Ablation of PVC

When to Decide?

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True or False

- PVCs could be asymptomatic
- Short-coupled PVCs are more benign than long-coupled PVCs
- Ablation is indicated in most patients with PVCs
- Long-coupled PVCs could trigger VT/VF
- PVCs could induce cardiomyopathy

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- PVC, an overview
- PVC Origin
- PVC Mechanism
- Approach to a Patient with PVC
- When to Treat, When to Ablate
- Guidelines and PVC Ablation

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First reported PVCs?

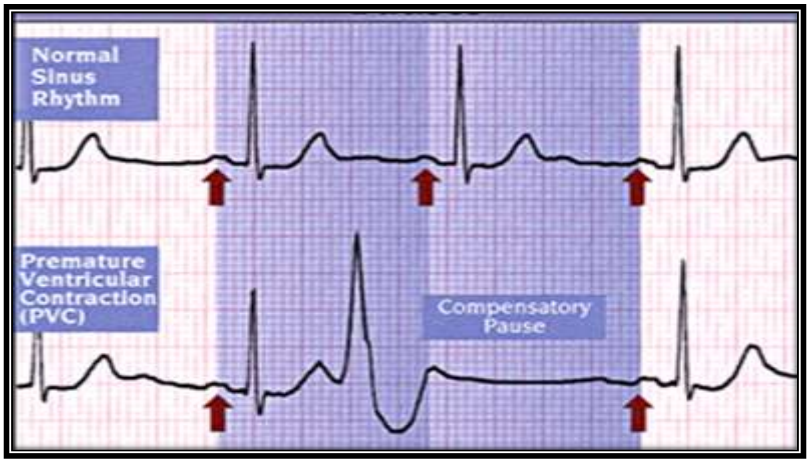
The first recorded description of intermittent perturbations interrupting the regular pulse, that could be consistent with PVCs, was from the early Chinese physician **Pien Ts'Io**, around **600 BC**, who was the master in pulse palpation and diagnosis

Definition

Premature ventricular complex (PVC) reflects activation of the ventricles from a site below the atrioventricular node (AVN).

ECG characteristics:

- QRS duration >120 msec.
- T wave is usually large& opposite polarity to QRS
- Not preceded by a P wave
- Followed by compensatory pause



Prevalence

- Depends on study population, the detection method, and the duration of observation.
  - No known heart disease:

✓ 0.5-1% of routine 12-lead ECG

✓ 30-40% in Holter ECG ambulatory monitoring

✓ There is an age-related increase in the prevalence of PVCs

  - Structural heart disease:

✓ 40-70% in HF and IHD, in 12-lead ECG

✓ Up to 80-90% in HF on Holter ECG

Who is at risk?

