

Cardiac resynchronization therapy using left ventricular septal pacing: An alternative to biventricular pacing?



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Introduction

Cardiac resynchronization therapy (CRT) has been established as a standard therapy for patients with heart failure (HF) and ventricular dyssynchrony. Previous prospective randomized studies have demonstrated that CRT not only improves quality of life and increases exercise capacity, but also reverses myocardial remodeling and decreases all-cause mortality.^{1,2} CRT typically is attempted with biventricular pacing (BiVP). We are presenting a case of dilated cardiomyopathy and left bundle branch block (LBBB) with significant improvement in electrical and mechanical dyssynchrony, HF symptoms, and left ventricular ejection fraction (LVEF) after initiation of left ventricular septal pacing (LVSP) therapy.

Case report

The patient was a 79-year-old man with a history of dilated cardiomyopathy and severely reduced left ventricular (LV) function, LBBB, and NYHA III functional status on optimal medical therapy. He underwent CRT pacemaker implantation 4 years ago and his HF symptoms improved thereafter. The patient was not followed up regularly until exertional dyspnea reappeared 3 months ago. CRT pacemaker interrogation showed loss of LV pacing capture owing to lead fracture, demonstrated by radiologic study (Figure 1A).

Given that the fractured LV lead had been implanted for 4 years, extracting the fractured lead and implanting a new one could be challenging. Alternative options such as surgical

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KEY TEACHING POINTS

- Biventricular pacing (BiVP) has some challenges, including the nonrespond, high pacing threshold, unavoidable phrenic nerve stimulation, and lack of targeted veins that limit the success of left ventricular pacing.
- His bundle pacing as an alternative to BiVP for cardiac resynchronization therapy has some deficiencies, such as difficulty in identifying the location of the His bundle and high capture threshold with potential long-term instability.
- Left ventricular septal pacing could be an alternative therapy to BiVP for patients with heart failure and ventricular dyssynchrony.

epicardial or endocardial LV lead placement could also be considered, but the patient refused. His bundle pacing (HBP) and LVSP were discussed in detail and the patient agreed to proceed.

Venous access was obtained in the left subclavian vein. A 7F preshaped guiding catheter (Model C315; Medtronic, Minneapolis, MN) was then inserted into the left subclavian vein over a guidewire. The fixed-screw ventricular lead, Select Secure model 3830 (Medtronic), was advanced through the sheath with its tip just beyond the distal part of the sheath for unipolar pacing and local activation potential recording. First, we tried the HBP (Figures 1B and 2A). Threshold for His bundle capture (Figure 2B) was 1.5 V @ 0.4 ms, while to correct LBBB and acquire a narrow QRS (Figure 2C), a higher output (≥ 5.5 V @ 0.4 ms or ≥ 3.5 V @ 1.0 ms) was required. Then, LVSP was attempted by advancing the sheath 1.5–2 cm towards the ventricle side from the His bundle region (Video 1). During the lead placement, the implanter repeatedly alternated between right

