Right ventricular lead proarrhythmia: A novel intervention for an under-recognized phenomenon

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Introduction

Large clinical trials have demonstrated the benefit of cardiac resynchronization therapy (CRT).[1,2] As such, CRT has become an important guideline-recommended, device-based therapy for the management of congestive heart failure.[3] However, these devices also pose a risk of rare adverse effects. One adverse outcome, which has been well-documented in case reports and cohort studies, is the proarrhythmic effect of CRT devices in a small subset of patients, which typically is attributed to the epicardial pacing of the left ventricular (LV) lead.[4,5] Specifically, newly implanted LV leads may increase the frequency of ventricular tachycardia (VT) and electrical storm owing to pacing into regions of scar and slow conduction. However, the potentially proarrhythmic effects of right ventricular (RV) pacing in a CRT device is not often considered, but is equally plausible if septal or apical scar/regions of slow conduction are present.

Here, we present a case of VT in a patient with a CRT device where the RV lead was felt to be initiating VT, and we describe our approach to CRT programming that suppressed the clinical arrhythmia.

Case report

The patient was a 73-year-old man with a history of ischemic cardiomyopathy with an ejection fraction (EF) $< 20\%$ with a Biotronik Lumax 740 HF-T biventricular implantable cardioverter-defibrillator (ICD) that had been implanted in 2008. Though his EF remained $< 20\%$ with CRT, he had significant clinical response. He subsequently had a decline in functional status when his LV lead became dislodged. His symptoms then improved again when the lead was revised.

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The patient presented to an outside facility with shortness of breath, dizziness, and left-sided chest pain, and an electrocardiogram (ECG) demonstrated monomorphic VT. He was initiated on amiodarone and lidocaine with resolution of his VT, and he was discharged on oral amiodarone. He then re-presented several days later with VT, and again was started on intravenous amiodarone and lidocaine in addition to beta-blockers. He was transferred to our facility for consideration of radiofrequency ablation. Basic laboratory and chemistry values were unremarkable. An ECG of his VT is shown in Figure 1.

He underwent endocardial ablation on hospital day 8. His baseline pacing parameter was set for simultaneous RV-LV pacing. Prior to the procedure, as per protocol, his ICD was reprogrammed for RV pacing only, and VT occurred spontaneously (Figure 1A). The clinical VT had several spontaneous initiations in the electrophysiology laboratory with tachycardia cycle length 435–460 ms with a left bundle branch morphology, rS in V_2 and QS in V_3–V_6, left superior axis, and dominant R wave in I and aVL (Figure 1B). This morphology was very similar to the patient's ICD RV pacing QRS morphology. Endocardial voltage mapping of the LV revealed a large septal anterior and apical scar, close to the site of the patient's RV ICD lead (Figure 2C). One day post-ablation, he had recurrence of VT with similar morphology, which required multiple antitachycardia pacing therapies (Figure 1C). His biventricular ICD was reprogrammed with his LV lead preceding RV pacing by 55 ms without any titration of antiarrhythmic medications (Figure 1D). This was the shortest delay that clearly altered the pacing vector. Following this, the patient had no further episodes of VT and he was discharged to a skilled nursing facility 7 days later on oral amiodarone and mexiletine. Mexiletine was discontinued at a 2-month follow-up visit, as he was doing well. At 10-month follow-up visit, he has had no further VT events on biventricular ICD interrogation.

Discussion

There are multiple reports in the literature of CRT-induced ventricular arrhythmias, primarily relating to the LV

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KEY TEACHING POINTS

- Proarrhythmic effects due to the right ventricular lead in cardiac resynchronization devices is a rare phenomenon.
- When clinical ventricular tachycardia (VT) morphology is similar to right ventricular (RV) pacing morphology, this suggests the RV lead may be pacing into a critical area of slow conduction within the VT circuit.
- Reprogramming a cardiac resynchronization therapy device to a delay of RV pacing after left ventricular pacing can result in arrhythmia suppression when the clinical VT is likely due to RV pacing.

lead.[4–7] Reported management strategies for VT resulting from CRT devices include antiarrhythmic therapy, catheter ablation, or device reprogramming, such as turning off LV pacing. Theories of proarrhythmia due to CRT devices center around effects of LV pacing. Among its numerous effects, LV pacing reverses the normal activation wavefront pattern to epicardium to endocardium, and it also has the potential of pacing into scar, which may enter critical isthmuses, leading to sustained VT.[4,5,8] Additionally, canine experiments have demonstrated that transmural dispersion of repolarization increases when pacing occurs from the epicardium vs the endocardium.[9] In humans, biventricular and LV epicardial pacing have been shown to prolong the QT interval and enhance transmural dispersion of repolarization compared to RV endocardial pacing.[10]

