

EP CASE REPORT

Multipoint pacing controlled the electrical storm induced by cardiac resynchronization therapy

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A 42-year-old man with the diagnosis of non-ischaemic cardiomyopathy and atrial fibrillation was referred to our institution for cardiac resynchronization therapy (CRT). He had a history of decompensated heart failure following single chamber pacemaker implantation due to total AV block 4 months ago. A cardiac resynchronization device with a defibrillator was implanted following extraction of single chamber pacemaker system.

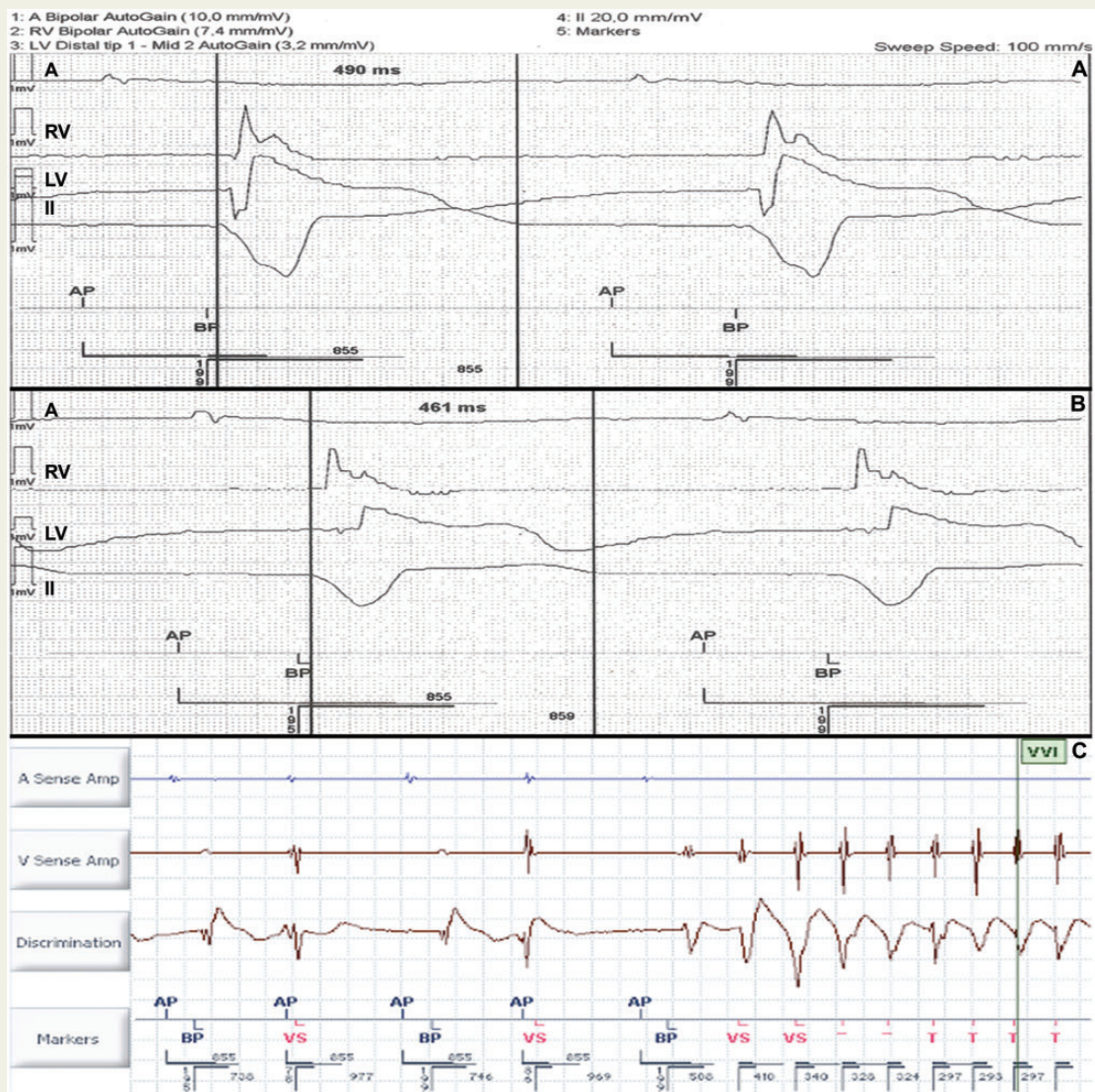


Figure 1 Comparison of intracardiac and surface ECG recordings during conventional (LV distal 1-2, RV) (A) and multipoint (distal 1-2, proximal 4-2, RV) BiV pacing (B). PMVT initiated by a ventricular extra systole (C). A, atrium; ECG, electrocardiogram; LV, left ventricular; PMVT, polymorphic ventricular tachycardia; RV, right ventricular.

Five hours after implantation, the patient suffered an electrical storm with recurrent polymorphic ventricular tachycardia (PMVT) episodes and had seven shocks in the day of implantation. Blood samples were normal, and patient has no history of ventricular arrhythmia since the diagnosis of heart failure. Antiarrhythmic drugs including intravenous beta-blockers, lidocaine, amiodarone, and conscious sedation were tried. Overdrive biventricular (BiV) pacing was also performed. Unfortunately, patient had eight more shocks during the second day, and atrial fibrillation turned to sinus rhythm. Whenever CRT device was programmed to DDD-right ventricular (RV) only pacing mode, PMVTs were ceased and reinitiated with BiV pacing. Since the quadripolar left ventricular (LV) lead was implanted, different LV pacing sites and polarities were programmed to overcome pacing site-dependent arrhythmic effects. However, every attempt resulted in ventricular arrhythmias. Finally, multi-point LV pacing was programmed to create a larger depolarization wavefront. Furthest LV electrodes distal 1-2 and the proximal 4-2 were selected. Immediately after this adjustment, ventricular arrhythmias were ceased, and patient was discharged after 5 days. During hospitalization, whenever the device was reprogrammed to BiV-single LV site pacing mode in order to reduce energy consumption, PMVTs were observed suggesting the causal relationship.

