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## Paced ECG Morphology – Reveals More than What It Conceals

Ajay Bahl

*Department of Cardiology, Postgraduate Institute of Medical Education and Research,  
Chandigarh,  
India*

### 1. Introduction

Ventricular paced rhythms can mask ECG changes of several conditions. This is because the paced ventricular rhythm does not follow the normal pattern of depolarization through the bundle of His and the bundle branches. Instead, depolarization initially occurs at the tip of the pacemaker lead which is usually at the apex or the septum. The depolarization then proceeds through the myocardium from one cardiomyocyte to another rather than through the conduction system of the heart. This results in abnormal depolarization and repolarization patterns. Thus the QRS complex is usually widened and the T wave is usually of opposite polarity to the QRS complex. Reaching a diagnosis from a single ECG already rendered abnormal by the paced rhythm is not easy. It is thus useful to have a baseline ECG available for comparison. Changes in the axis, QRS width and QRS complex morphology can give important clues to the diagnosis. ECG changes of acute myocardial infarction in patients with a ventricular paced rhythm are well described. There is very limited data on the usefulness of paced ECG in the diagnosis of other conditions.

### 2. Acute myocardial infarction

Like most other conditions, ECG findings of acute myocardial infarction can be masked by paced rhythms. Pacing leads are traditionally placed in the right ventricular apex. Most studies of ECG findings of myocardial infarction are in patients with pacing leads placed at this location. The ECG pattern of right ventricular apical pacing resembles a left bundle branch block (LBBB) and the diagnostic criteria are also similar. The increasing use of other pacing sites like the interventricular septum and outflow tract as well as biventricular pacing will result in different ECG findings but some general rules will still apply. There is lack of data on the ECG diagnosis of myocardial infarction when the pacing lead tip is placed at locations other than the apex. As these alternate pacing sites gain popularity, the criteria for myocardial infarction recognition that is discussed below may not apply.

Traditionally ECG changes in myocardial infarction in the setting of LBBB include ST segment abnormalities, abnormal Q waves and Cabrera's sign.<sup>1</sup> There are however some differences in the setting of LBBB and ventricular pacing. The most important difference is in value of Q waves in the diagnosis. Abnormal Q waves in leads V5 and V6 along with

increased r in lead V1 is very specific for anterior wall myocardial infarction in the setting of LBBB. In right ventricular pacing, these Q waves however could simply reflect differences in the lead tip position rather than myocardial infarction.<sup>1,2</sup> Thus Q in leads V5 and V6 could be normal finding in right ventricular pacing. A well positioned lead at the RV apex rarely generates a qR complex in lead I, and probably never produces a qR complex in V5 and V6 in the absence of an MI. Thus presence of Q waves in these leads, though useful should be used with the caveat that if the lead tip is not at the right ventricular apex, then Q waves may also be normally seen in these leads. Another important point is to differentiate a qR/QR from a QS complex. This differentiation is important because a QS complex carries no diagnostic value during RV pacing in any of the leads (QS complexes can be normal in leads I, II, III, aVF, V5, and V6) whereas qR/QR complex can have diagnostic value.<sup>2</sup> One cannot determine the age of the MI from the QRS complex changes.

ST segment changes are the most useful findings in diagnosing myocardial infarction in patients with paced rhythms. The GUSTO-1 trial has provided useful information in this regard. In this trial, 32 patients had ventricular paced rhythm. The only ECG criterion with a high specificity and statistical significance for the diagnosis of an acute myocardial infarction was ST segment elevation  $\geq 5$  mm in leads with a negative QRS complex.<sup>3</sup> Shape of the ST segment which exhibit upward convexity are useful in this context. Two other criteria with acceptable specificity were ST elevation  $\geq 1$  mm in leads with concordant QRS polarity and ST depression  $\geq 1$  mm in leads V1, V2, or V3.<sup>3,4</sup> It is important to remember that though these criteria are fairly specific, their sensitivity is low. The ECG criterion with the highest sensitivity for the diagnosis of an acute myocardial infarction was ST segment elevation  $\geq 5$  mm in leads with a negative QRS complex and even this criterion has a sensitivity of only around 50%.

The GUSTO-1 trial also included 131 patients with LBBB.<sup>5</sup> These ST segment diagnostic criteria were the similar for patients with LBBB and ventricular paced rhythms. The difference in the setting of paced rhythm as compared to LBBB was that of the 3 criteria, the one with the greatest value for the diagnosis of an acute MI in paced rhythm was ST segment elevation  $\geq 5$  mm in leads with a negative QRS complex, whereas in the setting of LBBB the criteria with the greatest value was ST elevation  $\geq 1$  mm in leads with concordant QRS polarity.

