

**VENTRICULAR PREMATURE CONTRACTIONS**



*“Dante’s Dream at the Time of the Death of Beatrice”, oil on canvas Dante Gabriel Rossetti 1871, Walker Art Gallery, Liverpool, England.*

*“...The luminous heavens had already returned almost to the same place some nine times since my birth and the time when I first set eyes upon the glorious woman of my mind, who was known to many as Beatrice, although they did not know the meaning of this name. In this life she had lived for the span it takes the star hewn firmament to move east for one twelfth of a degree, and thus she had just turned nine when first she appeared before me, and when I saw her she was nearly ten. She came clad in vermillion, the noblest color, modest and virtuous girdled and adorned in harmony with her tender years. In that moment I must truly say that the vital spark, which abides in the most secret chamber of my heart, set to trembling with such violence that even the slightest beat of my heart seemed horribly strong...”*

*Dante Alighieri, Vita Nuova II, 1292-93.*

*Dante recorded in his Vita Nuova how when he first saw Beatrice he became for the first time uneasily aware of his own heart beat, his heart skipped a beat, and then seemed to beat again with “trembling violence”. We know virtually nothing of Beatrice, other than Dante was obviously in love with her from the moment he saw her at the age of just nine years. Beatrice however would eventually marry another man. She died suddenly at the age of 24 years in the year 1290. Dante was devastated. Later in his epic “Divine Comedy” he would immortalize this woman as the “shade” that would guide him through his journey of the afterlife. Having been guided by the shade of the Roman poet Virgil through the harrowing circles of hell, and the early terraces of purgatory, a badly shaken Dante is then taken by the hand and guided through the final stages of purgatory and into the celestial spheres of heaven. His namesake Dante Gabriel Rossetti six centuries later painted the shade of Beatrice bidding farewell to her earthly body, just as she takes Dante by the hand to lead him through the afterlife.*

*Ventricular premature contractions can have a great variety of causes, some are benign such as occur upon the sighting of a beloved, though some are sinister, and may represent a serious underlying pathology. We do not know of what Beatrice died but demise at such an early age could well have signified a cardiac condition. If so Beatrice’s last sensation on Earth may well have also been a “trembling of the heart”.*

*In the Emergency Department, it will be our task to determine whether the cause is likely to be benign or likely to be sinister.*



*“Dante and Beatrice”, oil on canvas, Henry Holiday 1884, Walker Gallery, Liverpool England.*

# VENTRICULAR PREMATURE CONTRACTIONS

## Introduction

Premature ventricular contractions (PVCs) are extremely common.

They are also known as premature ventricular complexes, ventricular extrasystoles and ventricular ectopic beats.

On the whole they are benign, however the approach to management will depend on:

- The nature of the PVCs, (according to Lown's classification)
- The cause, if known.
- The risk factor profile of the patient.

## Pathophysiology

Three mechanisms are responsible for PVCs:

1. Reentry:
  - This occurs when an area of one way block in the Purkinje fibers and a second area of slow conduction are present. This condition is frequently seen in patients with underlying heart disease that creates areas of differential conduction and recovery due to myocardial scarring or ischemia. During ventricular activation, the area of slow conduction activates the blocked part of the system after the rest of the ventricle has recovered, resulting in an extra beat. Reentry can produce single ectopic beats or can trigger paroxysmal tachycardia.
2. Triggered:
  - These beats are considered to be due to after-depolarizations triggered by the preceding action potential. These are often seen in patients with ventricular arrhythmias due to digoxin toxicity and reperfusion therapy after myocardial infarction.
3. Enhanced automaticity:
  - This suggests an ectopic focus of pacemaker cells in the ventricle that has a subthreshold potential for firing. The basic rhythm of the heart raises these cells to threshold, which precipitates an ectopic beat. This process is the underlying mechanism for arrhythmias due to excess catecholamines and some electrolyte deficiencies, particularly hyperkalemia.

## Causes

Virtually **any** cardiac pathology will have the potential to cause PVCs.

