

# Right ventricular lead position can be critical in determining clinical response to cardiac resynchronization therapy—A case of successful cardiac resynchronization response conferred by relocating the right ventricular pacing lead



Xiaoke Liu, MD, PhD,<sup>\*†</sup> Kyle Liu,<sup>‡</sup> Yong-Mei Cha, MD<sup>\*</sup>

From the <sup>\*</sup>Department of Cardiovascular Medicine, Mayo Clinic, Rochester, Minnesota, <sup>†</sup>Mayo Clinic Health System, La Crosse, Wisconsin, and <sup>‡</sup>University of Michigan, Ann Arbor, Michigan.

## Introduction

Despite advances in the implant technique over the last few decades, optimal cardiac synchronization and positive response still cannot be achieved in about 30% of patients.<sup>1</sup> More recently, conduction system pacing, especially left bundle branch area (LBBA) pacing, has rapidly emerged as a potential new alternative to biventricular pacing through the coronary sinus (CS) and has been used in some instances to improve the cardiac resynchronization therapy (CRT) response in patients who failed conventional CRT.<sup>2–5</sup> Nevertheless, even with LBBA pacing, there are still a significant number of cases where underlying left bundle branch block (LBBB) cannot be corrected.<sup>2–5</sup>

We here report a case in which a conventional CRT-defibrillator (CRT-D) nonresponder underwent an attempted LBBA pacing implant that failed to correct the underlying LBBB or shorten the QRS duration. However, simple relocation of the right ventricular (RV) pacing lead from a more apical position to an anteroseptal and basal location in combination with the previously implanted coronary sinus lead substantially narrowed the QRS and led to excellent CRT response.

## Case report

The patient is a 65-year-old man with a prior history of ischemic cardiomyopathy, ejection fraction (EF) 30%, NYHA class III, long-standing persistent atrial fibrillation (failed 2 ablation procedures), and chronic LBBB (QRS duration 176 ms). He underwent initial conventional CRT-D implantation in 2014 when he was still in sinus

## KEY TEACHING POINTS

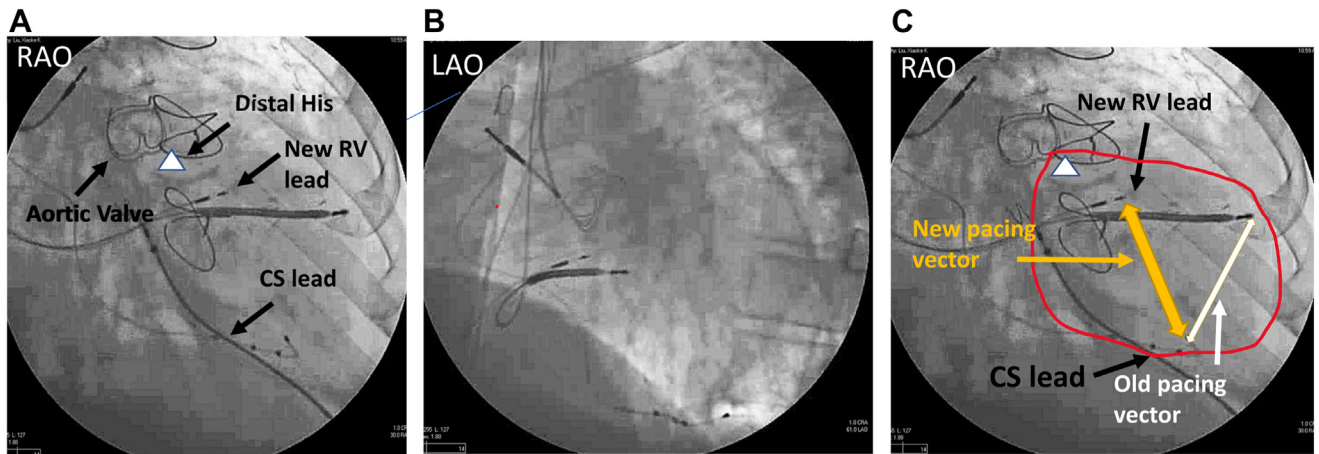
- Right ventricular pacing lead position may be critical in cardiac resynchronization therapy (CRT) response.
- The pacing vector that covers the most left ventricle myocardium may lead to superior CRT response.
- Failed left bundle branch area pacing lead may still confer benefit when combined with coronary sinus lead.

rhythm. He was found to have only 1 suitable target CS branch in the posterior position for left ventricular pacing. There were no other available branches in locations ideal for CRT. The QRS duration continued to be wide (168 ms) despite CRT. Left ventricular EF and heart failure symptoms did not improve after the CRT procedure. The patient ultimately developed class IV heart failure symptoms in 2019, for which the LBBA pacing was considered to optimize the management of his heart failure. The option of repeat atrial fibrillation ablation was also discussed but was declined by the patient owing to the failure of the 2 previous ablations and the presence of severe left atrial enlargement.

