Approach and Management of PVCs

Cardiology Topic Review

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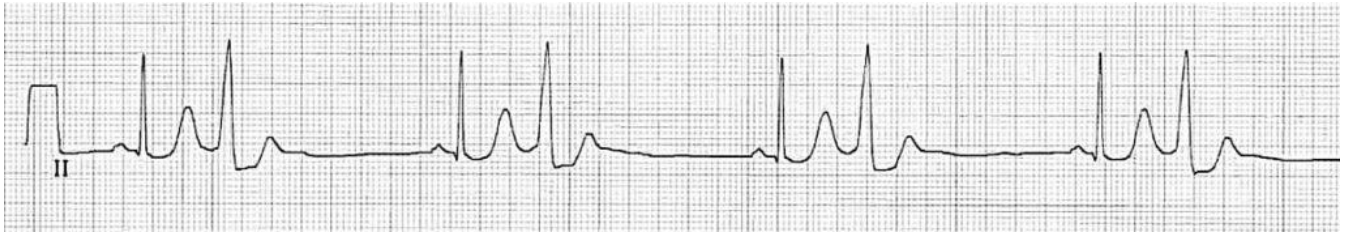
Background

- Premature ventricular contractions (PVCs) are extra heart beats that originate from the ventricles
- PVCs are common in the general population, found in up to 75% of individuals on Holter monitors
- Mechanisms for PVCs include
  - Increased automaticity
    - Accelerated generation of an action potential by either normal pacemaker cells (enhanced normal automaticity) or by abnormal tissue within the myocardium (abnormal automaticity)
  - Re-entry
    - Electrical impulse fails to terminate after activating the heart muscle and continues to propagate within a localized circuit
  - Triggered activity
    - Abnormal depolarizations following a regular action potential, caused by afterdepolarizations that reach the threshold and generate a new action potential



Ventricular Bigeminy

Clinical Presentation and Significance

- Symptoms vary widely and range from asymptomatic to patients experiencing:
  - Palpitations
    - Due to prolonged ventricular filling time after the PVC, resulting in an enhanced stroke volume
  - Fatigue
  - Dyspnea
  - Pre-syncope
- Symptoms can significantly impact daily activities and cause anxiety
- Physical exam may reveal an irregular pulse, but may be unremarkable
- PVCs may not generate enough ejection volume or pressure to allow the opening of the aortic valve leading to a concealed mechanical bradycardia
  - Cardiac rate on ECG may not reflect the true mechanical cardiac rate (ie, pulse)
- PVCs are most often benign, but can contribute to the development of a cardiomyopathy, particularly with a burden exceeding 10-15%

Diagnostic Approach

- Basic workup
  - History and physical exam to exclude heart disease, identify symptoms, common triggers, and exclude reversible contributors
  - Review family history of SCD
  - ECG to assess PVC morphology and suggestion of anatomical location (ie, RVOT vs. LVOT)
  - Holter monitor if significant symptoms or malignant family history to quantify real PVC burden, ideally >48 hours to 7 days
    - Substantial daily variation in PVC burden can occur and may require longer observation
  - ECHO to evaluate biventricular size, systolic function, and exclusion of major structural abnormalities
- Complementary tests
  - CMR for a more refined assessment of structural abnormalities and presence of scar
    - Useful in patients when the PVC is not arising from a common location (ie, RVOT) or when sustained VT is present
  - Coronary assessment (ie, functional or anatomic) if LVEF impairment or RWMA present