Male gender

Advanced age

African American race

Hypertension

Ischemic heart disease, and other structural HD

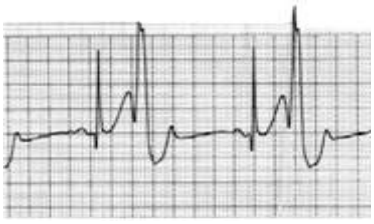
Bundle branch block on 12-lead ECG

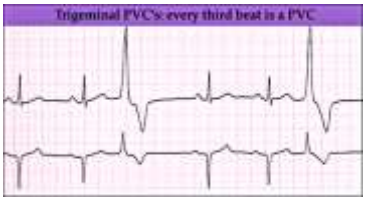
Hypomagnesemia

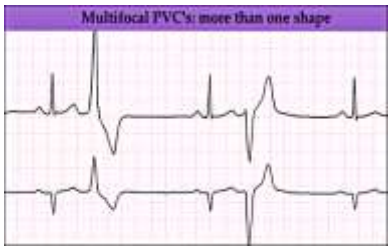
Hypokalemia

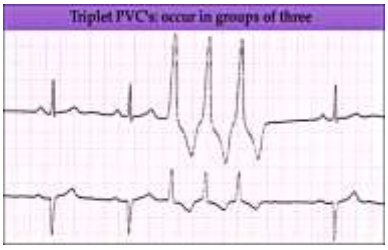
Clinical significance

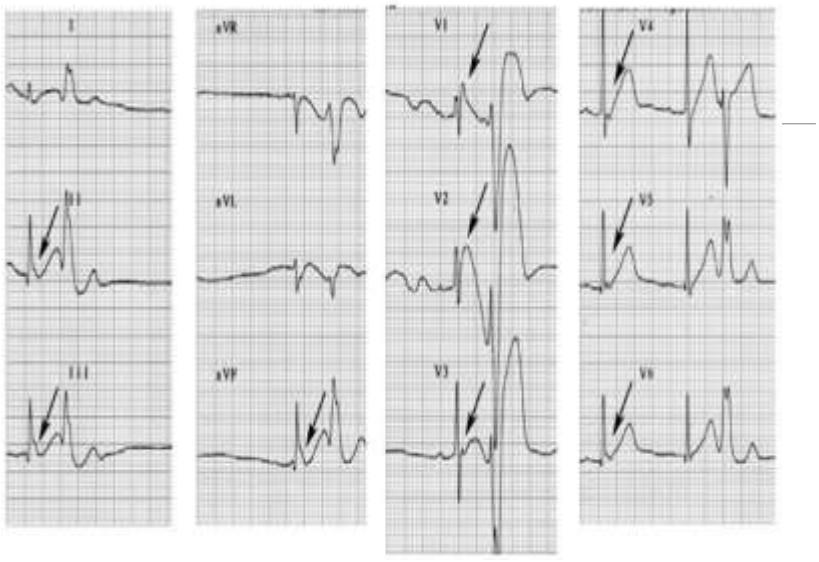
- Frequency
- Complexity
- Hemodynamic response
- Associated symptoms
- Structural heart disease

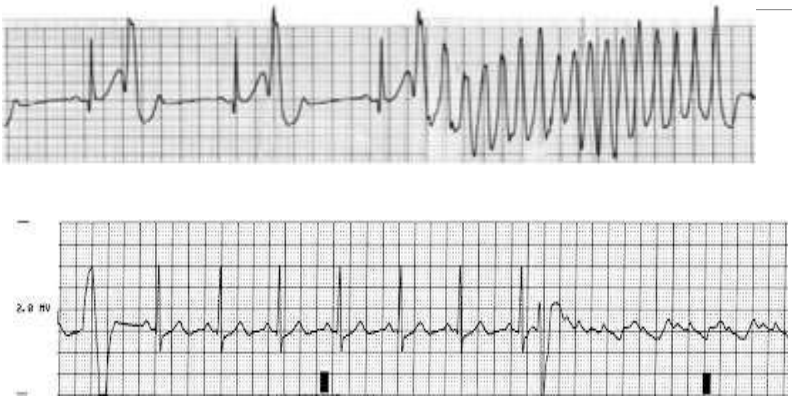






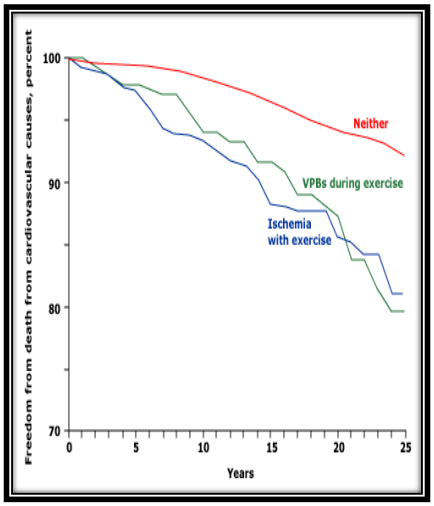



I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6


2.0 mV

Are PVCs in stress test dangerous?

- 6101 healthy men did EST & F/U for 23 years.
- Presence of PVCs during EST was associated with significant higher all-cause mortality (**41% vs. 26%** no VPBs) and CV death (**16% vs. 6.4%**).
- **Presence of PVCs prior to or during exercise was an independent predictor of CV mortality (relative risk 2.53) and had the same prognostic value as exercise induced ischemia (relative risk 2.63).**



Data from: Jouven X, Zureik M, Desnos M, et al. N Engl J Med 2000; 343:826.