Another classical sign described is the Cabrera's sign.<sup>1,2</sup> Like other signs, this has also been adapted from the criteria diagnosing myocardial infarction in the setting of LBBB. Cabrera's sign is described as notching of the ascending limb of the S wave usually in leads V3 and V4, and sometimes in leads V2 and V5. The notch should be  $\geq 0.03$  seconds and present in 2 leads.<sup>2</sup> It has low sensitivity but is a very specific sign.

The diagnostic criteria described above are for anterior wall myocardial infarctions. Infarctions at other sites are usually masked by the paced rhythm. Cabrera's sign in both leads III and aVF may be of some value in diagnosing inferior wall myocardial infarction. Similarly ST elevation in lead V4R may occur in acute right ventricular infarction. This sign should however be taken with extreme caution unless accompanied by signs of inferior myocardial infarction.

There are a few important factors that could confound the diagnosis. Retrograde P waves in the terminal part of the QRS complex may mimic Cabrera's sign. Because of cardiac memory repolarization abnormalities, mostly T wave inversions may occur if the patient reverts to spontaneous rhythm. These T wave inversions are secondary to pacing per se and not related to ischemia. These can occur even after very short duration of pacing.

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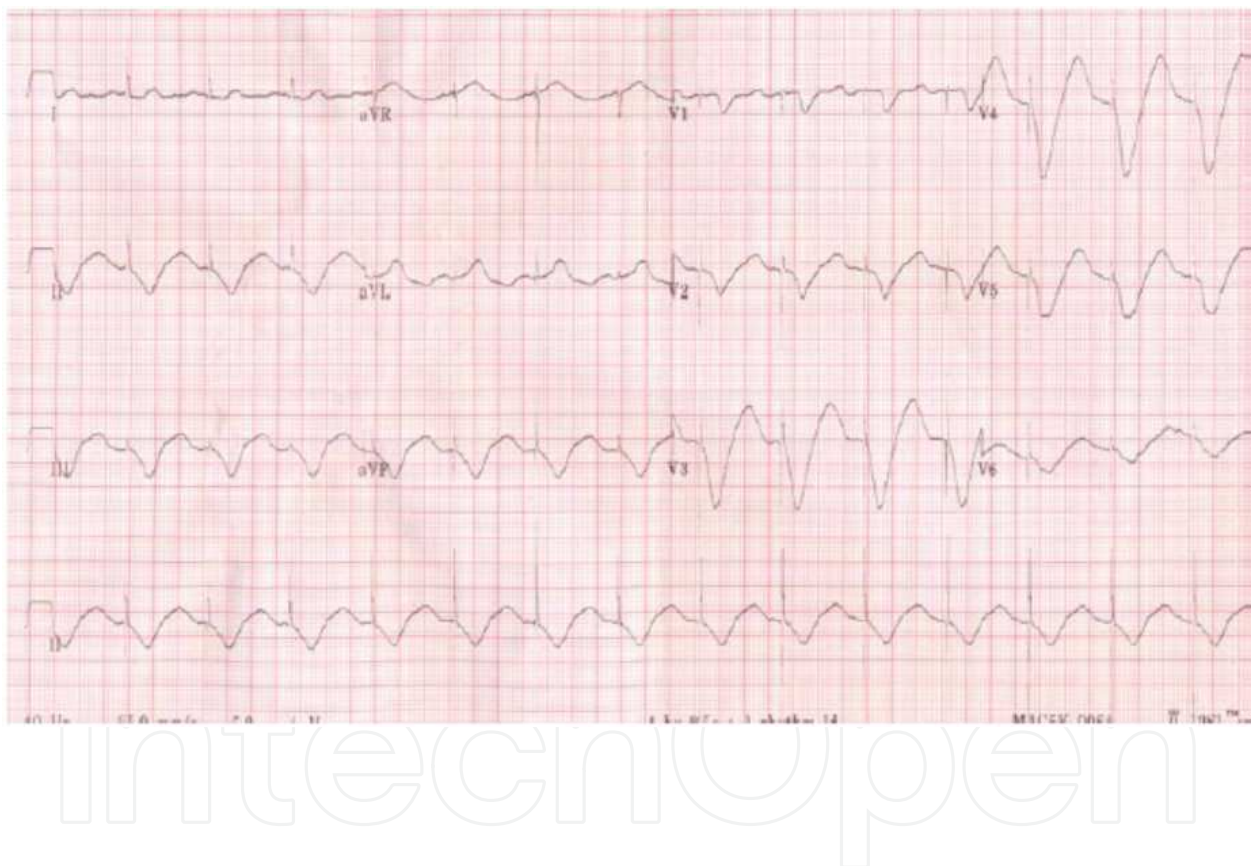