Proarrhythmic effects of single- and dual-chamber pacemakers/ICDs have also been reported.[11–13] A high percentage of RV pacing may worsen heart failure in patients with cardiomyopathy, which can lead to increased ventricular arrhythmias. Additional postulated mechanisms of proarrhythmia in these studies included local irritability of the lead occurring early after device implantation and/or fibrosis near the lead tip developing at any time after device placement. Additionally, any form of ventricular pacing has been shown to increase ventricular electrogram

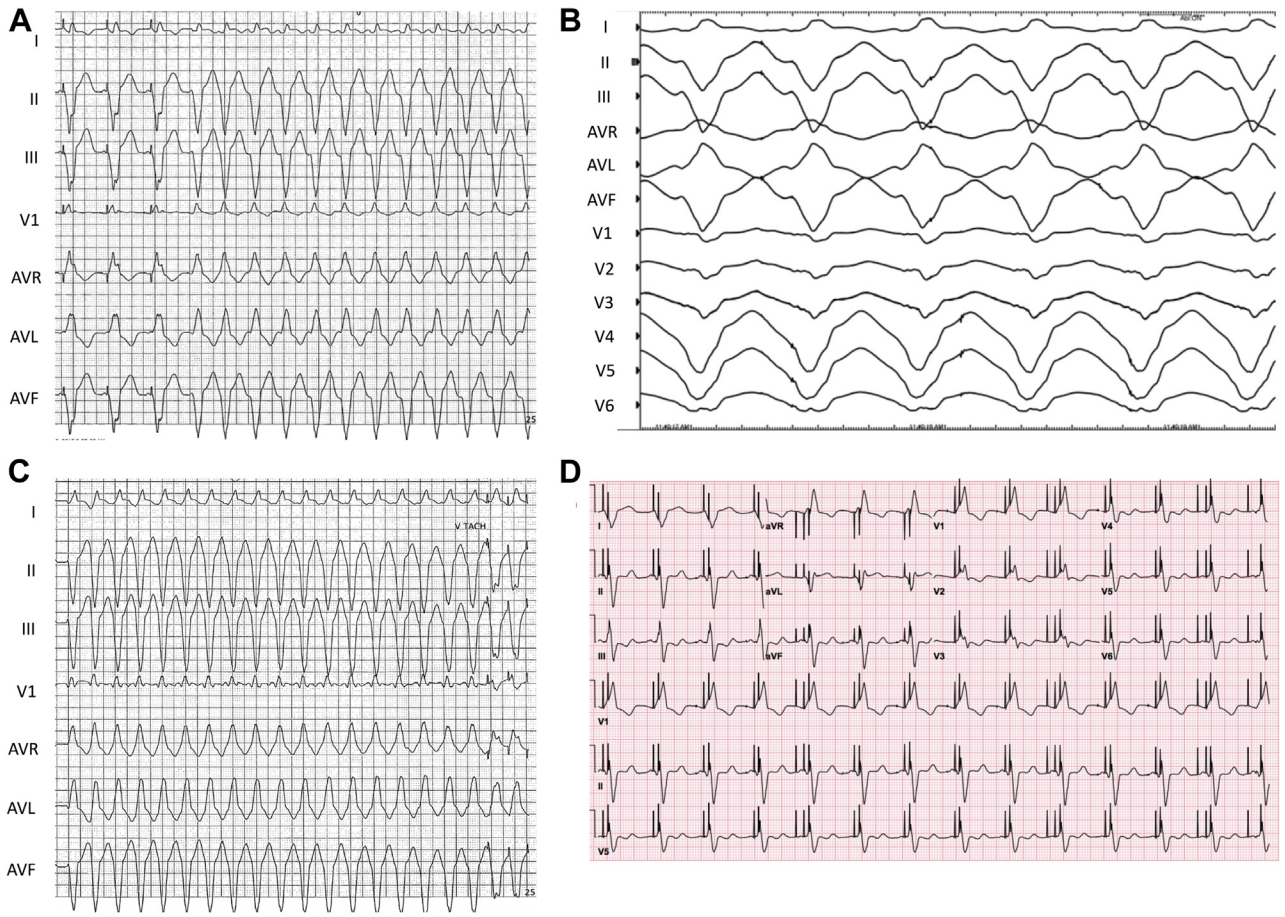

Figure 1 **A:** Telemetry strip of ventricular tachycardia (VT) at a rate of 135 beats/min, which occurs immediately after a sequence of right ventricular (RV) pacing. From top to bottom, the leads shown are I, II, III, V_5, aVR, aVL, and aVF. **B:** A 12-lead surface electrocardiogram (ECG) of the clinical ventricular tachycardia during electrophysiology study. Precordial lead placement was not in typical location. **C:** Telemetry strip of recurrent VT with similar morphology following endocardial ablation, which required antitachycardia pacing. **D:** A 12-lead ECG following reprogramming of cardiac resynchronization therapy device with RV lead programmed with 55-ms delay after left ventricular pacing, which was the shortest delay that clearly altered the pacing vector.

