We describe a patient who showed remarkable and unexplained QT-T wave changes after implantation of a cardiac resynchronization device with defibrillator (CRT-D). A 50-year-old female with non-ischemic cardiomyopathy, left ventricular ejection fraction 30%, NYHA functional class III, and QRS of 140 ms with typical left bundle branch block underwent a CRT-D implantation in November 2013. Her post-procedural electrocardiogram (ECG) showed wide and deeply inverted T waves across the precordium with prolongation of the QTc interval from 540 ms to 670 ms. Her chest X-ray showed appropriate lead position with no complications. Capture and sensing thresholds were stable. The right and left ventricles were pacing simultaneously. The patient denied chest pain, dyspnea, or headache. She did not receive any QT prolonging drugs peri-procedurally and there were no clinical signs of raised intracranial pressure. Serial cardiac enzymes and electrolytes were normal. There was no known family history of long QT and premature sudden cardiac death. The baseline ECGs of the patient’s daughter and her father were available and had normal QTc interval. Genetic testing was not done due to financial reasons. She was kept in the hospital for an additional day and serial ECGs showed a reduction in the QTc to 600 ms by discharge but the T-wave changes persisted. Follow-up ECG at 10 weeks showed progressive resolution of the T-wave inversion reversion of QTc back to the baseline level. From the available data, it was difficult to make a diagnosis of congenital long QT syndrome as the patient had no clinical symptoms or family history, and the QTc was difficult to interpret in view of the baseline conduction defect and prolonged QRS.

Chatterjee et al.1 and Rosenbaum et al.2 were the first to describe cardiac memory (CM) in pacing. It has been described after pacing, intermittent left bundle branch block, paroxysmal tachycardia, pre-excitation, radiofrequency ablation of accessory pathways, frequent premature ventricular contractions, and ventricular tachycardia.3 Following return to stable ventricular activation, the T waves tend to normalize. This is the metaphorical “memory” of the T wave.

CM is a diagnosis of exclusion. It can last for hours to months,4 but most often it resolves spontaneously and hence the diagnosis can usually be made only on follow-up. The pathophysiology of cardiac memory is unclear. Change in the order of ventricular activation seems to affect ventricular repolarization causing T-wave changes due to electrotonic interactions.2,5,6 Variation in the activation sequence appears to alter regional myocardial stretch. This causes release of angiotensin II-inducing changes in the transient outward current (Ito) and I-type calcium current resulting in cardiac memory.7–9

Our case demonstrates a unique situation, suggesting that dynamic and pronounced ST and T wave changes...
can follow biventricular pacing. Although the marked QT prolongation and T wave morphological changes were not arrhythmogenic, their significance and prognosis in this case remain unknown. This may be another form of cardiac memory.

References