Fig. 1. ECG at presentation showing that all ventricular beats are paced. The pacing spike is followed by a wide QRS complex (QRS duration 300 milliseconds). The QRS complex is merging with T waves. No definite P waves are seen. (With permission, Bahl A et al, Indian Heart J 2009;61:93-4).



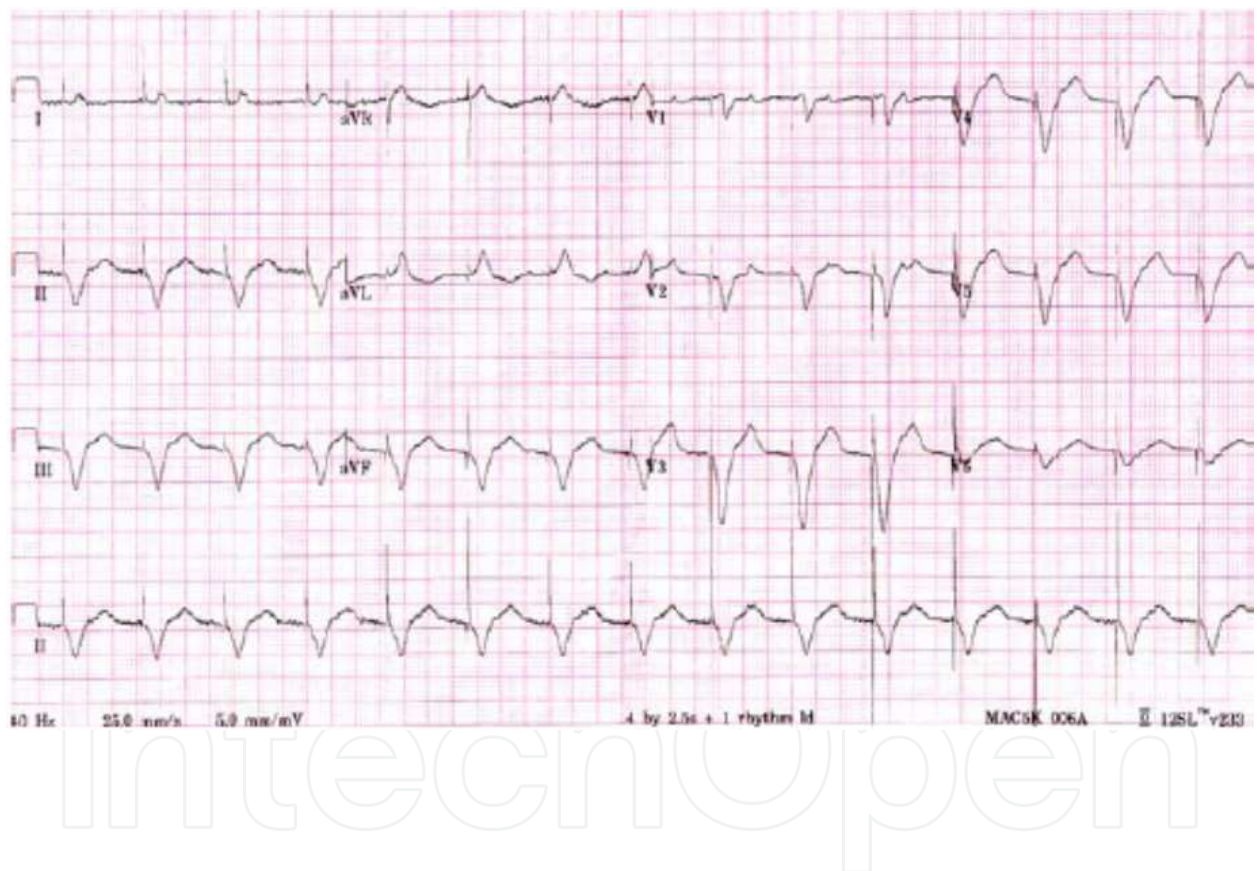


Fig. 2. ECG after correction of hyperkalemia. All ventricular beats are still paced but the QRS duration has narrowed to 190 milliseconds. The QRS complexes are greater in amplitude and the peaks are sharper in morphology. Definite P waves are seen in leads V1 and V2. (With permission, Bahl A et al, Indian Heart J 2009;61:93-4).