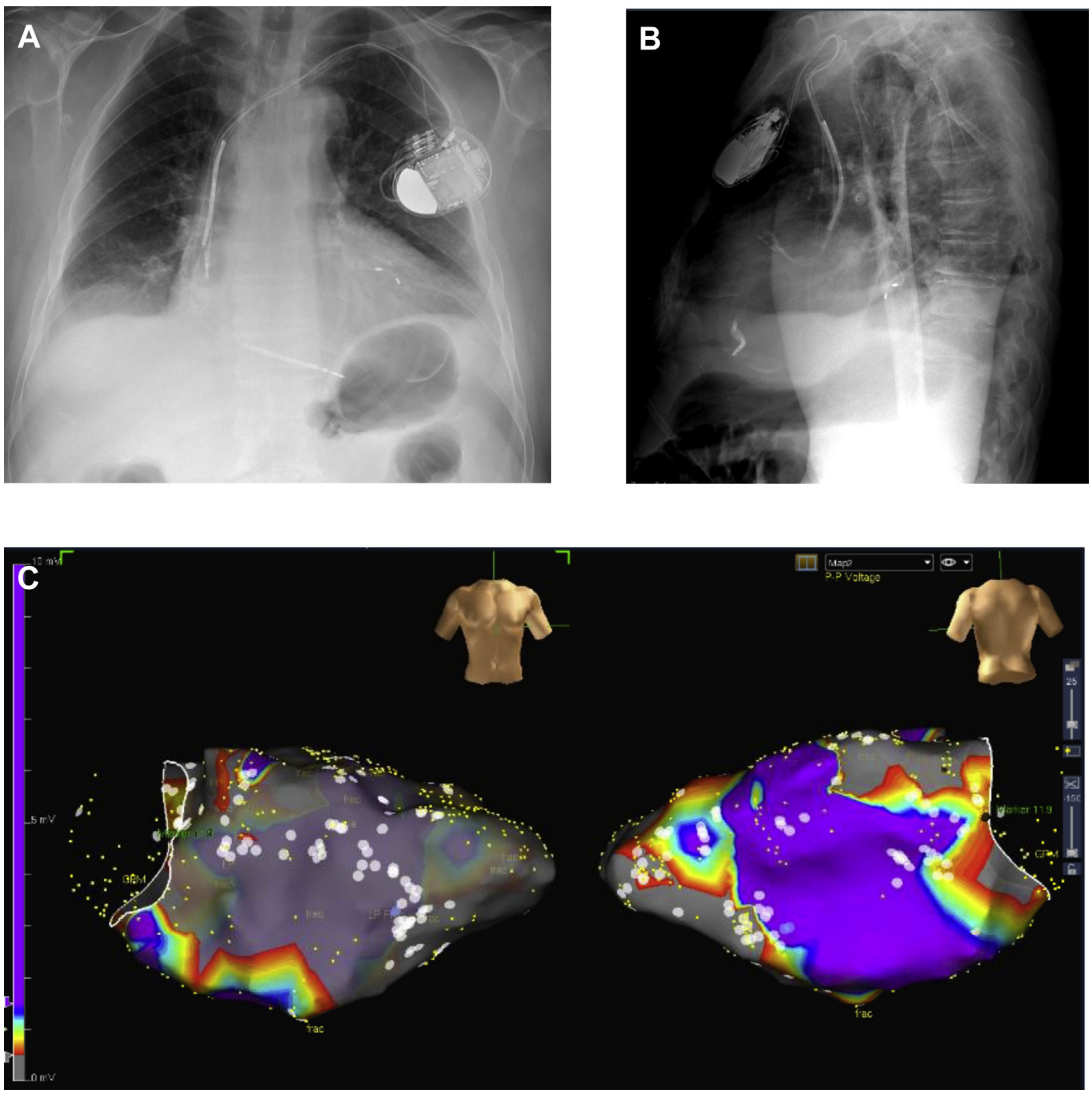

Figure 2 **A:** Posterior-anterior chest radiograph with cardiac resynchronization therapy device over left chest. **B:** Lateral chest radiograph with right ventricular lead visualized in the apex. **C:** Endocardial voltage map (Ensite, St. Jude, Minneapolis, MN) of the left ventricle at standard scar settings (0.5–1.5 mV) demonstrating very large septal anterior and apical scar with areas of preserved voltage in mid-inferior and mid-lateral walls (white dots = ablation lesions).