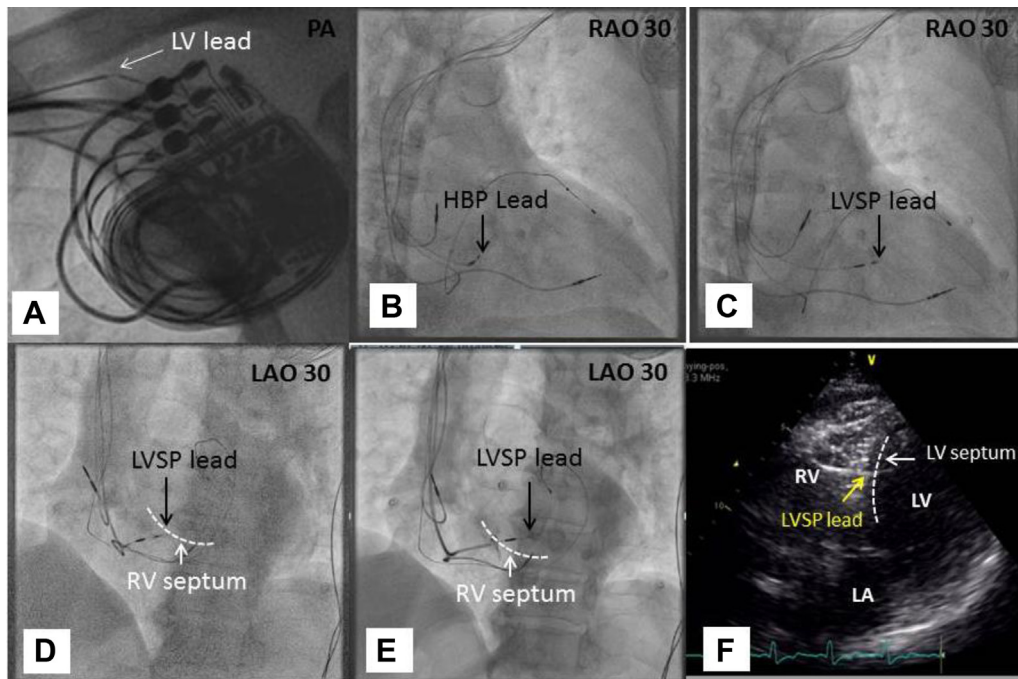


Figure 1 Images of the implantation procedure. **A:** Posteroanterior (PA) fluoroscopy showed the fractured part (*white arrow*) of the left ventricular (LV) lead. **B:** His bundle pacing (HBP) was performed in right anterior oblique (RAO) view; the lead tip (*black arrow*) was in the His bundle region. **C:** Left ventricular septal pacing (LVSP) was attempted by advancing the sheath 1.5–2 cm towards the ventricle side from the His bundle region. The LVSP lead (*black arrow*) was positioned perpendicularly against the right ventricular (RV) septum. **D:** Left anterior oblique (LAO) fluoroscopy showing the tip of the lead (*black arrow*) resting perpendicularly against the RV septum (*white arrow*) before being screwed in. **E:** While the lead was being rotated, repeated injections of contrast medium through the guiding catheter against the RV septum (*white arrow*) were used to assess the penetration depth. The part of the lead tip (*black arrow*) protruding into the RV septum was not covered by contrast medium. **F:** Ultrasonography 4-chamber view of the heart showed that the lead tip (*yellow arrow*) was on the LV septum (*white arrow*). LA = left atrium.

anterior oblique (Figure 1C) and left anterior oblique (LAO) (Figure 1D) view to ensure the tip of the lead perpendicularly against the right ventricular septum (RVS). The sheath and the lead touched the RVS and pacing with an output of 5.0 V / 0.5 ms was applied, which created electrocardiogram QRS morphology of LBBB pattern. The pacing lead was then screwed towards the left side of the septum with a force slightly greater than that used during right ventricular pacing lead placement. During rotation of the lead, the implanter repeatedly assessed the penetration depth in LAO view. Small amounts of contrast medium were injected through the guiding catheter against the interventricular septum under fluoroscopy in LAO view for depth estimation (Figure 1E, Video 2). In addition, pacing was repeatedly performed from the tip electrode while screwing the lead. Once electrocardiogram QRS morphology during pacing resulted in a pattern of right bundle branch conduction block (RBBB) (Figure 2D–F), the lead had been at or near the left side of the septum and the lead advancement was stopped. The development of an RBBB pattern is an important clue that the LBBB has been engaged. Pacing thresholds and impedances were measured to ensure that the helix did not protrude into the LV cavity. Finally, a transthoracic echocardiography ultrasound was used to verify the position of the lead tip on the LV septum (Figure 1F). Device testing showed that the LVSP threshold, sensing, and impedances were 0.4 V @ 0.4 ms, 15.5 mV, and 880 Ω , respectively. The LVSP

lead was connected to the LV port and DDD pacing mode (right atrial pacing and LVSP) was finally adopted. During LVSP, QRS duration was significantly shortened from 190 ms at baseline to 132 ms (Figure 2A and F).

At 3-month follow-up, cumulative percent LVSP was 99.6%. His HF symptoms were significantly improved from NYHA functional class III to class I, with 6-minute walk test from 352 m to 545 m, LV end-diastolic volume from 175 mL to 123 mL, and LVEF from 39% to 53%. Two-dimensional speckle tracking strain imaging demonstrated a significant improvement in overall ventricular longitudinal strain pattern and mechanical dispersion (defined as the standard deviation of contraction duration from Q/R on the electrocardiogram to peak strain in 17 LV segments) was less pronounced after CRT therapy (Figure 3A and B).