### 3. Widening of the QRS complex

Widening of the QRS complex is an important though non-specific sign in patients with paced rhythm. A baseline paced ECG is ideally required if this sign is to be used in clinical situations. QRS prolongation should trigger an alarm bell. Any observer during failed cardiopulmonary resuscitations in patients on temporary or permanent pacemakers would have noted gradual prolongation of the QRS complex with the passage of time. The QRS complex gradually becomes very wide and resembles a T wave. This is a poor prognostic sign and indicates progressive myocardial ischemia and dysfunction.

Studies using balloon inflation at time of percutaneous coronary intervention have shown that QRS prolongation could also be a marker of myocardial ischemia on the paced electrocardiogram.<sup>6</sup> In addition, electrolyte imbalance as in hyperkalemia could also result in QRS prolongation. Thus any change in QRS duration from baseline should be noted as it could be an important indicator of several pathologies.

### 4. Hyperkalemia

ECG is a useful guide in diagnosing hyperkalemia in patients in sinus rhythm. Initial change is usually a peaked T wave. This is followed by widening of QRS complexes, intraventricular conduction defects, prolongation of PR interval, absence of P wave, heart blocks, and the classical sine wave. This is because severe hyperkalemia decreases the phase 0 of the action potential resulting in widening of the QRS complex. The QRS complex continues to widen and ultimately blends with the T wave resulting in a sine wave morphology. Typical ECG changes of hyperkalemia can also be seen even during paced rhythm. These changes include QRS widening and reduction in amplitude of the QRS complex.<sup>7,8</sup> The QRS widening can be quite marked in some cases. These changes are especially well appreciated if an old ECG is available for comparison. Typical sine waves may also be seen on a paced ECG rhythm. These changes revert back to the baseline with correction of hyperkalemia. These ECG findings of hyperkalemia that reverted back to baseline after correction of hyperkalemia are illustrated in figures 1 and 2.

To summarize, a number of medical conditions are best diagnosed during paced rhythms when a baseline ECG is available. All patients with pacemakers should have a recorded baseline 12 lead ECG available. In case the patient with pacemaker has his own rhythm a 12 lead ECG with magnet should be kept as a taken. This would be available as a record of the paced rhythm in case the patient later becomes pacemaker dependant and has a continuously paced rhythm.

### 5. Acknowledgements

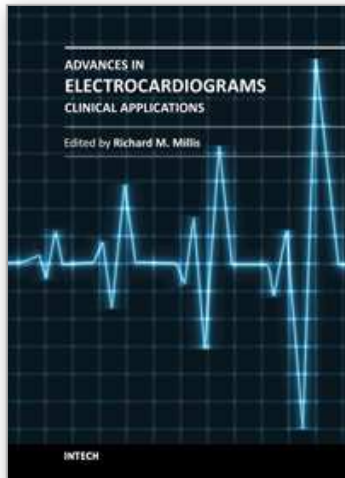
I thank the Honrary Editor, Indian Heart Journal for permission to use figures 1 and 2.

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Electrocardiograms have become one of the most important, and widely used medical tools for diagnosing diseases such as cardiac arrhythmias, conduction disorders, electrolyte imbalances, hypertension, coronary artery disease and myocardial infarction. This book reviews recent advancements in electrocardiography. The four sections of this volume, Cardiac Arrhythmias, Myocardial Infarction, Autonomic Dysregulation and Cardiotoxicology, provide comprehensive reviews of advancements in the clinical applications of electrocardiograms. This book is replete with diagrams, recordings, flow diagrams and algorithms which demonstrate the possible future direction for applying electrocardiography to evaluating the development and progression of cardiac diseases. The chapters in this book describe a number of unique features of electrocardiograms in adult and pediatric patient populations with predilections for cardiac arrhythmias and other electrical abnormalities associated with hypertension, coronary artery disease, myocardial infarction, sleep apnea syndromes, pericarditides, cardiomyopathies and cardiotoxicities, as well as innovative interpretations of electrocardiograms during exercise testing and electrical pacing.

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