob use
- PE: P 64  BP 140/90 mmHg
  Regular rhythm, no murmurs; lungs clear; no edema
- Meds: ASA, metoprolol, simvastatin
PVC Pt #1

- 24 hr Holter showed 3200 PVC’s, 3 episodes NSVT
- ECHO: LVEF 40%; anteroseptal hypokinesis
- Cardiac cath: patent LAD stent; minimal other CAD
PVC Pt #1

- EP study was performed
PVC Pt #2

• 23 yr old male with palpitations. No syncope or near syncope.
• No PMH
• No medications
• No significant family history
• Consumes 3 energy drinks per day
• Normal physical exam
• Normal ECG
• 24 hr Holter shows 550 PVC’s
• Echo - normal
• ETT - exercised 14 min no increase in PVC’s; no VT
PVC’s in Pts With Structurally Normal Hearts

- Usually occurs in young to middle age pts
- Catecholamine sensitive
- Frequently exacerbated by exercise
- Monomorphics, LBBB/RA on ECG
- Originate in RVOT (occasionally LVOT)
- Can be highly symptomatic, but rarely result in SCD
- Respond to beta blockers & Ca channel blockers
- Can be cured by catheter ablation
PVC’s in Pts with a Structurally Normal Heart
PVC Pt #3

• 40 yr old female presents with progressive dyspnea, fatigue, & palpitations. The pt used to run, but is now currently unable to exercise.
• No PMH
• No significant family history
• No medications
• PE: P 96 BP 100/60 mmHg
  Irregular rhythm
  Lungs clear
  No edema
PVC Pt #3

- 24 hr Holter showed 27K PVC’s
- Cardiac cath showed no CAD
PVC Pt #3
PVC Pt #3

- EP study confirmed RVOT location; No inducible VT
- Successful catheter ablation performed
PVC Induced Cardiomyopathy

- Frequent PVC’s have been associated with reversible cardiomyopathy
- PVC burden >24% on 24 hr Holter
- PVC’s >20K on 24 hr Holter
- QRS duration (>160 ms) of the PVC’s may be associated with development of CM
PVC Induced Cardiomyopathy

- Catheter ablation of idiopathic PVC’s (RVOT/LVOT) reported to have >80% success
- LVEF normalized within 4 months in most
- Epicardial origin of PVC’s & greater QRS duration of PVC’s associated with delayed recovery of LVF

Heart Rhythm 2013; 10(2):172
Figure 2  EFs before and after catheter ablation of frequent PVCs in patients with a successful outcome. The mean EFs and standard deviations are indicated.
PVC Pt #4

- 20 yr old male with palpitations & near syncope. He is on the college wrestling team.
- No PMH
- No medications
- Father drowned. First cousin diagnosed with a seizure disorder.
PVC Pt #4

- PE P 60 BP 110/70 mmHg
  - Regular rhythm. Grade 2/6 systolic murmur LLSB
  - Lungs clear bilat.
  - No edema
PVC Pt #4

- Holter monitor 3K PVC’s, 2 episodes NSVT
- Cardiac catheterization showed normal coronary anatomy & LVEF 55%
- Echo showed dilated RA/RV
PVC Pt #4
PVC Pt #4
Arrhythmogenic RV Dysplasia

- Genetically inherited cardiomyopathy involving predominantly the RV
- Fibrofatty replacement of the RV myocardium initially producing wall motion abnormalities & progressing to RV dilatation
- Both autosomal dominant & recessive inheritance
- Prevalence 1:1000 - 1:2000
- Important cause of SCD in young adults
Arrhythmogenic RV Dysplasia

- ECG - Incomplete RBBB, TWI V1-V3, prolonged S wave V1-V3, Epsilon wave V1
- Echo - dilated RV with reduced RV systolic function
- Cardiac MRI - dilated RV, RV wall motion abnormalities, RVEF <40%
- Electrophysiologic study may be helpful in risk stratification
- Pts should not participate in competitive sports
- Beta blockers often recommended; no controlled data
- ICD may be indicated for high risk pts
PVC’s in Athletes

- Not necessarily associated with increased risk for SCD
- Prognostic importance depends on the presence of underlying structural heart disease
- Exclusion of underlying heart disease is essential
PVC’s in Athletes Evaluation

- ECG
- 24 hr Holter
- ETT
- Echo
- Cardiac MRI
- Cardiac catheterization in selected athletes with abnormal echo/MRI
PVC’s in Athletes
Recommendations

• Athletes without structural heart disease can participate in all sports

• If the athlete has PVC’s that increase with exercise to the point of causing dyspnea, fatigue, or impaired consciousness, then they can participate in class IA sports only