PVC-Mediated Cardiomyopathy

- PVCs first ? or cardiomyopathy first?
- A high PVC burden (>24%) in patients with LV dysfunction and a rather short coupling interval of the PVCs (>300 ms) suggest PVC-induced cardiomyopathy
- **Pathogenesis**: Ventricular dyssynchrony, hemodynamic impairment, increased oxygen demand, autonomic dysregulation, and abnormal Ca handling
- **Predictors for development of PVC-mediated cardiomyopathy**:
  - Underlying cardiac structural abnormalities
  - Obesity
  - Absence of palpitations
  - PVCs characteristics (> 20%, LBBB, wide QRS, short/variable coupling intervals)

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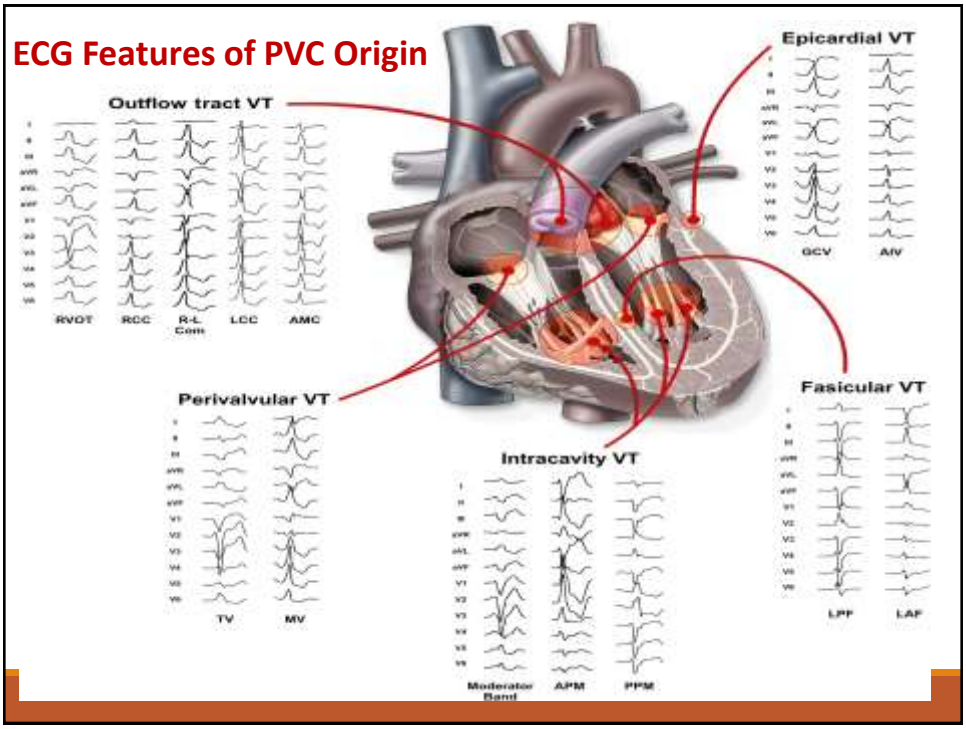
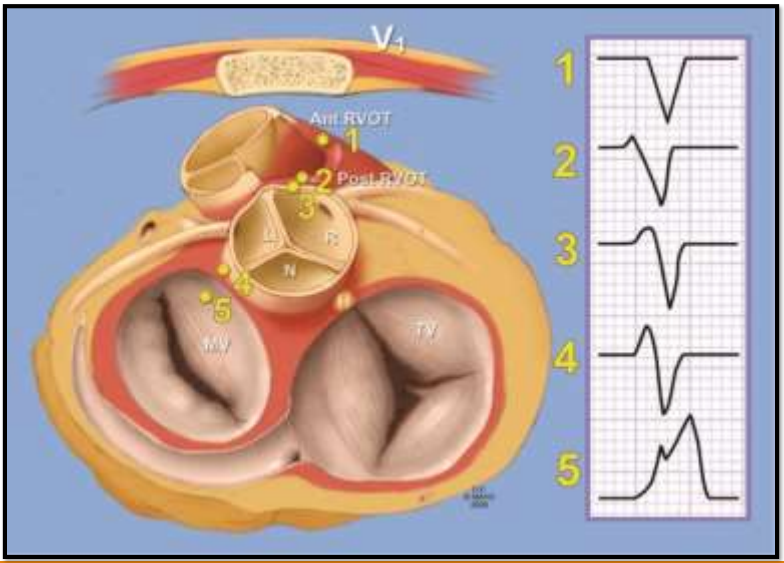
Premature Ventricular Complex (PVC)

