Premature Ventricular Contractions

Ralph Augustini, MD FACC FHRS
Premature Ventricular Contractions:

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death

Background

- PVCs are ectopic impulses originating from an area distal to the His Purkinje system
- Most common ventricular arrhythmia
- Significance of PVCs is interpreted in the context of the underlying cardiac condition
- Ventricular ectopy leading to ventricular tachycardia (VT), which, in turn, can degenerate into ventricular fibrillation, is one of the common mechanisms for sudden cardiac death
- The treatment paradigm in the 1970s and 1980s was to eliminate PVCs in patients after myocardial infarction (MI).
- CAST and other studies demonstrated that eliminating PVCs with available anti-arrhythmic drugs increases the risk of death to patients without providing any measurable benefit
Pathophysiology

Three common mechanisms exist for PVCs, (1) automaticity, (2) reentry, and (3) triggered activity:

- **Automaticity:** The development of a new site of depolarization in non-nodal ventricular tissue.

- **Reentry circuit:** Reentry typically occurs when slow-conducting tissue (eg, post-infarction myocardium) is present adjacent to normal tissue.

- **Triggered activity:** Afterdepolarization can occur either during (early) or after (late) completion of repolarization.

- Early afterdepolarizations commonly are responsible for bradycardia associated PVCs, but also with ischemia and electrolyte disturbance.
Triggered

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Epidemiology

- Frequency
  - The Framingham heart study (with 1-h ambulatory ECG)
    - 1 or more PVCs per hour was 33% in men without coronary artery disease (CAD) and 32% in women without CAD
    - Among patients with CAD, the prevalence rate of 1 or more PVCs was 58% in men and 49% in women.
    - 24-hour ambulatory monitoring showed a VPC prevalence rate of 41% in healthy teenage boys aged 14-16 years, 50-60% in healthy young adults, and 84% in healthy elderly persons aged 73-82 years.
  - PVCs also are common in patients with hypertension, ventricular hypertrophy, cardiomyopathy, and mitral valve prolapse.
  - Data from the GISSI-2 study
    - 64% of patients with prior MI had ventricular arrhythmia
    - 20% of patients had more than 10 PVCs per hour with 24h holter
Mortality/Morbidity

- **Prognosis** depends on the frequency and characteristics of PVCs and on the type and severity of associated structural heart disease.

  - PVCs are associated with increased risk of death, especially with CAD
  - The relationship between VPC frequency and mortality is not robust and no benefit results in suppressing PVCs to improve survival in any population
  - In asymptomatic patients, frequent ventricular ectopy was associated with 2.5-fold increased risk of cardiovascular death
  - Multiform PVCs confer a poorer prognosis than uniform PVCs
  - Post-MI, frequent PVCs (>10/h) are associated with increased mortality in the pre-thrombolytic era, but the association in patients receiving thrombolytic is weak.
Mortality/Morbidity

- In 2 studies, a frequent VPC during any given stage of stress testing, bigeminy, trigeminy, couplets, triplets, sustained or non-sustained ventricular tachycardia, ventricular flutter, torsade de pointes, or ventricular fibrillation was an independent predictor of death.

- Another study, frequent PVCs only during exercise did not independently predict an increased risk; Instead frequent PVCs during recovery was a stronger predictor of death.

- Frequent PVCs, especially when they occur in a bigeminal pattern, can precipitate tachycardia-induced cardiomyopathy that can be reversed by elimination of the PVCs through catheter ablation.

- In some circumstances, very frequent PVCs may decrease cardiac systolic function, and suppression by ablation may have a beneficial effect.
Clinical Presentation

**Palpitations:** due to an augmented post-VPC beat and may be sensed as a pause rather than an extra beat

- Lightheadedness
- Fatigue
- Sustained tachycardia is not uncommon
- True syncope is infrequently seen
Physical Examination

- Variable or decreased intensity of heart sounds.
- The augmented beat following a dropped beat (pause) heard frequently.
- Bounding jugular pulse (cannon a wave) from a loss of AV synchrony may be present.
- The follow-up beat after a VPC is stronger due to the post-extra systolic compensatory pause, allowing greater left ventricular (LV) filling, causing greater intensity of that beat.
- Conversely, the VPC itself may be underperfused and consequently not perceived by radial pulse, resulting in a spurious documentation of bradycardia.
Cardiac Causes

- Acute myocardial infarction
- Valvular heart disease, especially mitral valve prolapse
- Cardiomyopathy (ischemic, dilated, hypertrophic, infiltrative)
- Myocardial stretch
- Cardiac contusion
- Bradycardia
- Tachycardia (high-catecholamine state)
Non-cardiac Causes

- Electrolyte disturbances (hypokalemia, hypomagnesemia, or hypercalcemia)
- Medications (eg, digoxin, tricyclic antidepressants, aminophylline, amitriptyline, pseudoephedrine, fluoxetine)
- Other drugs (eg, cocaine, amphetamines, caffeine, alcohol)
- Anesthetics
- Surgery
- Infection
- Stress
Laboratory Studies

- Look for correctable causes of PVCs:
  - Medications
  - electrolyte disturbances
  - infection
  - myocardial ischemia or MI

- Check serum electrolyte and magnesium levels
Imaging Studies

- Look for underlying structural heart abnormalities that can predispose to PVCs
- Assess the degree of LV dysfunction with echo radionuclide imaging
- Echo preferable because it also provides structural information about the heart
Other Tests

- **24-hour Holter monitor**
  - Severity of LV dysfunction, along with the complexity and frequency of the PVCs
  - Suppression of PVCs by beta-blocker or calcium blocker, together with a typical LBBB inferior axis, morphology helps to establishing typical RVOT ectopy.
  - Suppressing all PVCs themselves is not the focus of treatment

- **ECG** performed to look for structural cardiac abnormalities
  - Wide beats
  - No preceding premature P waves occur
  - The T wave usually is in the opposite direction from the R wave.
  - Compensatory pause is common.
  - PVCs originating from the left ventricle typically RBBB pattern.
  - PVCs originating from right ventricle typically LBBB pattern.