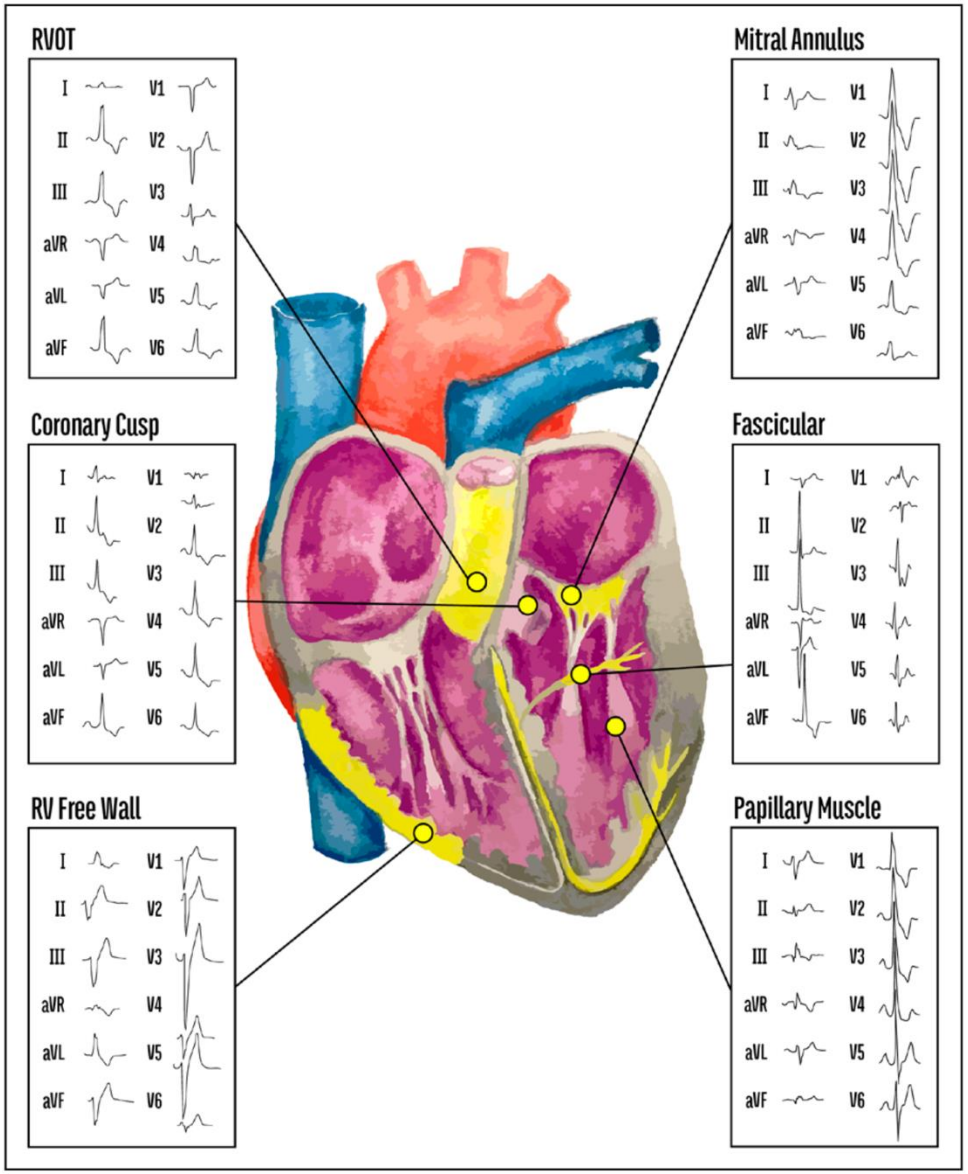
Triggers of PVCs

Triggers	Tests
Alcohol	Alcohol level
Caffeine (ie. coffee, tea)	
Recreational drugs	Urine and serum toxicology
Electrolyte derangements (ie. K and Mg)	Serum electrolytes
Hypoxia (ie. COPD, OSA)	Pulse oximetry, sleep study, ABG
Uncontrolled hypertension	Blood pressure measurement
Hyper/hypothyroidism	TSH
Digoxin toxicity	Digoxin level
CHF exacerbation	NT-proBNP
Anemia	CBC
Psychological stress/anxiety	
Menopausal transition	

PVC Anatomical Location

- A PVC rising from the outflow tract is most common
- Outflow tract PVCs characteristically exhibit a negative QRS complex in both aVL and aVR, consistent with a vector that is predominantly arising from the top of the heart, and by the same token the inferior leads will be positive
- Generally, a RVOT and LVOT PVC will exhibit a LBBB and RBBB morphology respectively
- The precordial transition on ECG is useful in PVC anatomic localization
- A PVC arising from the RVOT is often considered benign, but can still trigger VF particularly in patients with Brugada syndrome

Common Locations of PVCs



Risk Stratification and Prognosis

- CAST trial (1991) demonstrated that PVC suppression post MI led to excess morbidity and mortality
- Approach of suppressing PVCs to improve patient prognosis was abandoned
- Studies in early 2000s showed improvements in LVEF after successful catheter ablation of PVCs, and subsequent studies established that a higher burden of PVCs is associated with heart failure
  - PVC-induced cardiomyopathy occurs at burden generally >10%
- 2015 Cardiovascular Health study followed patients for 14 years and demonstrated that a greater frequency of PVCs was associated with a 5-year reduction in LVEF, an overall increased risk for clinically relevant heart failure, and an increased risk for death