Discussion

CRT is a well-established therapy for patients with HF and ventricular dyssynchrony. CRT typically is attempted with BiVP. However, BiVP does have some challenges, including the nonrespond, high pacing threshold, unavoidable phrenic nerve stimulation, and lack of targeted veins that limit the success of LV pacing.

HBP has been evaluated as an alternative to BiVP with resynchronization of ventricular activation.^{3–6} However, challenges with HBP include difficulty in identifying the

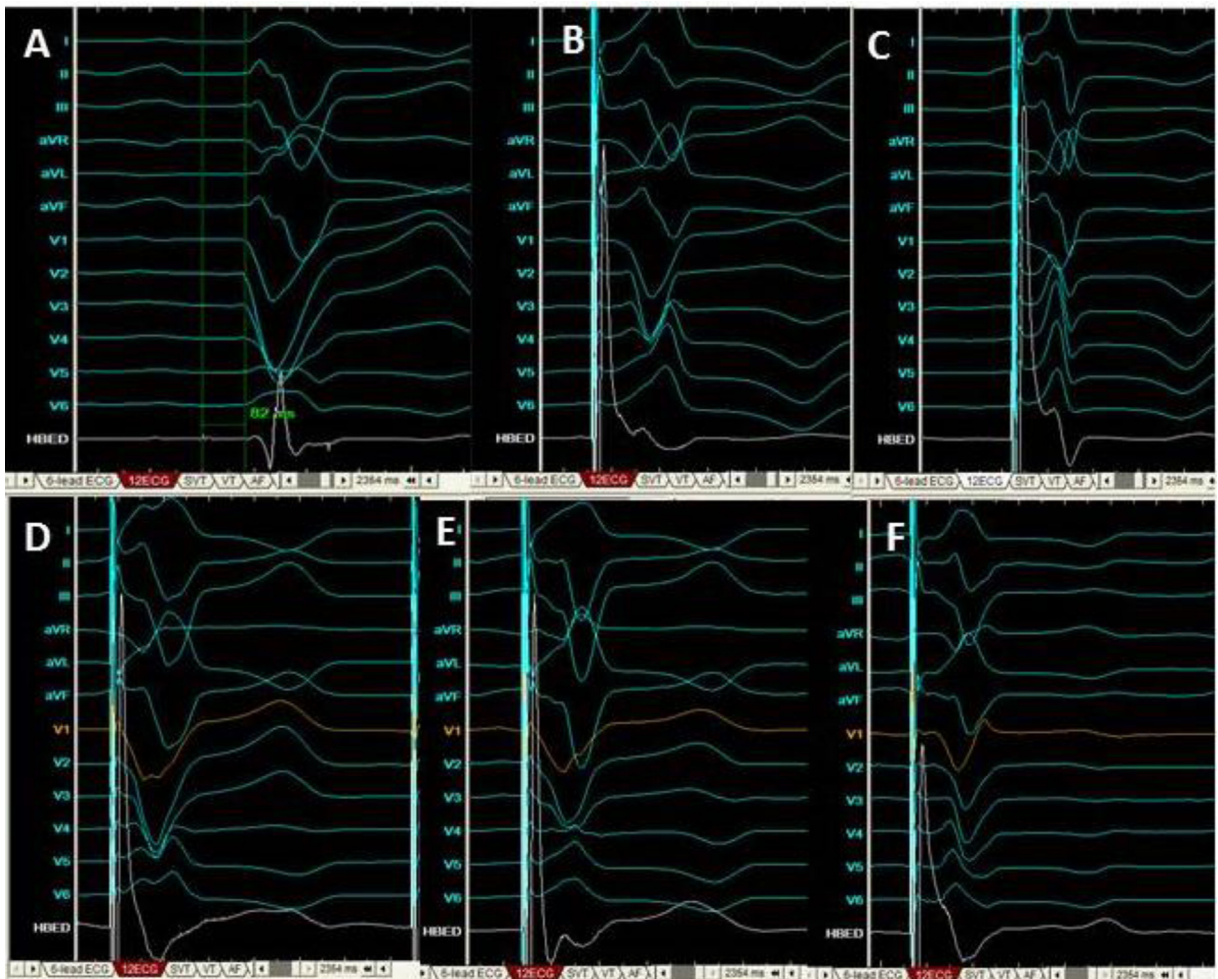


Figure 2 Electrocardiogram of the implantation procedure. **A:** The intrinsic rhythm was left bundle branch block (LBBB) and the HV interval was 82 ms. **B:** A lower output (≥ 1.5 V @ 0.4 ms) can capture the His bundle and adjacent ventricular tissues but cannot correct LBBB. **C:** A higher output (≥ 5.5 V @ 0.4 ms or ≥ 3.5 V @ 1.0 ms) can correct LBBB and acquire a narrow QRS. **D:** Right ventricular septum pacing QRS morphology before screwing the lead. **E:** Paced QRS morphology changed while screwing the lead. **F:** Paced QRS showed right bundle branch block morphology while the tip of the lead arrived at the left ventricular septum.

