Left bundle branch pacing with dynamic retrograde His bundle potential and intracardiac isoelectric interval: A case report

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Introduction

Physiological pacing has gained growing popularity in the past years because it mitigates the various deleterious effects of right ventricular (RV) pacing.[1] While His bundle pacing (HBP) is arguably the most physiological form of pacing, it has limitations including low sensed R-wave amplitude and high capture threshold. In 2017, Huang and colleagues[2] first reported the feasibility of left bundle branch pacing (LBBP). LBBP provides low and stable threshold compared to HBP and corrects distal conduction system disease. Here, we report 2 patients with atrial fibrillation combined with complete right bundle branch block (RBBB) and left bundle branch block (LBBB), respectively. Both patients received HBP and LBBP. We present what is, to our knowledge, the first cases in which retrograde His bundle potential with varying S-H intervals was observed during transseptal placement of the pacing lead using John Jiang's connecting cable. Unlike traditional connecting cable, which has to be disconnected while the pacing lead was screwed into the septum, John Jiang's connecting cable allows simultaneous monitoring and recording of electrocardiogram and intracardiac electrogram during lead deployment. Moreover, selective LBBP (S-LBBP) and nonselective LBBP (NS-LBBP) with vastly different stimulus-to–left ventricle activation time (Stim-LVAT) was noted during threshold testing. Distinct isoelectric stimulus-QRS interval was recorded in intracardiac electrogram during S-LBBP.

KEYWORDS Conduction system pacing; Intracardiac electrogram; Isoelectric interval; Left bundle branch pacing; Retrograde His bundle potential
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KEY TEACHING POINTS

- Retrograde His bundle activation occurs transseptally during left bundle branch pacing.
- Distinct isoelectric interval in intracardiac electrogram indicates selective left bundle branch pacing.
- Distinct isoelectric segment accounts for longer stimulus-to–left ventricle activation time during selective left bundle branch pacing.

Case report

Case 1

A 67-year-old man presented with symptoms of dizziness for 1 month. Electrocardiogram revealed atrial fibrillation, complete RBBB, and a QRS duration of 144 ms. Holter monitoring revealed 4211 long R-R intervals greater than 2 seconds with the longest R-R interval of 4.4 seconds. He was indicated for permanent DDD pacemaker implantation with HBP and LBBP. HBP was attempted first. The pacing lead (SelectSecure, Model 3830; Medtronic, Minneapolis, MN) was successfully implanted; the capture threshold was 1.0 V. Four attempts of LBBP were made using a second pacing lead. At all 4 initial implantation sites (RV septum), retrograde His bundle potentials with prolonged stimulus-to–His bundle potential interval (S-H interval) were recorded. For the final and successful attempt of LBBP, electrocardiograms and intracardiac electrograms were continuously recorded while the pacing lead advanced from the RV septum to the left ventricular septal subendocardium under the pacing output of 2 V / 0.5 ms using John Jiang's connecting cable. Retrograde His bundle potential was observed and recorded, and the S-H gradually decreased from 82 ms to 43 ms (Figure 1A). Multiple impendence measurements (in which the pacing output increased to 5 V / 0.5 ms) were performed as the lead was screwed into the