Management Approach

- If the PVC burden is low (<10%) and basic workup reveals no relevant underlying condition, structural heart disease, and normal LVEF, reassurance may be reasonable and sufficient
- Key factors to consider when initiating management
    1. Symptoms
    2. PVC burden (%)
    3. Presence or absence of structural heart disease
- The optimal approach to asymptomatic patients with a high PVC burden but normal LVEF is unclear, but routine surveillance with annual in-person evaluation and ECHO is reasonable
- Pursue medical treatment in the following patients
    1. Patients with symptoms that interfere with their QoL despite receiving reassurance
    2. Patients with a reduced LVEF

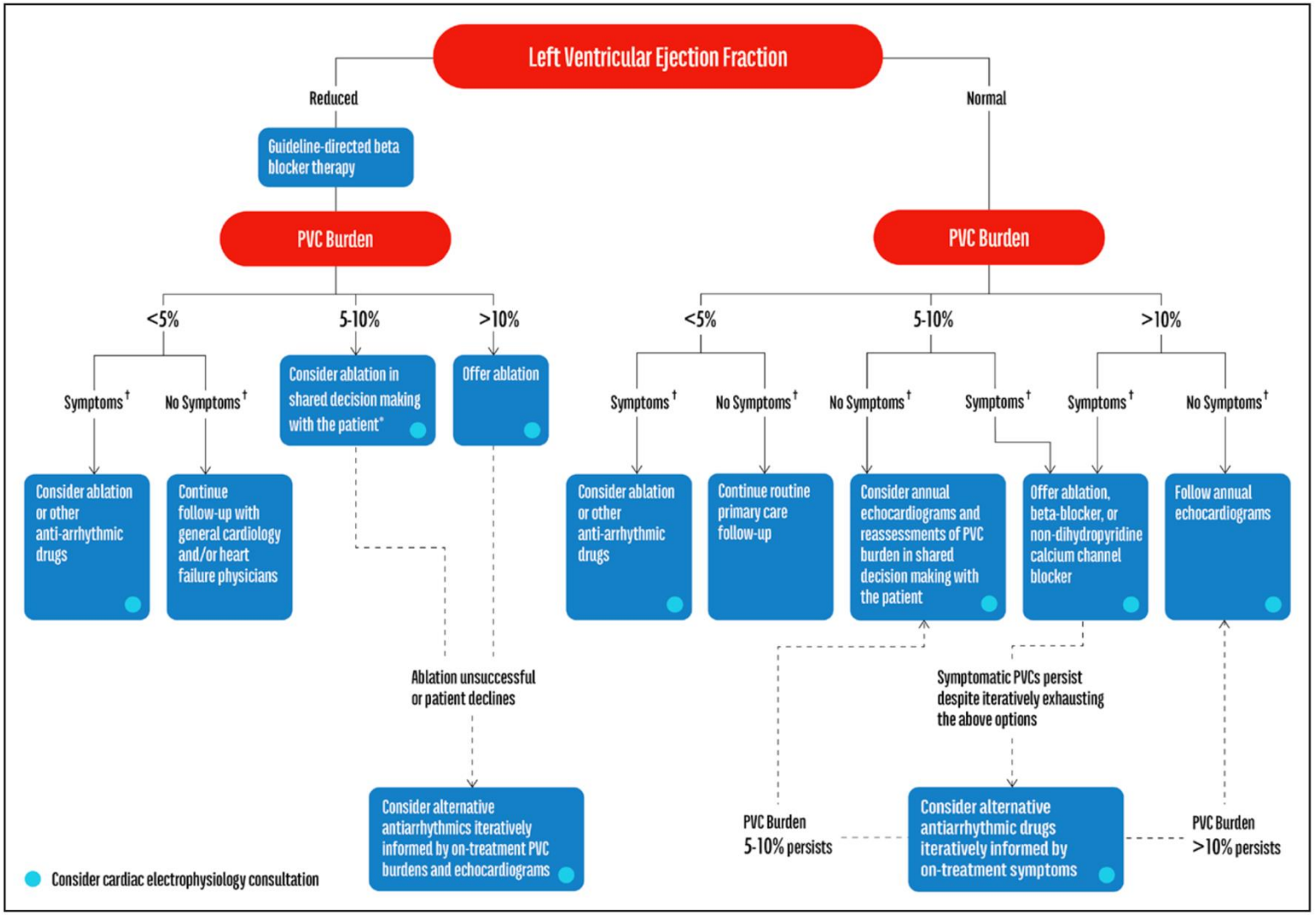
Management Strategy

- Patients need to be counselled on identification and avoidance of triggers
- Either medical treatment or catheter ablation are reasonable first line options
  - Catheter ablation exhibit superior effectiveness, but may have limited access and represent greater up-front risks
  - Either β-blockers or nondihydropyridine calcium channel blockers (ie, diltiazem or verapamil) are first-line medicine for PVCs
- Although better than placebo, RCTs have shown that β-blockers result in a clinically meaningful reduction in PVCs in only 12-24% of patients
- Reasonable to trial a CCB if a β-blocker fails (and vice versa)
- If initial drugs fail, catheter ablation should be considered next
- In patients who strongly prefer to avoid catheter ablation or are not candidates, additional antiarrhythmic drugs can be considered
  - Antiarrhythmic options include flecainide, propafenone, sotalol, and amiodarone
  - Need to consider risk profile, side effects, and contraindication to antiarrhythmics before prescribing

Catheter Ablation

- More efficacious than medicines to treat PVCs
- Class 1 indication to treat PVCs if medicine are not tolerated, not effective, or preferred by the patients
- Success of PVC catheter ablation procedures range from 80-95%
  - Higher success rate with RVOT origin PVC, compared to LVOT
  - Higher success rate with monomorphic PVC, compared to polymorphic
- Main issue with catheter ablation are limited access and complications which are observed in 0-5% of cases and mostly related to vascular assess

Sample PVC Approach and Management Workflow



Practical Considerations

- β-blockers result in a clinically meaningful reduction in PVCs in only 12-24% of patients
- Tang et al. (2021)
  - Enrolled patients with frequent PVCs (≥5 on ambulatory ECG) who had normal cardiac function and no structural heart disease
  - Patients divided into three groups based on treatment: β-blockers/CCBs, AADs, or conservative therapy
  - Primary outcome was the change in PVC burden
  - Showed that AADs showed superior effectiveness in reducing PVC burden compared to β-blockers/CCBs and conservative therapy, with a median relative reduction of 81.3% in the AAD group