The patient's left precordium was prepared and draped in the usual fashion. The axillary vein was reaccessed. A deflectable electrophysiology catheter was then inserted and used to map the distal His bundle position. After successful registration of the His bundle potential position, a Medtronic C315 delivery sheath was advanced to the vicinity of the left bundle area, defined as an area 1–1.5 cm distal from the recorded His bundle signal, toward the left ventricular apex (Figure 1A). A Medtronic 3830 lead was advanced over the delivery sheath to engage the LBBA. Unfortunately, despite multiple attempts, we failed to advance the lead tip

**KEYWORDS** Left bundle; His bundle; Cardiac resynchronization; Coronary sinus; Right ventricular pacing  
(Heart Rhythm Case Reports 2023;9:764–767)

**Address reprint requests and correspondence:** Dr Xiaoke Liu, Mayo Clinic Health System, 800 West Ave. S., La Crosse, WI 54601 and Mayo Clinic, Rochester, MN. E-mail address: [liu.xiaoke@mayo.edu](mailto:liu.xiaoke@mayo.edu).



**Figure 1** Fluoroscope views of left bundle branch area lead and coronary sinus (CS) lead positions. **A:** Right anterior oblique (RAO) view. **B:** Left anterior oblique (LAO) view. **C:** Pacing vectors before and after right ventricular (RV) lead relocation.

deep into the ventricular septum toward the left ventricular endocardium and the underlying LBBB with wide QRS failed to correct (Figure 2A and 2C). The superficial location of the 3830 lead was later confirmed by postoperative surface echo (Figure 3). Prior to abandoning the upgrade procedure, we decided to test a trial of biventricular pacing from the previously implanted CS lead and newly implanted RV pacing lead that failed LBBB pacing because there seemed to be excellent separation between these 2 leads on fluoroscopy. Simultaneous pacing from the CS lead (pole 2 to pole 3) and the 3830 lead, now positioned in basal and anteroseptal right ventricle, resulted in a much narrower QRS morphology. The new pacing configuration resulted in a marked shortening of the paced QRS to 130 ms and a very short left ventricular activation time as estimated by the R-wave peak time of <70 ms in  $V_6$  (Figure 2D). The newly implanted LBBB pacing lead was then connected to the atrial port with the pacing mode programmed to DDDR using a minimal AV delay at 30 ms, because the LBBB pacing lead was *not compatible with* the DF-4 right ventricular pacing port (Figure 2D). The pacing output from the previously implanted RV pace/sense/defibrillation lead was set to minimum to avoid RV apical pacing.

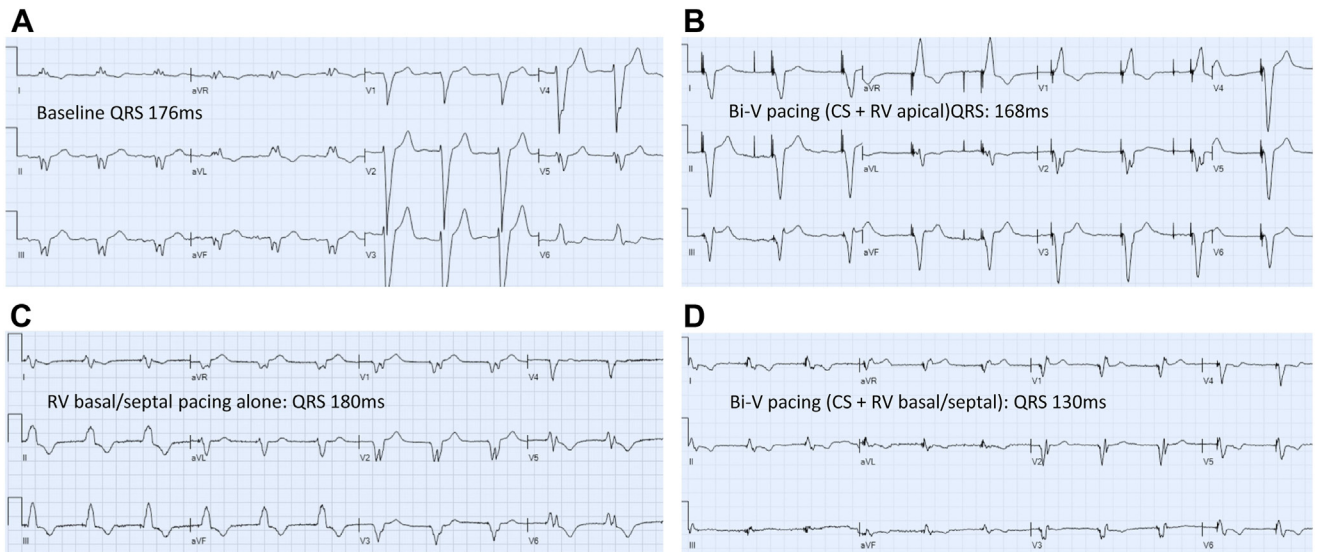
After successful CRT system revision, a repeat echo 2 months post procedure showed an increase in EF from 30% to 45%. The patient's heart failure symptoms improved by 1 functional class.