In board terms the following 3 general categories will need to be considered:

### Benign physiological:

Occasional PVCs can be seen in any patient regardless of age or risk profile.

A range of precipitating factors is possible including:

- Emotional stress
- Anxiety

### Intrinsic cardiac disease:

1. Ischemic heart disease:
  - Acute coronary syndrome
  - Ischemic cardiomyopathy in general.
2. Congenital heart disease.
3. Acquired cardiomyopathies.
4. Primary electrophysiological disorders.
5. Pericarditis/ myocarditis.
- 6.. Valvular heart disease
  - Mitral valve prolapse in particular.

### Secondary Causes:

1. Drugs/ toxins.

In particular:

- Sympathomimetic agents, including caffeine.
  - **Any** drug with potential cardiotoxicity, eg (digoxin, TCAs, quinine)
2. Infection/ fever/sepsis from any cause.
  3. Metabolic:

## Respiratory

- Hypoxia
- Hypercarbia.

## Electrolyte disorders

- Acidosis/ alkalosis.
- Hyperkalemia
- Hypokalemia
- Less commonly disturbances of magnesium or calcium

4. Myocardial trauma/ contusion.

5. Endocrine:

- Thyrotoxicosis.

6. Environmental:

- Hypothermia.
- Hyperthermia, (from any cause).
- Electrical injury.

## Clinical Features

### Symptoms

1. Palpitations:

- Premature ventricular complexes may cause symptoms of palpitations or an awareness of the heart beat. Palpitations are due to an augmented post-VPC beat and post ventricular pause. Symptoms may be sensed as a pause or an extra beat. Pauses may be experienced as the heart “stopping” This can be very anxiety provoking to some.

2. Pre-syncope or syncope:

- This may occur with very frequent PVCs. Frank hypotension, however is rare.

3. Occasionally atypical chest pains may be experienced.

## Signs

### 1. Pulse character:

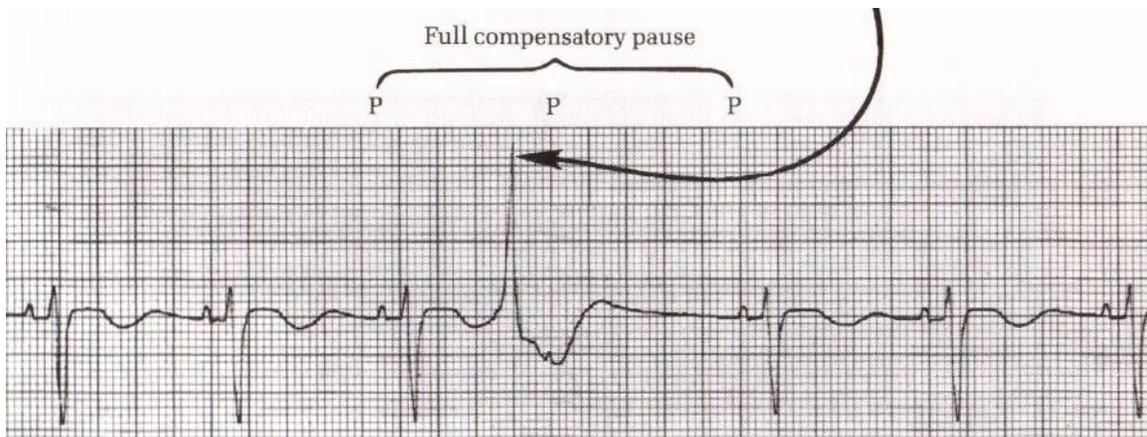
- The pulse strength of the PVC is weaker than a normal pulse because it has not allowed enough time for complete ventricular filling.
- The PVC inhibits the next sinus beat, as the ventricular myocardium is still in a refractory state. This results in the compensatory pause.
- The next sinus beat that does occur will be stronger than a normal sinus beat because the pause has allowed for increased ventricular filling.

### 2. JVP:

- Cannon A waves may be observed in the jugular venous pulse if the timing of the PVC causes an atrial contraction against a closed tricuspid valve.