fractionation. This indicates possible functional conduction slowing, which can progress to conduction block and the development of reentry. In cases of VT induced by ventricular pacing from single- or dual-chamber devices, arrhythmias can be eliminated by either turning pacing off or decreasing pacing output to a subthreshold level.[12,14] Turning off or decreasing pacing output was possible in these reports because the patients were not pacemaker-dependent, but such a method would be suboptimal in patients with CRT devices, where pacing is required in order to achieve clinical benefit. In another report, by Lee and colleagues,[15] arrhythmias were eliminated in select patients by extracting ICD leads. Device-induced VT has also been reported in patients whose devices were performing automatic threshold measurements in both a CRT and standard ICD device.[16] In these patients, disabling the autocapture feature caused arrhythmias to subside.

In our patient, VT appeared to be the result of RV pacing, with the clinical VT resembling RV pacing morphology. Our patient had large septal and apical scars close to the site of the RV lead. The mechanism of VT in this case was likely pacing within or very close to a region of slow conduction responsible for the clinical VT circuit. As described in the manuscript by Tung and colleagues,[17] during VT ablation, a "pace-map induction" of VT suggests that the pacing site is near or at a critical region of slow conduction. VT occurred spontaneously during biventricular pacing and was more frequent when pure RV pacing was programmed for the ablation procedure. Subsequently the arrhythmia subsided when the device was adjusted to delay RV pacing after LV pacing, allowing for a different RV activation vector. In this patient, VT resolved after device reprogramming without any need for further ablation or antiarrhythmic therapy. Theoretically, LV pacing alone might have been attempted; however, our

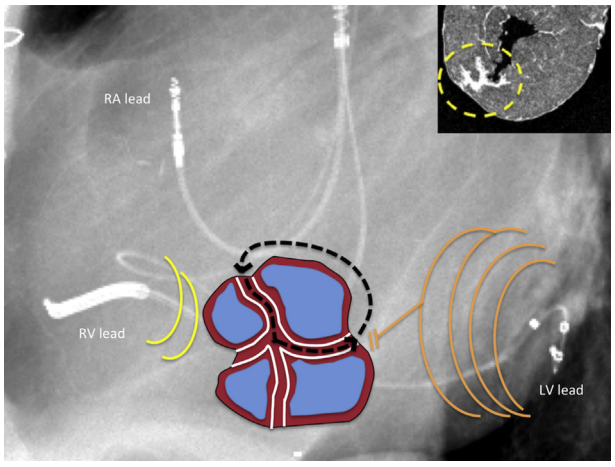

Figure 3 Proposed mechanism of ventricular tachycardia (VT) suppression. Lateral projection of plain chest radiograph demonstrating right ventricular (RV) lead pacing directly into a VT circuit within scar. Offsetting left ventricular pacing prior to RV pacing creates a refractory limb of the circuit, which does not allow for reentry. **Inset:** Schematic histopathologic representation of scar-based substrate, which can promote reentry.

patient had clinical benefit from biventricular pacing and therefore it was felt best to remain in a biventricular mode. Finally, the contribution of antiarrhythmic therapy in suppressing VT cannot be excluded, as the patient remained on amiodarone. However, medications had been successfully weaned, and temporally, ventricular arrhythmias ceased after device reprogramming.

The hypothesized mechanism of VT suppression in this case is repetitive conduction into a critical site of the reentrant VT circuit by the pre-excited LV pacing wavefront. If sufficient time exists between the LV pacing impulse and the RV stimulus, the VT circuit may remain refractory, and activation cannot proceed through the circuit within the septal scar (Figure 3). This mechanism likely requires unidirectional block to be present such that when the RV impulse encounters the opposing wavefront, this results in bidirectional block. The exact region/limb of refractory tissue cannot be ascertained from the data available and may be variable from case to case.

Conclusion

To our knowledge, this is the first reported case of monomorphic VT resulting from RV pacing in a patient with a CRT device. The incidence of this phenomenon is unknown. In our patient, arrhythmias resolved by programming LV preceding RV pacing. Such a technique might be considered in cases where clinical VT closely resembles RV pacing in patients with CRT devices; however, this requires further study.

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