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Case Report

# Ventricular bigeminy after electroconvulsive therapy: a case report

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## Abstract

Electroconvulsive therapy (ECT) is an effective tool for treatment resistant depression. In this case report we highlight the case of an elderly Asian patient who developed ventricular bigeminy immediately after his 19th session of ECT. Electrocardiogram (ECG) variations including T wave and ST segment changes have been reported in the literature post ECT. However this case offers various learning points; ventricular bigeminy occurred within 1 hour and there was a discordance between the clinically palpated pulse by nursing staff and the rate on the ECG. These changes were also transient and did not occur again in subsequent sessions of ECT. Reassuringly these ECG changes are commonly benign, and ECT remains a safe choice of treatment even with those with cardiac dysfunction. However, awareness of the effects of ECT on the heart, specifically the ECG changes is important and should be recognised by both the psychiatric and anaesthetic team.

## Key words

Catatonia, Depression, Electrocardiogram, Electroconvulsive Therapy, Pulse Deficit, Ventricular Bigeminy,

## Introduction

There have been several reported cases of electrocardiogram (ECG) changes following electroconvulsive therapy (ECT). In particular, sinus arrhythmias, ST segment changes and changes to T waves such as inversion and flattening have been reported.[1-3] Furthermore, changes suggestive of Wolf Parkinson White have also been highlighted.[4] We present a case of a patient who experienced ECG changes consistent with ventricular bigeminy following ECT session.

ECT is an effective intervention for treatment resistant psychiatric disorders including severe depression, catatonia and prolonged manic episodes.[5] It is accepted as a low risk intervention, being suitable to be given to patients even with severe cardiac disease.[6] The exact mechanism of ECT is still unknown, with numerous biological, psychological and psychoanalytical theories proposed.[7]

Ventricular bigeminy is described when every normal sinus beat is followed by a premature ventricular complex (PVC); thus, every second beat is repeated as a PVC.[8] This occurs when there is premature ventricular depolarisation and hence contraction of the ventricles due to electrical impulses from the ventricular myocardium.[9] As these impulses are not originating from the atrioventricular node, they have an unusual ECG waveform. The slow ventricular conduction leads to a wide QRS complex,[10] with discordant ST segment and T-wave changes.[11] In ECGs with fixed coupling (where there is a constant time interval between the sinus beat and PVC), this can be indicative of the sinus beat controlling the PVC via a re-entry mechanism,[12] or other causes.

PVCs can be seen in patients with cardiac disease such as infection, ischemia and inflammation. Electrolyte imbalances, stress states such as hypoxia or surgery and substances such as caffeine, alcohol and tobacco can also all lead to PVCs. Furthermore, autonomic stimulation can lead to the production of PVC's.[13]

In the majority of patients, PVCs are benign with no intervention needed. Treatment is reserved to those with symptoms or with haemodynamic compromise and includes different classes of anti-arrhythmic agents or radiofrequency ablation.[13]

## Case Summary

A 77 year old Asian male was admitted to an old age psychiatric unit with severe depression with catatonic symptoms including being mute and loss of motor movements. He had 19 sessions of ECT with limited response. Following the 19th session, it was noted he had become bradycardic. An ECG was performed which showed ventricular bigeminy with several ectopics with a heart rate (HR) of 104 and QTc of 520 (figure 1). These changes were not present on a previous ECG, which was sinus rhythm. Bloods including urea and electrolytes, thyroid function tests, phosphate, magnesium, calcium were all done and were normal.