- PVCs commonly arise from RVOT and sometimes LVOT.
- Other sites: His-Purkinje system, papillary muscles, moderator band, false tendons, and annuli of the aortic, pulmonary, and both AV valves.
- In structural heart disease, PVCs can originate from re-entry with unidirectional block and slow conduction through electrically viable tissue within areas of scar.

ECG Features of PVC Origin



Summary of ECG features suggesting PVC origin



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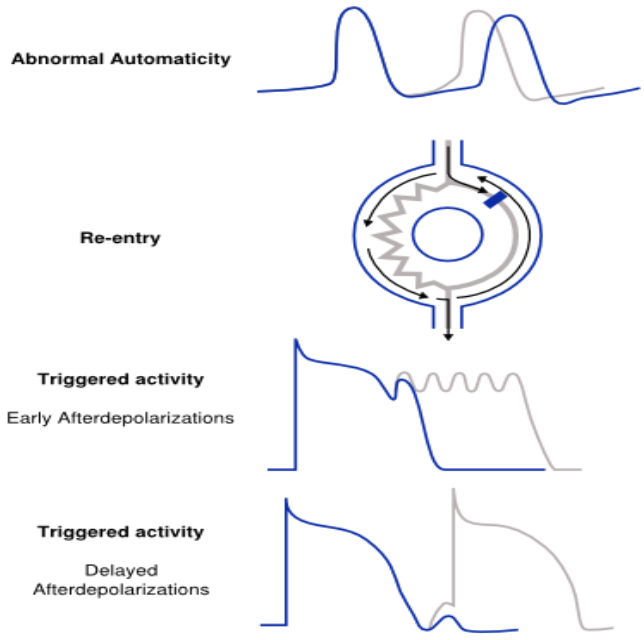


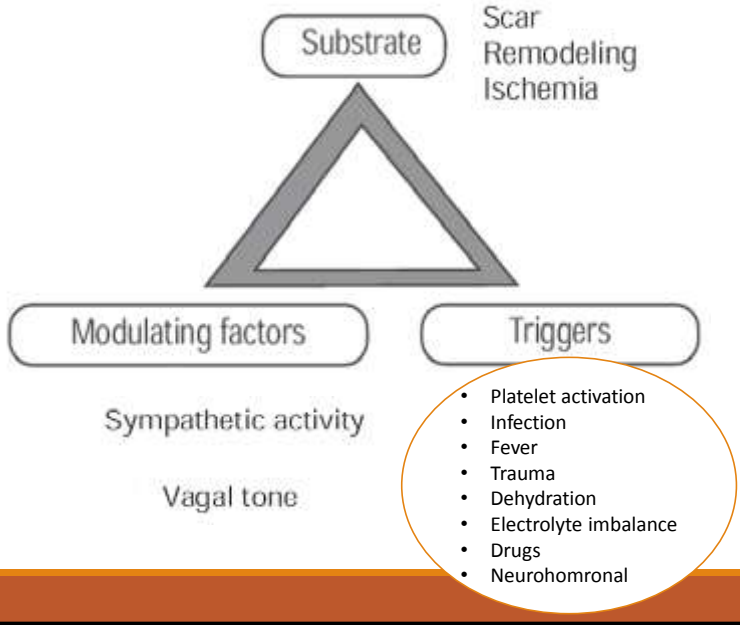
PVC Mechanisms

- **RVOT/LVOT**: PVCs originate from embryonic remnant conduction tissue.
- **Delayed after depolarizations**: RVOT PVCs provoked by catecholamines, exercise, menstrual cycle, and inhibited by adenosine. Channelopathies, CPMVT, and digoxin toxicity.
- **Triggered automaticity**: RVOT PVCs provoked by catecholamines, exercise, and menstrual cycles, and inhibited by adenosine.
- **Reentrant PVCs**: extensions of cardiomyocytes above the fibrous valvular annuli could allow conduction slowing and unidirectional block.