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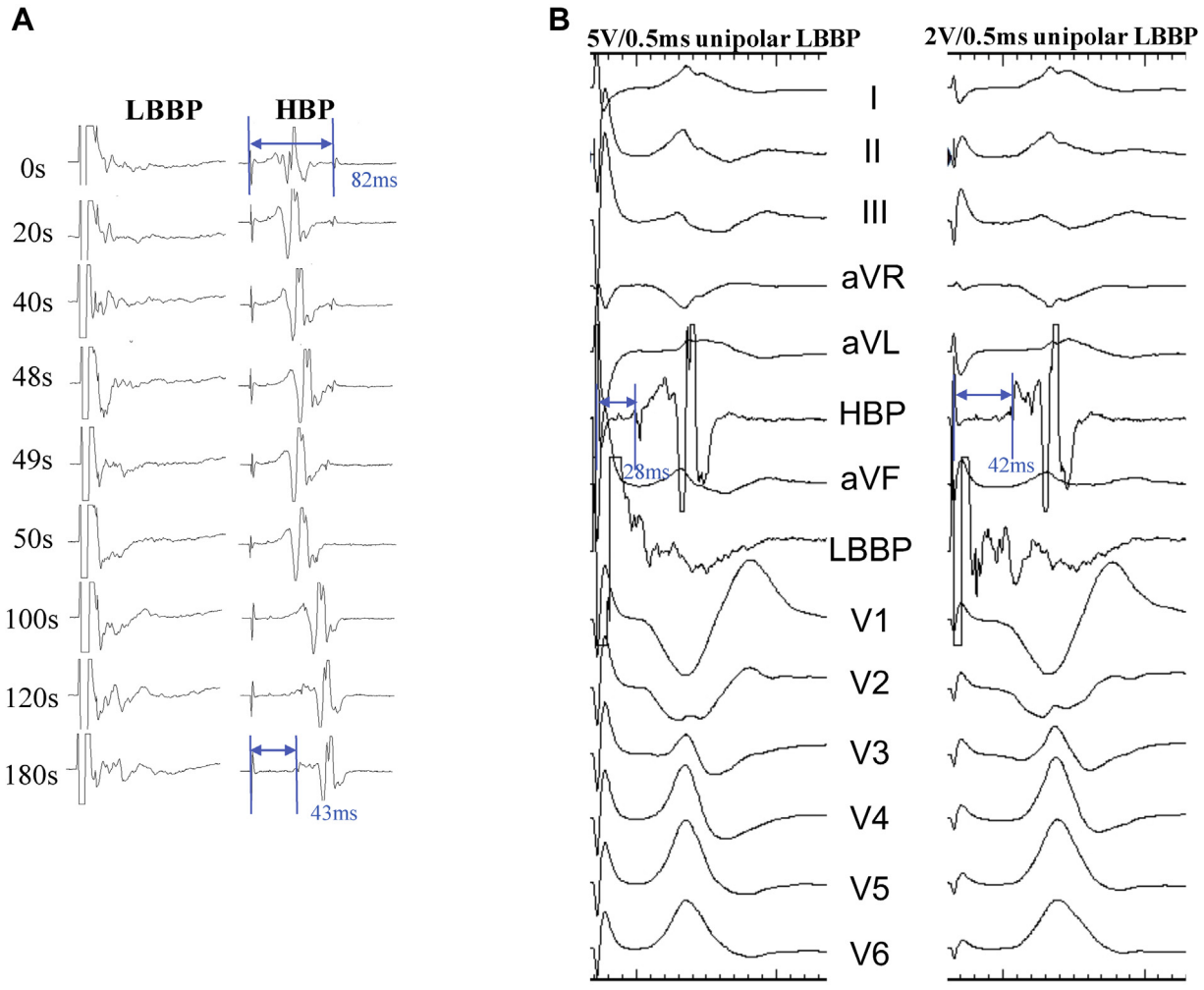

Figure 1 Stimulus-to–His bundle potential (HBP) interval dynamic in right bundle branch block patient during left bundle branch pacing (LBBP). **A:** Gradual decrease in stimulus-to-HBP interval as the tip of the LBBP lead was screwed deep into the septum during unipolar pacing at 2 V / 0.5 ms. **B:** Stimulus-to HBP interval under the pacing output of 5 V / 0.5 ms and 2 V / 0.5 ms during unipolar LBBP at the lead fixation site: 28 ms vs 42 ms.

ventricular septum. When comparing 2 adjacent S-H intervals under the pacing output of 5 V / 0.5 ms and 2 V / 0.5 ms, shortening of S-H interval was recorded. Notably, as the pacing lead approached the left bundle branch (LBB) region, S-H intervals under 2 V / 0.5 ms and 5 V / 0.5 ms were consistently 42 ms and 28 ms, respectively (Figure 1B). Finally, the lead was fixed 16 mm deep. Unipolar pacing at the location demonstrated RBBB morphology with a capture threshold of 0.5 V. R-wave amplitude was 8.8 mV, and impendence was 927 Ω.

Case 2

A 77-year-old man presented with symptoms of shortness of breath and fatigue for 1 week. Electrocardiogram revealed atrial fibrillation, high-grade atrioventricular block, and LBBB. DDD pacemaker implantation with HBP and LBBP were attempted. HBP lead was successfully implanted with a capture threshold of 1.5 V. During transseptal placement of the LBBP lead, retrograde His bundle potential was initially not observed. The first observed His bundle potential had an S-H interval of 24 ms, and it gradually increased to 44 ms at the site of lead fixation (Figure 2A). Then, S-H interval was tested under different pacing outputs. The interval was 53 ms under low pacing output of 1 V / 0.5 ms, and it shortened to 35 ms when the output increased to 8 V / 0.5 ms (Figure 2B).