- **Exercise stress testing**
  - coronary ischemia, exercise-induced arrhythmia
Electrophysiology Study

- Indicated for 2 types of patients with PVCs:
  
  (1) those with a structurally normal heart with symptomatic PVCs, for whom pharmacological treatment or catheter ablation is indicated.
  
  (2) those with PVCs and structural heart disease, for whom risk stratification for sudden cardiac death is indicated.

- According to current ACC/AHA guidelines:
  
  class I indications for EPS are patients with CAD, low EF (< 0.36), and NSVT on ambulatory ECG.
  
  Class II indications for catheter ablation apply to patients with a highly symptomatic uniform PVCs, couplets, and NSVT.
Classification

- Classification according to frequency:
  - Frequent - 10 or more PVCs per hour (by Holter monitoring) or 6 or more per minute
  - Occasional - Fewer than 10 PVCs per hour or fewer than 6 per minute

- Classification according to relationship to normal beats:
  - Bigeminy - Paired complexes, VPC alternating with a normal beat
  - Trigeminy - VPC occurring every third beat (2 sinus beats followed by VPC)
  - Quadrigeminy - VPC occurring every fourth beat (VPC following 3 normal beats)

- Couplet - 2 consecutive PVCs
- NSVT - 3 or more consecutive PVCs (< 30 s)
Common PVC Locations

- RV Inflow – Anterior TA
- RVOT – Free Wall, Septal AoV Cusps
- Endocardial LVOT
- Epicardial LVOT
- Aorto-Mitral Continuity
- Superior Mitral Annulus
Outflow Tract Embryology – Neural Crest Cell Migration
Treatment

- **Absence of structural heart disease**
  - Asymptomatic = require no therapy.
  - Symptomatic PVCs = patient education and reassurance, avoidance of aggravating factors, and anxiolytic drugs if needed
  - Beta-blockers and non-dihydropyridine calcium channel blockers
  - Anti-arrhythmic therapy is only used to prevent symptoms.

- **Presence of underlying heart disease**
  - The presence of 2 or more of the following variables, (1) LV EF less than 0.40, (2) ventricular late potentials (on signal-averaged ECG), and (3) repetitive PVCs.
  - Treatment of transient ischemia.
  - Optimal treatment for congestive heart failure (CHF), CAD, or both should be instituted.
  - Maintain electrolyte balance.
  - Blood pressure control.
Treatment

- The 2006 ACC/AHA/ESC guideline recommends that ablation therapy should be considered in the following:
  - Patients with frequent, symptomatic, and monomorphic PVCs refractory to medical therapy
  - Patients who choose to avoid long-term medical therapy
  - Patients with ventricular arrhythmia storm that is consistently provoked by PVCs of a similar morphology
Treatment

- Patients deemed to be at high risk of sudden cardiac death may benefit from implantable cardioverter defibrillator (ICD) implantation
51 Year Old Female with Long History of PVC’s
Presented with Palpitations/Near Syncope
Echo/Cardiolite/Cardiac MRI – Negative
51 Year Old Female with Palpitation/Near Syncope
NSVT During Isuprel Infusion
51 Year Old Female – RVOT VT

Pacemap Vs. Spontaneous VT
51 Year Old Female – RVOT VT – Carto Map

RF Sites

- LAT 2-Map PVC 2 > 42 Points
- LAT 2-Map PVC 2 > 42 Points
- RAO
- AP
- 1.18 cm
- 1.18 cm

Medical Center
Benign PVCs? RFA of Frequent, idiopathic PVCs:

60 pts with PVCs referred for RFA. 22 with decreased EF
Mean PVC Burden: 21± 17%
PVC location: RVOT 31 (52%), LVOT 9 (15%)
Other 20 (33%)
RFA Successful in 48 (80%)
LVEF normalized in 18 / 22 (82%) with baseline LV dysfunction

Conclusions: LV dysfunction in the setting of frequent PVCs may be a reversible with catheter ablation.

Bogun et al, Heart Rhythm 2007
51 year old male with a Hx of “Benign” PVCs
NIDCM Dx’d 2003 (EF = 20%). Meds = ACEI, Coreg, Aldactone
Holter: Uniform PVCs = 20% of QRS complexes (18,000)
Referred for ICD Implant
LAO

Pace Map of Distal CS / Proximal Aspect of Anterior CS Branch
AP
Pace Map of Distal CS / Proximal Aspect of Anterior CS Branch
Arterial Pressure Recordings During PVC’s
Arterial Pressure Recordings
Post RFA PVC’s
Follow-up 3 months following ablation: EF = 42%