PVC Mechanisms

- **Early after depolarizations:** PVCs that provokes TdP in LQT syndrome.
- **Reentry**: PVCs from fibrosis, infiltration or scars (cardiomyopathy, IHD, ARVD, sarcoidosis, HCM, valvular cardiomyopathy), Cong. HD, muscular dystrophies, and metabolic disorders (mitochondrial diseases).
- **Fascicular PVCs:** small reentry circuits involving the fascicles or due to triggered or enhanced automaticity.

Recipe for Arrhythmia



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PVC: Symptomatic Arrhythmia and Cardiac Arrest

- PVCs could be infrequent and asymptomatic in most cases
- In some cases: frequent& symptomatic (palpitations, chest pain, dyspnea)
- Short-coupled PVCs can trigger VT, polymorphic VT, or VF
- PVCs in bi-leaflet MVP could initiate VT/VF
- PVCs could trigger re-entrant VT in patients with structural heart disease

Assessment of a Patient with PVC

History & Exam.

- Prior diagnosis/RFs of SHD?
- Symptoms of SHD?
- Non-cardiac disorders that may affect the heart?
- Syncope?
- Family Hx SCD? SHD?
- Evidence for SHD?

ECG

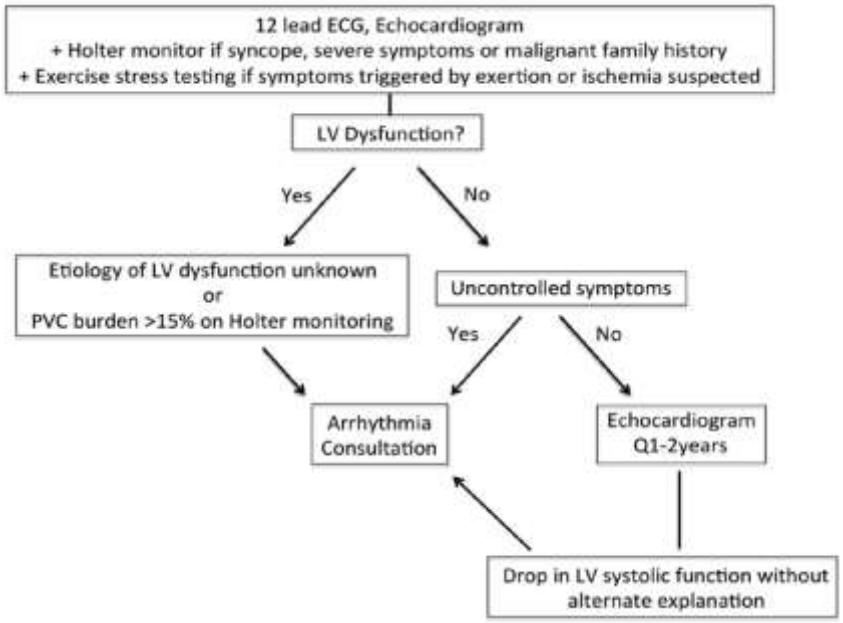
- PVCs (site, frequency, CI, morphology)
- QT duration
- Qs (post MI)
- LVH/RVH