**Figure 1: ECG of patient following a session of ECT, the ECG shows changes consistent with ventricular bigeminy**

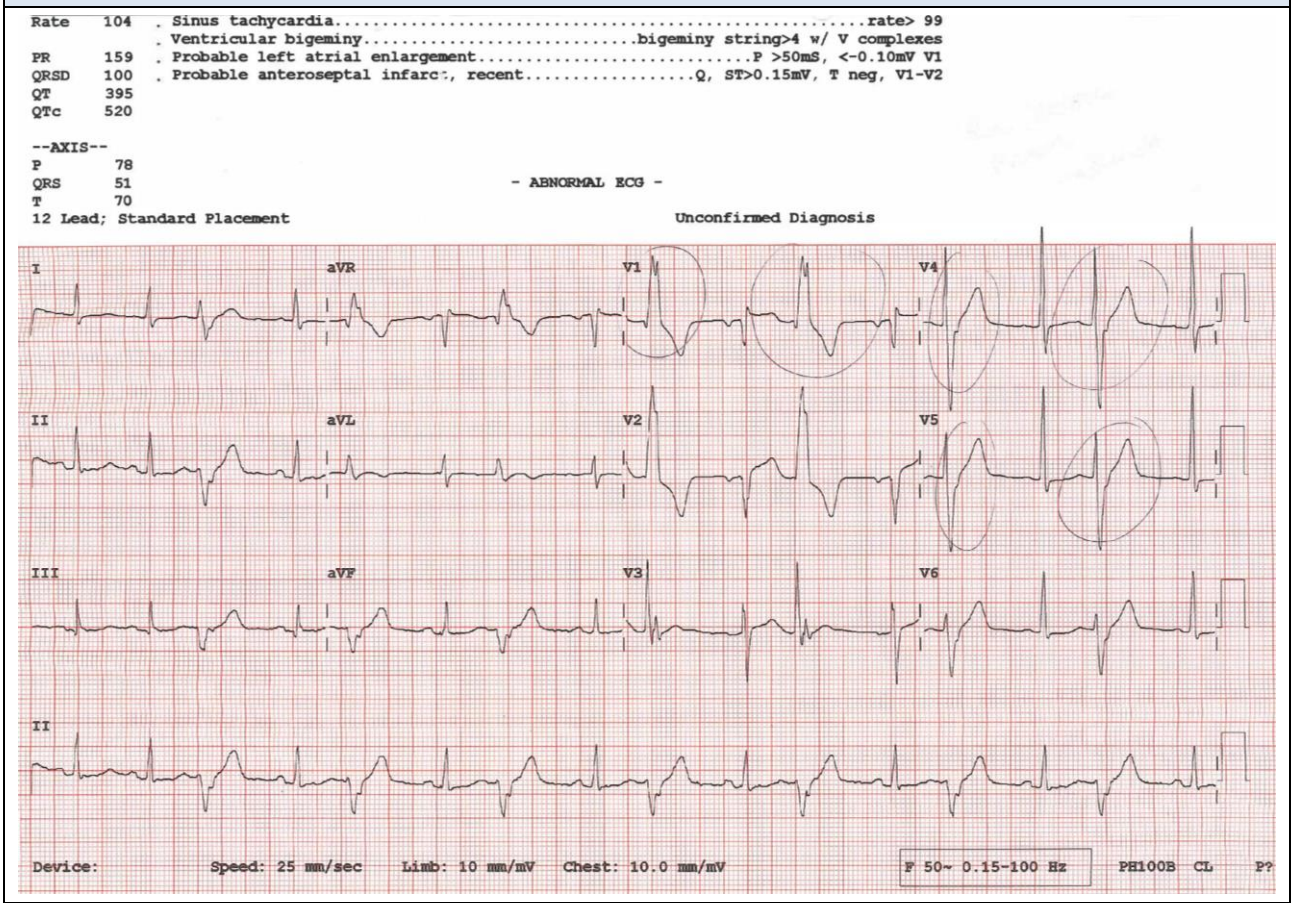
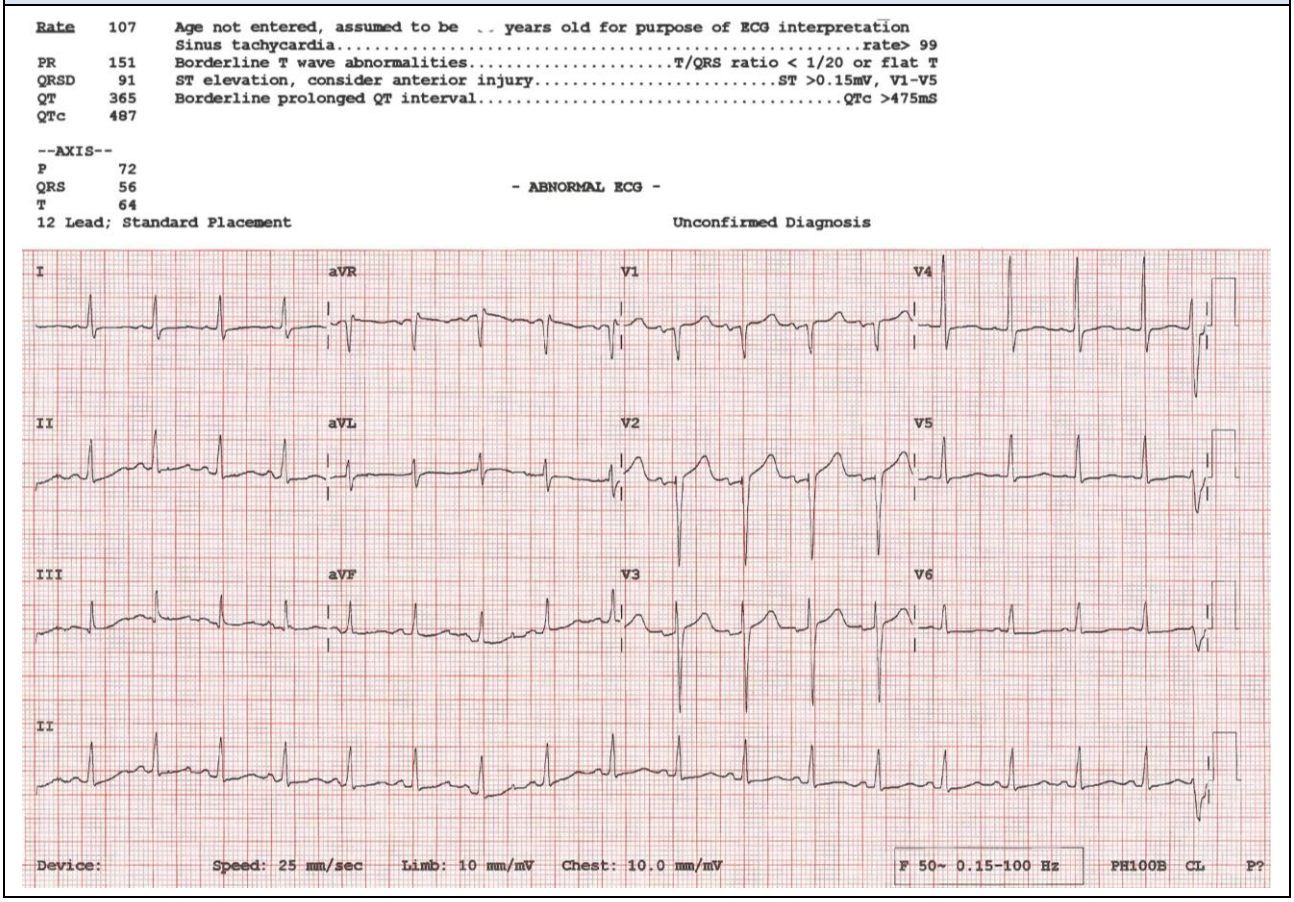