## ECG Features<sup>1</sup>

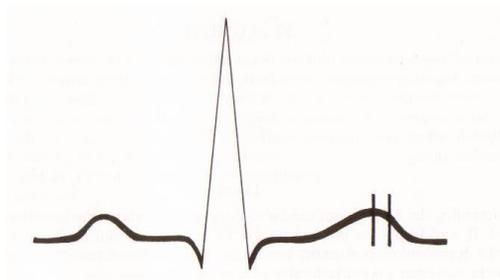
- The underlying rate may be normal, slow or fast, however PVCs are more likely to appear when the underlying rate is slow, when there is more time for them to emerge.
- The PVC does not have a related P wave, (unless there is a retrograde conduction of one, and then the P wave is abnormal and either follows the QRS of the PVC or is buried within its T wave)
- A normal P wave may be seen buried within the PVC, (see example below)
- The PVC is premature.
- It is followed by a compensatory pause.
- The QRS complex of the PVC is wide (ie greater than the normal 0.14 seconds or the width of 3 ½ small squares).
- The amplitude of the PVC is generally greater than the normal sinus beat.
- Its T wave is of opposite polarity to its R wave, as the depolarization of the ventricle is abnormal, so will be its repolarisation.



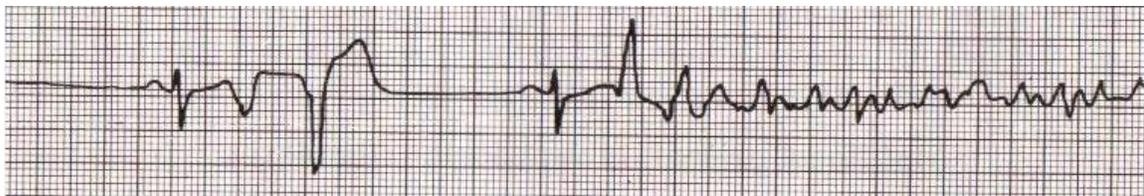
*Features of the PVC: it is wide, premature, larger amplitude, has an oppositely directed T wave and is followed by a compensatory pause. Note in this example the normal P wave can also be seen buried with the T wave complex. <sup>1</sup>*

### Types of PVCs

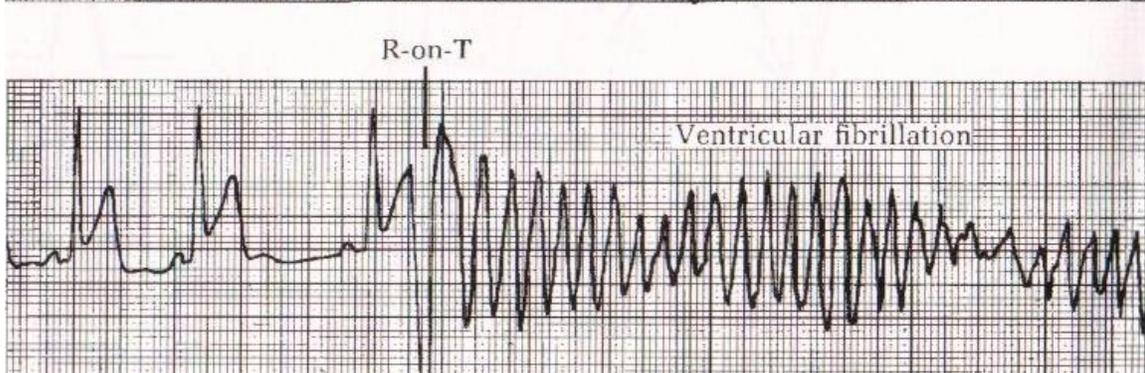
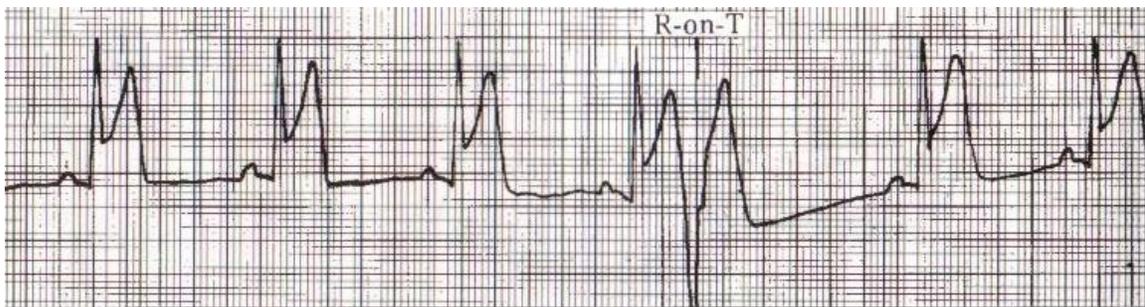
1. Unifocal:
  - PVCs with identical morphologies on a tracing are unifocal in origin, ie they originate from the same focus.
2. Multifocal:
  - If the PVCs demonstrate 2 or more different morphologies, they are multifocal in origin.
3. Regular patterns:
  - PVCs may appear in a pattern of bigeminy, (every second beat) trigeminy, (every third beat) or even quadrigeminy, (every fourth beat).
4. Salvos:
  - These are 2 or more consecutive PVCs. According to strict definition 3 or more actually constitutes VT when at a rate of 100 or greater.
5. R on T:
  - **This is the most malignant form of PVC, due to its propensity to initiate VT or VF, (see below)**
  - The synchronizer mechanism on defibrillators ensures that an R wave does not occur on a T wave in patients who undergo cardioversion.