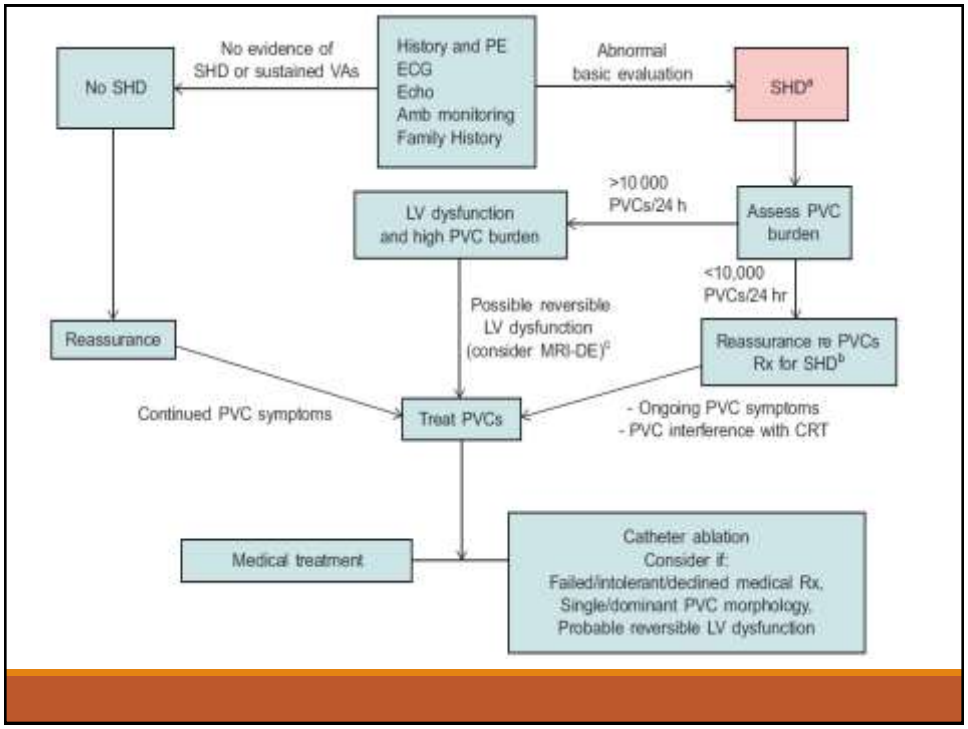
Echocardiogram

- LVH/RVH
- LV/RV size & function
- Ischemia/SWMA
- Scar/Infiltrative
- Valvular diseases
- PAP

Additional Testing …..

- **Holter Monitoring**: identify the various morphologies of PVCs, burden, diurnal variations, and coupling intervals, though these can vary on a day-to-day basis.
- **Exercise Testing**: provoke "triggered" RVOT or fascicular PVCs and papillary muscle PVCs in MV prolapse. It might suppress long-coupled re-entrant PVCs as the sinus rate accelerates.
- **Coronary Angiography**
- **Cardiac Magnetic Resonance Imaging**





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Treatment Indications of PVCs without Structural Heart Disease

- Asymptomatic & infrequent PVCs: assurance.
- Treatment is dedicated for:
  - Symptomatic patients.
  - Frequent PVCs with LV systolic impairment/ dilatation.
  - PVCs> 10% burden, F/U echocardiography and Holter monitoring is considered.

Indications for treatment of PVCs with structural heart disease

- Symptoms form the primary grounds for treatment
- Elimination of high burden PVCs (>10%) in patients with impaired LVEF is associated with improvement of LVEF
- Helpful when frequent PVCs interfere with CRT

Table 5 Anti-arrhythmic drugs available for the treatment of ventricular arrhythmias in most European countries