• Athletes with structural heart disease can participate only in class 1A sports

JACC 2005;45(8):1354
# PVC’s in Athletes

## Classification of Sports

<table>
<thead>
<tr>
<th>I. Low (&lt;20 percent MVC)</th>
<th>II. Moderate (20 to 50 percent MVC)</th>
<th>III. High (&gt;50 percent MVC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bobsledding/luge**</td>
<td>Body building**</td>
<td>Boxing*</td>
</tr>
<tr>
<td>Field events (throwing)</td>
<td>Downhill skiing**</td>
<td>Canoeing/Kayaking</td>
</tr>
<tr>
<td>Gymnastics**</td>
<td>Skateboarding**</td>
<td>Cycling**</td>
</tr>
<tr>
<td>Martial arts*</td>
<td>Snowboarding**</td>
<td>Decathlon</td>
</tr>
<tr>
<td>Sailing</td>
<td>Wrestling*</td>
<td>Rowing</td>
</tr>
<tr>
<td>Sport climbing</td>
<td></td>
<td>Speed-skating**</td>
</tr>
<tr>
<td>Water skiing**</td>
<td></td>
<td>Triathlon**</td>
</tr>
<tr>
<td>Weight lifting**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Windsurfing**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Archery</td>
<td>American football*</td>
<td>Basketball*</td>
</tr>
<tr>
<td>Auto racing**</td>
<td>Field events (jumping)</td>
<td>Ice hockey*</td>
</tr>
<tr>
<td>Diving**</td>
<td>Figure skating*</td>
<td>Cross-country skiing*</td>
</tr>
<tr>
<td>Equestrian**</td>
<td>Rodeoing**</td>
<td>(skiing technique)</td>
</tr>
<tr>
<td>Motorcycling**</td>
<td>Rugby*</td>
<td>Lacrosse*</td>
</tr>
<tr>
<td></td>
<td>Running (sprint)</td>
<td>Running (middle distance)</td>
</tr>
<tr>
<td></td>
<td>Surfing**</td>
<td>Swimming</td>
</tr>
<tr>
<td></td>
<td>Synchronized swimming*</td>
<td>Team handball</td>
</tr>
<tr>
<td>Billiards</td>
<td>Baseball/softball*</td>
<td></td>
</tr>
<tr>
<td>Bowling</td>
<td>Fencing</td>
<td></td>
</tr>
<tr>
<td>Cricket</td>
<td>Table tennis</td>
<td></td>
</tr>
<tr>
<td>Curling</td>
<td>Volleyball</td>
<td></td>
</tr>
<tr>
<td>Golf</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Riflery</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Increasing static component**

**Increasing dynamic component**
PVC Pt #5

- 75 yr old female with palpitations, nausea, & weakness
- PMH: PAF, HPTN, NIDDM
- Meds: Lisinopril, digoxin, glipizide
- PE: P 64  BP 104/68 mmHg
- Labs: K 3.2, creat 1.8, dig 3.3
Digoxin toxicity

- Symptoms include nausea, vomiting, anorexia, abdominal pain, confusion
- Serum dig level >2 ng/ml, however, toxicity can occur with levels in the “normal” range
- Hypokalemia, hypomagnesemia, hypercalcemia can predispose to toxicity
- PVC’s are the most common arrhythmia & often seen early in dig toxicity
- PVC’s usually occur in a bigeminal pattern
Digoxin Toxicity Management

- Admit pt for cardiac monitoring
- If no evidence of renal failure, electrolyte abnormality, or severe arrhythmia, pts can be safely discharged after at least 6 hours of monitoring
- Correct electrolyte abnormalities - hypo or hyperkalemia
- Fluid resuscitation if evidence of renal failure
- Digoxin specific antibody fragments for life threatening arrhythmias
PVC Pt #6

• 28 yr old pregnant female in her second trimester presenting with palpitations. No syncope or CP.
• No significant PMH
• No significant family history
• Meds: Prenatal vitamins
PVC Pt #6

- **PE:** P 76  BP 108/70
  - Irregular rhythm. No murmurs or gallop
  - Lungs clear
  - Trace edema
- Normal resting ECG
- Holter monitor 320 unifocal PVC’s
- Echo - normal
PVC’s in Pregnancy

• Study of 110 symptomatic & 52 asymptomatic pregnant women

• 40% incidence in asymptomatic women

• 49% incidence in symptomatic women

AM J Cardiology 1997;79(8);1061
PVC’s in Pregnancy Evaluation

- ECG
- Electrolytes
- 24 hr Holter
- Echo
PVC’s in Pregnancy Management

• Usually no therapy is needed

• Correct electrolyte abnormalities

• Discontinue smoking, caffeine, stimulants

• If symptoms are intolerable, metoprolol can be used
PAC’s

- P waves that occur earlier than expected in the cardiac cycle
- Often have a different morphology than the sinus P wave & different PR interval
PAC’s

- Occurs commonly in young, elderly, pts with & without structural heart disease
- Increased incidence in pts with mitral valve disease & LV dysfunction
- Can be exacerbated by smoking, alcohol, theophylline, & caffeine
- The mechanisms - same as PVC’s
  - Reentry
  - Abnormal automaticity
  - Triggered activity
PAC’s

- Usually produce few or no symptoms
- Palpitations are the most common symptom
- Rarely cause hemodynamic compromise
- Irregular pulse on exam; sometimes can cause cannon A waves
- ECG obtained for evaluation
- 24 hr Holter can also be used
- Do not need tx
- For severe symptoms beta blockers, class IC & class III antiarrhythmic drugs can be used
Summary

- PAC’s occur commonly
- Palpitations are the most common symptom
- PAC’s may be associated with structural heart disease but their presence alone does not signify increased risk
- PAC’s should not be treated unless the pt is severely symptomatic
- Try to identify underlying disease processes & precipitating factors
Summary

- PVC’s can be markers for increased risk of CV events & mortality
- PVC’s can also occur in otherwise healthy pts
- It is crucial to differentiate the low risk from the high risk pt
- Beta blockers & class III AAD’s can be used when it becomes necessary to suppress symptoms
- An ICD should be considered for the high risk pt
- Frequent PVC’s can cause cardiomyopathy
- Catheter ablation should be considered for symptomatic PVC’s in pts with structurally normal hearts.