*The vulnerable period occurs at the peak of the T wave and just after this as shown above. During this interval not all fibers of the heart are refractory, some are able to accept a stimulus and produce a propagated impulse. The result may be electrical “chaos” resulting in VT or VF when the current meets with fibers that are responsive or conducting slowly.*



*Multifocal PVCs with an R on T initiating VF. <sup>1</sup>*



*An R on T is seen in the top panel and in the bottom panel a second is seen which initiates a course VF (or polymorphic VT).*

## Significance of PVCs

When 3 or more consecutive ventricular contractions occur, this will by definition be VT. Minor runs are referred to as non-sustained VT whilst VT that persists for 30 seconds or causes hemodynamic collapse is called sustained VT, (see separate guidelines for VT).

The significance of PVCs will depend on the clinical setting:

1. Young otherwise healthy patients:
  - PVCs in young, healthy patients without underlying structural heart disease are usually not associated with any increased rate of mortality.
2. Patients with cardiovascular disease/ risk factors:
  - Frequent premature ventricular complexes have prognostic significance only in patients with underlying heart disease, particularly those with advanced left ventricular dysfunction.<sup>3</sup> They are associated with an increased risk of adverse cardiac events, particularly **sustained ventricular dysrhythmias** and **sudden death**.<sup>2</sup>
3. The setting of ACS:
  - In the setting of ACS, the risk of malignant ventricular arrhythmias and sudden death is related to the complexity and frequency of the PVCs according to the Lown grading system.

Patients with PVCs in Lown classes 3-5 are at greatest risk, ie, multifocal, salvos and especially the R on T phenomenon.

The Lown Classification for PVCs is shown below:<sup>2</sup>

<b>Class</b>	<b>Ventricular Arrhythmia</b>
<b>0</b>	None
<b>1</b>	Unifocal and < 30 per hour.
<b>2</b>	Unifocal and $\geq$ 30 per hour.
<b>3</b>	Multifocal

4A	Two consecutive.
4B	Three or more consecutive.
5	R-on-T phenomenon.

4. Secondary causes:

- The prognosis of these will depend on the underlying pathology and the prompt correction of this.

### Investigations

**Investigations will depend on the suspected underlying cause and on the risk profile of the patient.**

In young healthy individuals with infrequent PVCs and no obvious cause or risk factors will not require investigation, only reassurance.