Anti-arrhythmic drugs (Vaughan Williams class)	Oral dose# (mg/day)*	Common or important adverse effects	Indications	Cardiac contra-indications and warnings
Amiodarone (III)	200–400	Pulmonary fibrosis, hypothyroidism and hyperthyroidism, neuropathies, corneal deposits, photosensitivity, skin discolouration, hepatotoxicity, sinus bradycardia, QT prolongation, and occasional TdP.	VT,VF	Conditions and concomitant treatments associated with QT interval prolongation; inherited LQTS; sinus bradycardia (except in cardiac arrest); sinus node disease (unless a pacemaker is present); severe AV conduction disturbances (unless a pacemaker is present); decompensated HF or cardiomyopathy.
Beta-blocker (II)	Various	Bronchospasm, hypotension, sinus bradycardia, AV block, fatigue, depression, sexual disturbances.	PVC, VT, LQTS, CPVT	Severe sinus bradycardia and sinus node disease (unless a pacemaker is present); AV conduction disturbances (unless a pacemaker is present); acute phase of myocardial infarction (avoid if bradycardia, hypotension, LV failure); decompensated HF; Prinzmetal's angina.
Disopyramide (IA)	250–750	Negative inotrope, QRS prolongation, AV block, pro-arrhythmia (atrial flutter, monomorphic VT, occasional TdP), anticholinergic effects.	VT, PVC	Severe sinus node disease (unless a pacemaker is present); severe AV conduction disturbances (unless a pacemaker is present); severe intraventricular conduction disturbances; previous myocardial infarction; CAD; HF; reduced LVEF; hypotension.
Flecainide (IC)	200–400	Negative inotrope, QRS widening, AV block, sinus bradycardia, pro-arrhythmia (atrial flutter, monomorphic VT, occasional TdP), increased incidence of death after myocardial infarction.	PVC, VT	Sinus node dysfunction (unless a pacemaker is present); AF/flutter (without the concomitant use of AV-blocking agents); severe AV conduction disturbances (unless a pacemaker is present); severe intraventricular conduction disturbances; previous myocardial infarction; CAD; HF; reduced LVEF; haemodynamically significant valvular heart disease; Brugada syndrome; inherited LQTS (other than LQTS3); concomitant treatments associated with QT-interval prolongation.

Ablation versus Medical Therapy

- Medications can suppress PVCs based on aetiology/mechanism of PVCs.
- **Outflow tract PVCs** are suppressed with beta-blockers and CCBs.
- **Fascicular PVCs** are particularly sensitive to verapamil.
- **PVCs triggered by delayed after depolarizations** are suppressed with Na channel-blockers (class I antiarrhythmics) flecainide and mexiletine.
- **Re-entrant PVCs** are suppressed with class III antiarrhythmics including sotalol, dofetilide, and amiodarone. Class I agents also have a complementary role.
- **Sotalol or amiodarone** are rarely required for outflow tract or fascicular PVCs.

Ablation versus Medical Therapy

- Medical therapy is limited by lack of efficacy in many patients.
- Beta-blockers, CCBs, and Na channel-blockers might cause fatigue or reduced ventricular inotropy and are difficult to take regularly on a long-term basis.
- Class I (e.g. flecainide) and class III (e.g. sotalol and dofetilide) antiarrhythmic drugs have the risk of life-threatening pro-arrhythmia.
- So, patients may choose to pursue catheter ablation of PVCs implicated in causing symptoms, ventricular tachyarrhythmia, or cardiomyopathy.

Considerations for ablation of PVCs
1. Symptomatic PVCs when drug therapy is ineffective, not tolerated or not preferred
2. PVC-mediated cardiomyopathy
3. PVC (often fascicular) repeatedly inducing ventricular fibrillation

PVC Catheter Ablation

- Success rate of PVC ablation is 75-98% of patients
- The complication rate is 1%
- most studies included highly symptomatic patients/high burden of PVCs
- Procedural success is low in patients with:
  - Coronary veins or epicardial foci
  - Polymorphic PVCs
  - Non-induced clinical PVC morphology

PVC Catheter Ablation Outcomes

- RVOT & fascicular PVCs have success rate of 80-100%
- LV function improves within 4 months in 70% of PVC- cardiomyopathy
- Recurrence is possible due to remodelling of the arrhythmogenic substrate
- Risk of Coronary arteries injury should be considered (coronary veins/cusps, epicardial space, and posterior RVOT close to the course of the LM artery)
- Chordal apparatus entrapment, aortic valve perforation and thromboembolic complications could occur with vigorous ablation movement

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NEW RESEARCH PAPERS

Multicenter Outcomes for Catheter Ablation of Idiopathic Premature Ventricular Complexes

