

## Case Report

# Temporary cardiac pacing induced electrocardiographic changes simulating myocardial infarction

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### ABSTRACT

Temporary transvenous pacing is an immediate lifesaving measure in patients with Stokes-Adams syndrome and in patients with symptomatic bradycardia. Bradyarrhythmias are known to occur in acute myocardial infarction. But in a paced heart, it is difficult to diagnose myocardial infarction from electrocardiogram (ECG) because pacemaker rhythm causes distortion of natural wave forms. On the other hand, remarkable T wave inversions and ST depressions do occur in the ventricular paced ECG as secondary changes. The case report describes a patient who developed profound de novo T wave inversions and ST depressions in the unpaced ECG following temporary transvenous pacing simulating MI.

**Keywords:** ECG changes, Myocardial infarction, Temporary pacing

### INTRODUCTION

Temporary transvenous pacing is an immediate lifesaving measure in patients with Stokes-Adams syndrome and in patients with symptomatic bradycardia.<sup>1,2</sup> Temporary pacemakers are also being used for overdrive pacing to revert life threatening arrhythmias refractory to pharmacological agents. Recently temporary pacing has become an essential component of many cardiac interventions like coarctation of aorta stenting and percutaneous aortic valve replacement.

Bradyarrhythmias are known to occur in acute myocardial infarction. But in a paced heart, it is difficult to diagnose myocardial infarction from electrocardiogram (ECG) because pacemaker rhythm causes distortion of natural wave forms. On the other hand, remarkable T wave inversions and ST depressions do occur in the ventricular paced ECG as secondary changes.<sup>3</sup>

The case report describes a patient who developed profound de novo T wave inversions and ST depressions in the unpaced ECG following temporary trans venous pacing. These changes are proved not due to an acute MI, but resulted from primary abnormality of ventricular repolarization.

### CASE REPORT

A 65-years lady was admitted to IMS and SUM Hospital, Bhubaneswar with complaints of recurrent syncope for one day. There were no other antecedent cardiac events. On examination, her heart rate was 20 beats per minute and blood pressure was 70/50 mmHg.

No significant findings observed over the chest. No murmurs or gallops were auscultated. The ECG at presentation showed complete heart block with bradycardia. Patient was immediately shifted to the cath

lab and temporary pacemaker was implanted through right femoral vein and pacing lead tip was positioned near right ventricular apex under fluoroscopic guidance. The procedure was uneventful. Pacing rate was kept at 60

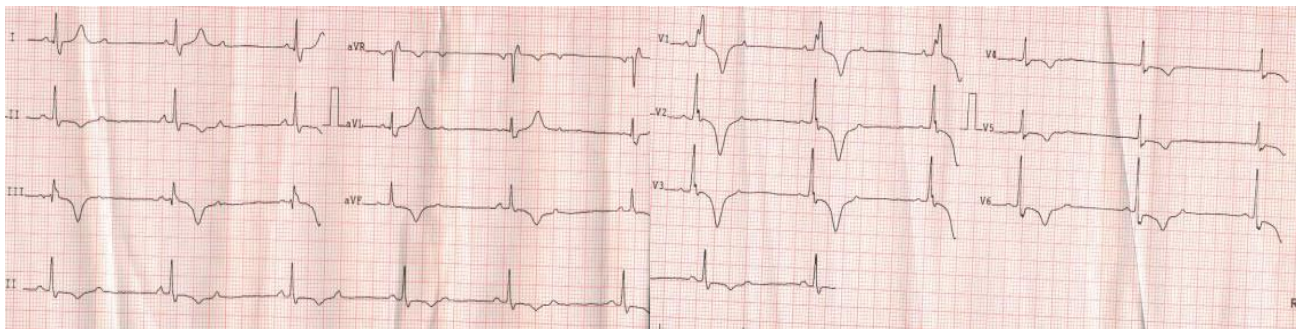
beats per minute. Patient's vitals got stabilized and sensorium improved. Though patient's serum K<sup>+</sup> level was 6.1 meq/l at presentation, it got corrected next day. But there was persistent 2:1 AV block in ECG (Figure 1).



**Figure 1: After one day of continuous pacing, the unpaced ECG showing second degree block with 3:1 AV conduction and upright T waves.**

So, patient was planned for permanent pacemaker implantation. Surprisingly on day 3, when ECG was

taken after switching off the pacemaker, it showed new onset deep T wave inversions and ST depressions in V1-V6, II, III and aVF leads (Figure 2).



**Figure 2: After two days of continuous pacing, the unpaced ECG showing striking T wave inversions in II, III, aVF and V1-V6.**

These ECG changes persisted on repeated recordings. Patient was not giving any definitive history suggestive of acute coronary syndrome and troponin T test was negative.

Echocardiography did not show any regional wall motion abnormality and LV systolic function was found to be normal. But, considering patient's age and ECG changes, coronary angiography was done, which also revealed normal coronary arteries Figure 3a and 3b).

All the findings proved that, the AV block was due to degenerative disease of the conduction system of heart and the ST-T changes are likely result of persistent ventricular pacing.

Permanent pacemaker was implanted due to the irreversible nature of AV block and the patient was discharged in a stable condition.



**Figure 3: a) Right anterior oblique (RAO) with caudal view demonstrating normal caliber of left anterior descending (LAD) and Left circumflex (LCX) arteries, b) Left anterior oblique (LAO) cranial view depicting normal caliber of right coronary artery.**

## DISCUSSION

ST segment depression and T wave inversion appeared on day 3 of temporary pacemaker implantation in our patient. These changes were not accompanied by alteration in QRS complex (no pathologic Q waves and no alteration of R wave amplitude. Lawrence Gould and colleagues had presented two case reports with similar ST-T wave changes.<sup>4</sup> Chatterjee and his associates had described similar repolarization changes in 29 of 31 patients paced primarily for heart block.<sup>3</sup> They noted that the ST-T wave changes occurred in post paced ECGs whether the rhythm was supraventricular or ventricular. The changes were independent of atrial rate and QRS duration. Moreover, the degree of ST-T wave change was related to the duration of pacing as well as to the amount of power used for ventricular pacing. The ECG changes could appear as early as 10 minutes of pacing when pacing was done with high voltage at fast rates. The duration of ST-T changes depended on the duration of pacing.

In our patient, we initially attributed the ST-T wave changes to myocardial infarction, which could also be responsible for AV block. However, this diagnosis was unlikely because of absence of QRS changes, presence of diffuse nature of T wave inversions and the lack of enzyme changes. Moreover, echocardiography did not show any regional wall motion abnormality and coronary angiography revealed normal coronary arteries. Rothfeld and Jucker had observed the ECG and VCG changes before and after experimentally produced myocardial infarction in dogs with right ventricular pacemakers.<sup>5</sup> After infarction, there was a striking change in conduction pathway and velocity of initial QRS forces.

Stokes-Adams attacks as well as cerebro-vascular accidents can produce ST-T wave changes.<sup>6,7</sup> Our patient had Stokes-Adams attacks before hospitalization, but the ST-T changes appeared only after two days of ventricular pacing in the hospital.

Though the pacemaker-induced ECG changes are unlikely to be of any clinical importance, they can simulate myocardial infarction and lead to wrong medications and unnecessary prolonged hospital stay.

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