This finding may prove the causal relationship. During 5 months of follow-up; the patient had no episodes under amiodarone and beta-blocker and the functional class improved to The New York Heart Association (NYHA) Class 3.

Discussion

Despite the benefits of CRT on mortality and morbidity, CRT-induced pro-arrhythmia has been reported in a rare subgroup of patients.¹ Although the mechanism remains unknown, two different mechanisms seem to play a role for different type of arrhythmias. Re-entry is the responsible mechanism for most of the monomorphic ventricular tachycardia (MMVT). Pacing near a critical site of slow conduction, is more likely to induce MMVTs. Reversal of normal myocardial activation sequence during epicardial pacing, as it occurs during CRT, increases the transmural dispersion of repolarization (TDR).^{2,3} Delayed activation and repolarization of mid-myocardial M cells during BiV pacing leads to a prominent increase in QT and TDR and prolongation of ventricular repolarization, makes the ventricle vulnerable to ventricular extrasystole that results in PMVTs (Figure 1C). In our case, multipoint LV pacing was associated with suppression of electrical storm (ES). Immediately after programming, QT interval was decreased to 460 from 490 within seconds, without other modifications of device setting (Figure 1A, B). Different pacing sites may produce different vectoral activation and may affect ventricular repolarization patterns. However, each attempt from different LV pacing sites and polarities failed, but multipoint pacing was effective. Larger depolarization wavefront initiated by multipoint LV pacing may result in more homogeneous repolarization pattern and may suppress the PMVTs. Depolarization of larger area within the epicardial scar may also be effective to control re-entrant MMVTs, especially when the patient has long stim to QRS interval during LV threshold testing.

Decompensation of heart failure, electrolyte disturbances, and increased sympathetic tone during per-operative state may also lead to ventricular arrhythmias. These independent factors are clear limitations and cannot be excluded. However, disabling single-point BiV, either by multipoint BiV or RV only pacing, was reproducibly inhibiting PMVTs under the same condition.

In conclusion, this case highlights for the first time the potential role of multipoint LV pacing for emergency treatment of ES induced by CRT.

References

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