location of the His bundle and high capture threshold with potential long-term instability, especially in patients who have already developed cardiac conduction disease such as LBBB.⁷⁻¹⁰ As shown in this case, the threshold to capture the His bundle is acceptable, while the threshold to correct

LBBB and acquire a narrow QRS is too high, which restricts the clinical application of HBP in HF patients with dyssynchrony.

The feasibility of LVSP was first demonstrated in the animal experiment by Grosfeld and colleagues,¹¹ who used

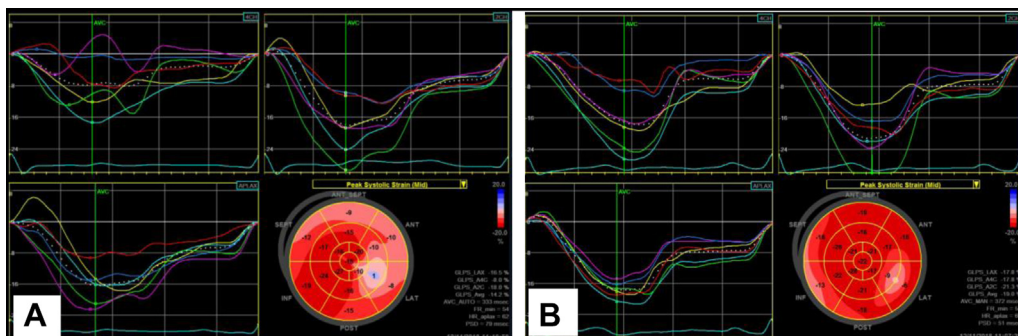


Figure 3 The offline analysis showing the regional and overall ventricular longitudinal strain pattern changes at baseline (**A**) and during 3-month follow-up (**B**). These regional curves show that the mechanical dispersion was improved during 3-month follow-up.

screw-in leads with a long insulated screw in goats. Then Mills and colleagues¹² achieved LVSP over a period of 16 weeks in 8 adult mongrel dogs using a custom pacing lead with extended helix. Mafi-Rad and colleagues¹³ revealed that LVSP reduces electric dyssynchrony and preserves acute LV pump function compared with right ventricular atrium and RVS pacing in 10 patients with sinus node dysfunction. They believed that electric activation of the working myocardium starts at the LV septal endocardium during normal ventricular conduction, which may explain why LVSP maintains a closer-to-normal electric activation. Huang and colleagues¹⁴ reported a case of LBBB corrected by LVSP, and speculated that LVSP can correct electric dyssynchrony because the pacing activation captures the left bundle branch.

In this case, we performed LVSP on the patient who failed with CRT because of LV lead fracture. The QRS duration at LVSP is not only shorter than intrinsic rhythm (LBBB), but also shorter than BiVP (QRS duration = 168 ms), indicating that LVSP may result in a better electric resynchronization. At the 3-month follow-up, the mechanical dispersion based on speckle tracking strain imaging was significantly improved, and the patient's HF symptoms, LV end-diastolic volume, and LVEF were also improved, denoting that this new pacing method may also restore mechanical synchronization and even reverse myocardial remodeling.

Several questions remain regarding LVSP as a viable option for CRT. Firstly, although pacing parameters including threshold, sensing, and impedances were stable during the 3-month follow-up, and no lead-related complication (dislocation, perforation, thromboembolism, etc) was observed, a long-term follow-up on the safety is needed. Secondly, this case notes LVSP is effective in restoring electric and mechanical resynchronization. More data are required to determine whether the LVSP's effectiveness is unusual or common.

Conclusion

LVSP therapy could be an alternative therapy to BiVP for patients with HF and ventricular dyssynchrony in select situations where effective BiVP is not achievable. More studies are needed to validate the effectiveness and safety of LVSP.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2019.03.011>.

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