  - Incidence of LV dysfunction over two years was low (3.3%), indicating that frequent idiopathic PVCs have a relatively benign course
  - Safety and tolerability were similar across groups
  - Study concluded that while β-blockers and CCBs are commonly used as first-line treatments for PVCs, their effectiveness is limited and comparable to conservative management.
  - Class I and III AADs offer greater effectiveness in reducing PVC burden

Case #1

- Clinical Presentation
  - A 34-year-old woman, 30 weeks pregnant, presents to the emergency department with palpitations.
  - ECG reveals frequent PVCs in sinus rhythm. Physical examination is normal, with no signs of structural heart disease. Her mother has a history of "extra beats," but no known cardiac disease. Family history is otherwise benign.
- Clinical Significance
  - Pregnancy can increase the cardiac output and volume, potentially exacerbating or unmasking arrhythmic symptoms such as PVCs.
  - The absence of structural heart disease and a benign family history are reassuring, yet the frequency of PVCs during pregnancy warrants close monitoring due to the potential for symptomatic burden and rare complications.
- Management
  - Initial Approach: Reassurance about the benign nature of PVCs in her context, coupled with monitoring to assess for any escalation in symptoms or change in PVC pattern.
  - Follow-Up: Recommend an echocardiogram to exclude structural abnormalities given her symptomatic presentation and to establish a baseline cardiac function.
  - Considerations: Beta-blockers, if pharmacological intervention becomes necessary, chosen carefully for fetal safety.

Case #2

- Clinical Presentation
  - A 67-year-old man with increasing shortness of breath on exertion, previously negative for cardiac disease.
  - ECG shows ventricular bigeminy; echocardiogram reveals global left ventricular (LV) dysfunction. Coronary angiogram is negative for obstructive disease, raising the question of PVC-induced cardiomyopathy
- Clinical Significance
  - This case highlights the potential of frequent PVCs to contribute to or cause cardiomyopathy, a reversible condition if the arrhythmic burden is appropriately managed.
  - The presence of frequent PVCs, especially in a pattern of ventricular bigeminy, in a patient without prior cardiac disease but with LV dysfunction, suggests a causal relationship.
- Management
  - Evaluation: Given the global LV dysfunction, further assessment to quantify PVC burden with a Holter monitor is essential.
  - Therapeutic Approach: Considering the potential for reversibility of cardiomyopathy, aggressive management of PVCs is indicated.
    - Pharmacological: β-blockers or AAD as initial therapy to reduce PVC burden.
    - Catheter Ablation: Strongly considered for potential curative treatment if pharmacotherapy is ineffective or not tolerated, especially given the likely direct impact of PVCs on his LV dysfunction.

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Thank you for listening!

Questions and Discussion