In those who warrant investigation based on their cardiovascular or other risk factors the following investigations will be need to be considered

1. 12 lead ECG, and ongoing ECG monitoring.
2. Bloods:
  - FBE
  - U&Es/ glucose, **potassium** level in particular.
  - Magnesium and calcium levels.
  - Thyroid function tests.
  - **Serum digoxin** level if the patient is on digoxin.
  - **Troponin.**
3. CXR:
  - To document any obvious cardiomegaly or heart failure.

*Further specialized investigations may be needed according to the index of suspicion for any given condition. These may include:*

4. Echocardiography:
  - To look for any structural heart disease or dysfunction.
5. Coronary angiography:
  - If ischemic heart disease with an acute coronary syndrome is suspected.
6. Holter monitoring:
  - May be done as an outpatient in those patients with a **probable benign** cause.
7. Electrophysiological studies:
  - Where symptoms are persistent and troublesome and a primary electrophysiological abnormality needs to be ruled out.

### Management

The management and disposition of premature ventricular contractions will depend on:

- The nature of the PVCs, (according to Lown's classification)
  - The cause, if known.
  - The risk factor profile of the patient.
1. Benign cases:
    - Reduction of caffeine or alcohol intake may lessen ectopic activity.
    - Usually specific drug treatment is not required, but if so, beta blockers may reduce symptoms and be justified particularly in patients with mitral valve prolapse, who are often quite troubled by the ectopic beats.

For *intolerable* symptoms, the following may be used: <sup>3</sup>

**Atenolol**                      25 to 100 mg orally, daily

**Metoprolol**                    25 to 100 mg orally, twice daily.

2. In the setting of ACS or significant intrinsic cardiac disease:
  - Prior to 1989 it was routine practice to suppress any ventricular arrhythmic activity in the setting of an ACS. However a landmark study known as CAST <sup>4</sup> in that year it was demonstrated that routine attempts to suppress all ventricular arrhythmic activity with class I agents, in the

setting of myocardial infarction resulted in the dramatic and unexpected result of increased mortality.

The **routine** suppression of *all ventricular PVCs* is therefore no longer undertaken.

Although there remains some diversity of opinion, in general terms:

- **Low class PVCs I- 4A are not be routinely treated.**
- **Salvos may or may not be treated**
- **R on T phenomenon should be treated.**

**In the case of Lown class 4B salvos, this is really VT by definition, (and which may be sustained or non-sustained) a different scenario to uncomplicated PVCs. See separate guidelines on VT for this scenario, however in general:**

- Occasional runs of non-sustained VT need not be treated unless very frequent.
- Recurrent non sustained runs of VT which are symptomatic should be treated.
- Sustained VT must always be treated.

**Additionally in the setting of ACS, the underlying ischemia must also be treated with anti-platelet agents / anti-thrombotic therapy and re-perfusion strategies.**

**Suitable agents to use include:**

Amiodarone

- 5 mg/kg IV, over 10-20 minutes.

Sotalol

- 1 to 2 mg/kg IV, over 10 to 30 minutes

Lignocaine

- 1 to 1.5 mg/kg (usually 75 to 100 mg) IV, over 1 to 2 minutes

### 3. Secondary causes:

Treatment in these cases must be primarily directed to the cause of the ventricular ectopy.

*Examples would include:*

- FAB fragments for digoxin toxicity.
- Bicarbonate for TCA overdose.
- Potassium replacement in cases of hypokalemia.

References

1. Conover M.B Understanding Electrocardiography 7<sup>th</sup> ed 1996, p.155-171.
2. Stahmer S. Ventricular Premature Complexes in eMedicine Website, Emergency Medicine/ cardiology, September 2005.
3. Cardiovascular Therapeutic Guidelines 2006
4. The Cardiac Arrhythmia Suppression Trial (**CAST**) Investigators. Preliminary report: effect of encainide and flecainide on mortality in a randomized trial of arrhythmia suppression after myocardial infarction. NEJM vol 321, 10 August 1989 p. 406-12

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