Rakesh Latchamsetty, MD,* Miki Yokokawa, MD,* Fred Morady, MD,* Hyungjin Myra Kim, ScD,† Shibu Mathew, MD,‡ Roland Tilz, MD,‡ Karl-Heinz Kuck, MD,‡ Koichi Nagashima, MD, PhD,‡ Usha Tedrow, MD,§ William Gregory Stevenson, MD,§ Ricky Yu, MD,‖ Roderick Tung, MD,‖ Kalyanam Shivkumar, MD,‖ Jean-Francois Sarrazin, MD,¶ Arash Arya, MD,# Gerhard Hindricks, MD,# Rama Vunnam, MD,** Timm Dickfeld, MD,** Emile G. Daoud, MD,†† Nishaki M. Oza, MD,†† Frank Bogun, MD*

Multicenter Outcomes for Catheter Ablation of Idiopathic PVCs

- 1184 patients, procedural success was achieved in 85% of patients
- PVC location was the most significant predictor of success ($p < 0.03$).
- RVOT PVC location was the significant predictor of continued success at F/U ($p < 0.01$).
- PVC-induced CMP reported in 245 patients (21%), EF improved (from 38% to 50%, $p < 0.01$)
- Independent predictors of PVC-induced CMP: male gender, PVC burden, lack of symptoms, and epicardial PVC origin ($p < 0.05$).
- Complication rate: 5.2%, no procedure-related mortality
- Catheter ablation of frequent PVCs is a low-risk & effective strategy to eliminate PVCs and associated symptoms. Cardiac function is frequently restored after successful ablation.

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2015 ESC Guidelines for the management of patients with ventricular arrhythmias

Treatment of patients with left ventricular dysfunction and premature ventricular complex

Recommendations	Class[a]	Level[b]	Ref.[c]
In patients with frequent symptomatic PVC or NSVT:			
– Amiodarone should be considered.	IIa	B	64
– Catheter ablation should be considered.	IIa	B	341–343
Catheter ablation should be considered in patients with LV dysfunction associated with PVCs.	IIa	B	341–343

2015 ESC Guidelines for the Management of Patients with Ventricular Arrhythmias

Treatment of outflow tract ventricular tachycardia

Recommendations	Class[a]	Level[b]	Ref.[c]
Catheter ablation of RVOT VT/PVC is recommended in symptomatic patients and/or in patients with a failure of anti-arrhythmic drug therapy (e.g. beta-blocker) or in patients with a decline in LV function due to RVOT-PVC burden.	I	B	525–528
Treatment with sodium channel blockers (class IC agents) is recommended in LVOT/aortic cusp/epicardial VT/PVC symptomatic patients.	I	C	529–531
Catheter ablation of LVOT/aortic cusp/epicardial VT/PVC by experienced operators after failure of one or more sodium channel blockers (class IC agents) or in patients not wanting long-term anti-arrhythmic drug therapy should be considered in symptomatic patients.	IIa	B	195, 531–533

EHRA/HRS/APHRS Expert Consensus on Ventricular Arrhythmias

- No treatment other than reassurance is needed for patients with neither SHD nor an inherited arrhythmogenic disorder who have asymptomatic or mildly symptomatic PVCs. **I LOE C**
- Flecainide and propafenone are not recommended to suppress PVCs in patients with reduced LV function (unless caused by ventricular ectopy itself), myocardial ischaemia, or myocardial scar. **III LOE A**

EHRA/HRS/APHRS Expert Consensus on Ventricular Arrhythmias

- Catheter ablation may be beneficial by improving symptoms or LV dysfunction in patients suffering from frequent non-sustained VAs (e.g. PVC >10000 per 24h) in patients with significant symptoms or LV dysfunction without another detectable cause. **II a LOE B**

Conclusion: When to Decide PVC Ablation

Considerations for ablation of PVCs
1. Symptomatic PVCs when drug therapy is ineffective, not tolerated or not preferred
2. PVC-mediated cardiomyopathy
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An Approach To Treatment Of Patients With PVC

Structural Heart Disease	Frequent PVCs/ VT	Frequent Symptoms	Treatment
–	– (↓ on exercise)	–	Reassure
–	–	+	β blocker
–	+ (monomorphic)	+	Catheter ablation
+	–	±	1. Assess SCD risk
			2. β blocker
+	+	±	1. β blocker
			2. ICD if high SCD risk