**Figure 2: Repeat ECG shows resolution of the previously seen changes**



The ECG was subsequently repeated the following day, where the aforementioned changes had gone (figure 2). Prior to the patient's next session of ECT, ECGs were repeated pre and post procedure which showed no acute changes.

The patient had an extensive cardiac history. He had ischemic heart disease requiring percutaneous intervention on two occasions; this has led to impaired left ventricular function. He had also had an incidental finding of a previous subcortical infarct on routine magnetic resonance imaging (MRI) scan during his admission. His other past medical history included hypertension and hypothyroidism. He was a non-smoker.

The patient's medications were as follows: diltiazem hydrochloride 120mg BD, atorvastatin 10mg, clopidogrel 75mg OD, thiamine 100mg BD, cholecalciferol with calcium carbonate 1tablet OD, Senna 15mg ON and macrogol 3350 with potassium chloride, sodium bicarbonate and sodium chloride 1 sachet BD. During his inpatient stay, he was commenced on mirtazapine 30mg ON, venlafaxine 187.5mg OD, diazepam 2mg BD and diazepam 1mg ON. Diazepam was stopped during the ECT.

The patient continued his course of ECT for 24 sessions, with a limited clinical response. There were reports of days where his catatonic behaviour improved however this was never for a prolonged period of time. He was therefore trialled on the mood stabiliser lamotrigine, after ECT, with minimal response. It was therefore concluded that this would be the patient's new clinical baseline and he was discharged back to his home. He had a good family support network and was also given home treatment team input on discharge.

## Discussion

There were several aspects of the case that highlight key learning points. The ECG changes that occurred were immediate, and resolved after 24 hours without further reoccurrence; this is contrary to the reports in literature.[2] Furthermore it highlights the importance of ECGs in quantifying heart rates; the patient's pulse was reported as bradycardic, whereas this did not match the heart rate of 104 on the ECG. This was likely to be due to non-conducted pulse associated with the premature ventricular contraction (PVC), this difference in heart rate and pulse rate is the pulse deficit.[13]

Several studies have looked at the effects of ECT on the cardiac system. A triphasic autonomic change has been noted during and after ECT, with an initial parasympathetic, then sympathetic and then finally parasympathetic drive occurring. The initial parasympathetic dominance has been hypothesised to be due to the rapid onset of the parasympathetic neurotransmitter acetylcholine relative to the sympathetic neurotransmitter noradrenaline.[14] The resulting seizure from ECT and its associated adrenomedullary catecholamine release has also been thought to be linked to the sympathetic response.

Studies have shown the incidence of premature ventricular complexes post ECT, with one study looking at the elderly noting a statistically significant rise in bigeminy/trigeminy 24 hours post ECT. There was however no change within 1 hour of ECT.[2]

Research into QT dispersion (QTD and QTcD), which is thought to be a marker of cardiac electric inhomogeneity,[8] was noted to be higher in the elderly relative to the young post ECT.[15] It was concluded that the older population were therefore at an increased risk of ventricular arrhythmias due to increased heterogeneity of ventricular depolarisation.[16] However, the potential for ventricular arrhythmias was subsequently found to not be due to repolarisation abnormalities.[17]

Furthermore, this patient's ECGs were subsequently performed pre and post ECT for the next rounds which were all normal. The transient cardiac changes which subsequently resolved suggest these changes were secondary to ECT on the background of cardiac disease; this has been previously hypothesised to be due to an increased sympathetic drive.[18]

## Conclusion

This is an interesting case of an elderly patient with ECG changes post ECT. This case highlights the importance of careful history taking to understand the risk of potential ECG changes. We are reassured that these ECG changes are often benign with no clinical significance and no detrimental effects.[1] However, recognition is important to ensure suitable management and follow up if necessary; this patient was seen by the cardiology department where an ECHO was organised in view of his physical health co-morbidities. In addition, highlighting causes of discrepancies in clinical observations will aid in improvement of patient care. The definitive measurement of heart rate is to do an ECG, with the added benefit of the identification of the electrical activity of the heart. Thus, if following ECT a patient becomes unwell or develops changes in the physiological parameters of their observations, care should be taken to thoroughly examine the patient. This is to potentially identify any physical health problems and seek help or advice from the medical team if deemed necessary.

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