## Discussion

Conduction system pacing, especially LBBB pacing, has recently gained substantial traction as an alternative to traditional coronary sinus pacing for CRT.<sup>3-5</sup> Numerous reports have shown impressive QRS shortening and improvement in heart failure together with excellent pacing parameters in patients who previously failed standard CS pacing and continued to have wide QRS duration.<sup>3-5</sup> However, there

are still a significant number of patients in whom LBBB pacing cannot correct the underlying LBBB, like the case presented here. The inability to correct the underlying LBBB in our case may be related to the patient's previous aortic surgery and septal scar tissue, making deep penetration of the septum into the LBBA difficult. Therefore, the newly implanted 3830 lead is essentially the equivalent of a conventional pacing/sensing lead placed in the anteroseptal and basal position of the right ventricle with the helix inside the superficial septum, as confirmed by the superficial location of the lead tip in the RV basal septum on surface echocardiogram (Figure 3).

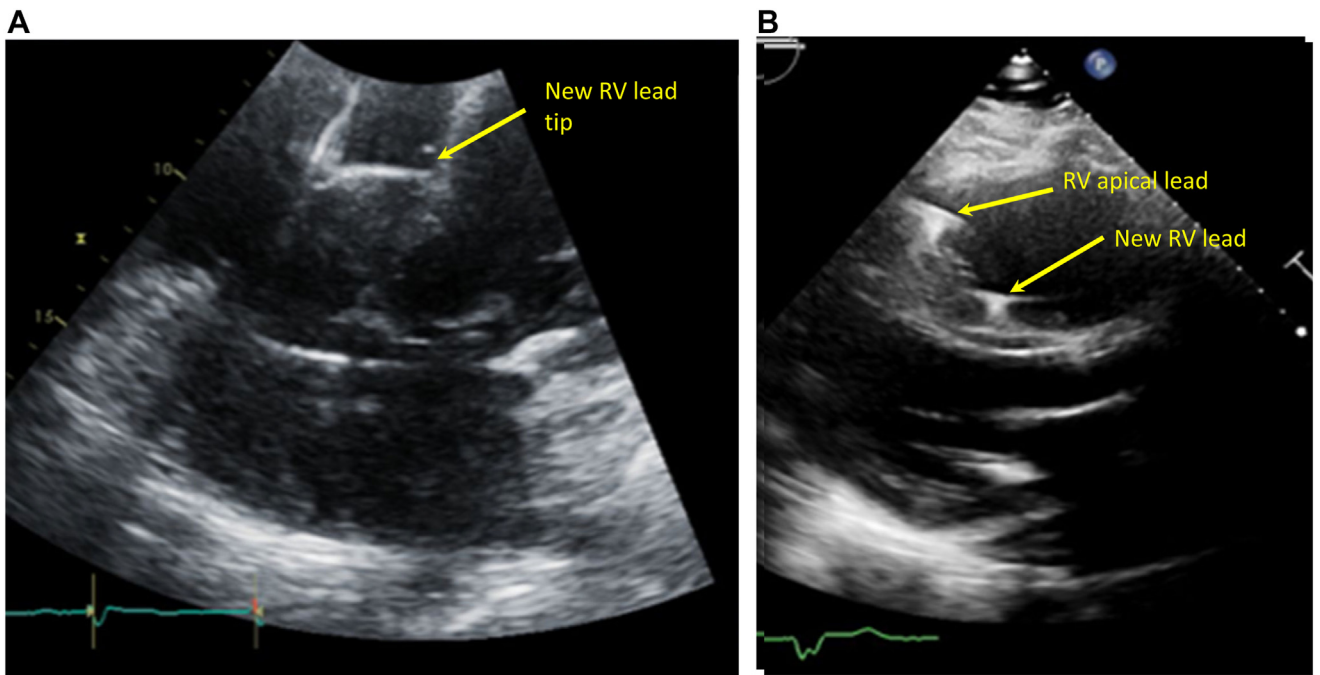
The exact mechanism underlying this synergy between the CS lead and the basal/septal RV pacing lead is not entirely clear, but the anatomical separation between the RV and CS leads may have played a critical role.<sup>6</sup> It is important to note that the CS lead was in a relatively apical and posteroseptal position instead of a more basal and lateral one, following the initial CRT-D implant. This suboptimal CS lead position was anatomically corrected by the apparently "failed" LBBB pacing lead placed in a more basal and anterior position by creating a wide separation between the 2 leads and a pacing vector that covers the majority of the LV myocardium (Figure 1C). In addition, RV apical pacing has been well known to cause worsening heart failure and increased mortality owing to the detrimental effect on the ventricular myocardium.<sup>7</sup> Relocating the RV lead from a more apical position to a more basal position may have mitigated that. In this regard, it is also interesting to note that the new adaptive CRT pacing algorithm, designed to avoid RV apical pacing, has demonstrated some promising results in clinical studies.<sup>8</sup> By relocating the RV pacing lead to the new basal and anterior position, away from the apex, the pacing vector between the CS and RV lead appears to cover a larger bulk of the left ventricle compared to the one between the CS and RV defibrillation leads (Figure 1C). This may have contributed to the observed CRT super-response.



**Figure 2** Marked QRS shortening and decreased ST deviation by simultaneous pacing of the right ventricular (RV) basal and coronary sinus (CS) leads (pole 2 to pole 3). The patient was in persistent atrial fibrillation in panels C and D. **A:** Native rhythm showing underlying left bundle branch block (LBBB) with QRS duration of 176 ms. **B:** Standard biventricular pacing produced minimal shortening of the QRS. Underlying rhythm was sinus with occasional atrial pacing. **C:** RV basal (and septal) pacing failed to correct the baseline LBBB or shorten the QRS. **D:** Simultaneous RV basal/septal and CS pacing led to improved electrical resynchronization and QRS shortening of 46 ms (from 176 ms to 130 ms).

In conclusion, our case demonstrated that RV basal pacing may be superior to apical pacing in certain clinical situations and positioning the RV pacing lead in a more basal (and anterior) position may improve CRT response. This contrasts with a previous study showing that RV lead position does

not seem to contribute significantly to CRT response, although the study was not designed to evaluate the difference between RV basal and apical pacing.<sup>9</sup> Future larger-scale studies are required to further confirm the findings in this case and help elucidate the underlying mechanism.



**Figure 3** Apical 4-chamber view (A) and parasternal long-axis view (B) of the new right ventricular (RV) basal/septal lead position. The new RV lead tip was seen located superficially over the septal and basal RV myocardium.

**Funding Sources:** This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

**Disclosures:** None.

## References

1. Linde C, Ellenbogen K, McAlister FA. Cardiac resynchronization therapy (CRT): clinical trials, guidelines, and target populations. *Heart Rhythm* 2012;9:S3–S13.
2. Jiang Z, Chang Q, Wu Y, Ji L, Zhou X, Shan Q. Typical BBB morphology and implantation depth of 3830 electrode predict QRS correction by left bundle branch area pacing. *Pacing Clin Electrophysiol* 2020;43:110–117.
3. Huang W, Su L, Wu S, et al. A novel pacing strategy with low and stable output: pacing the left bundle branch immediately beyond the conduction block. *Can J Cardiol* 2017;33:e1731–e1736.
4. Vijayaraman P, Subzposh FA, Naparkowski A, et al. Prospective evaluation of feasibility, electrophysiologic and echocardiographic characteristics of left bundle branch area pacing. *Heart Rhythm* 2019;16:1774–1782.
5. Zhang W, Huang J, Qi Y, et al. Cardiac resynchronization therapy by left bundle branch area pacing in heart failure patients with left bundle branch block. *Heart Rhythm* 2019;16:1783–1790.
6. Singh JP, Berger RD, Doshi RN, et al; ENHANCE CRT Study Group. Targeted left ventricular lead implantation strategy for non-left bundle branch block patients: the ENHANCE CRT study. *JACC Clin Electrophysiol* 2020;6:1171–1181.
7. Wilkoff BL, Cook JR, Epstein AE, et al. Dual Chamber and VVI Implantable Defibrillator Trial Investigators. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) Trial. *JAMA* 2002;288:3115–3123.
8. Singh JP, Cha YM, Lunati M, et al. Real-world behavior of CRT pacing using the AdaptivCRT algorithm on patient outcomes: effect on mortality and atrial fibrillation incidence. *J Cardiovasc Electrophysiol* 2020;31:825–833.
9. Leclercq C, Sadoul N, Mont L, et al; SEPTAL CRT Study Investigators. Comparison of right ventricular septal pacing and right ventricular apical pacing in patients receiving cardiac resynchronization therapy defibrillators: the SEPTAL CRT Study. *Eur Heart J* 2016;37:473–483.