The lead was placed 15 mm deep. LBB potential with LBB-V interval of 25 ms was recorded. R-wave amplitude was 10.2 mV, and impendence was 689 Ω. Unipolar pacing at the location demonstrated RBBB morphology. During threshold testing, changes in QRS morphology were observed at the pacing output of 1.5 V / 0.5 ms. Loss of capture occurred at 0.6 V / 0.5 ms. Notably, distinct isoelectric stimulus-QRS interval (S-V interval) was observed in intracardiac electrogram during 0.6 V / 0.5 ms pacing (Figure 3A). The pacing Stim-LVAT at 2.0 V / 0.5 ms (before QRS morphology change) was 81 ms, while the Stim-LVAT increased to 95 ms at 1 V / 0.5 ms (after QRS morphology change) (Figure 3B). At 0.6 V / 0.5 ms, the Stim-LVAT was 97 ms (Figure 3A). At 15-day follow-up, the changes in QRS morphology occurred at the output of 1.25 V / 0.4

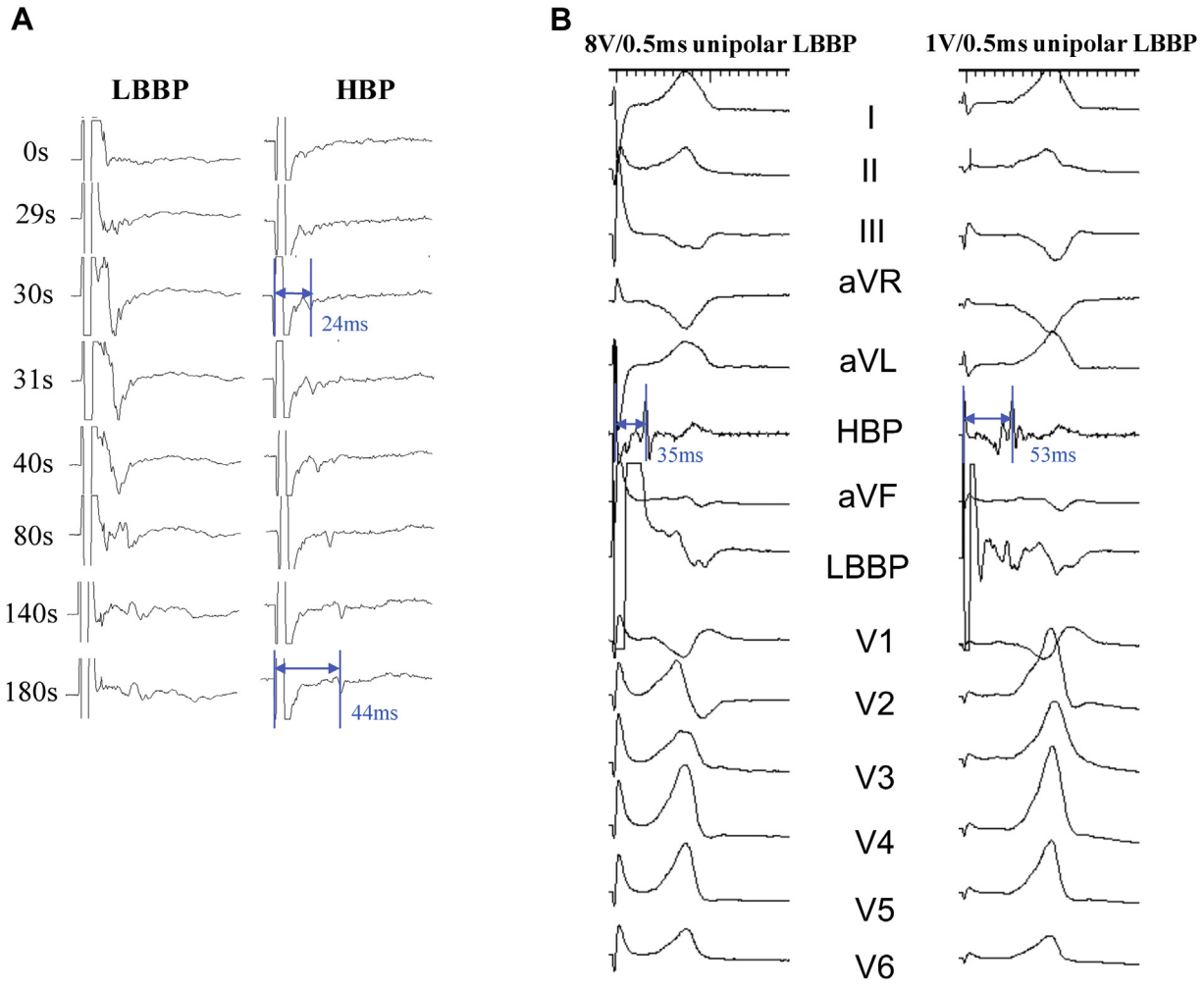

Figure 2 Stimulus-to–His bundle potential (HBP) interval dynamic in left bundle branch block patient during left bundle branch pacing (LBBP). **A:** Gradual increase in stimulus-to-HBP interval as the tip of the LBBP lead was screwed deep into the septum during unipolar pacing at 2 V / 0.5 ms. **B:** Stimulus-to-HBP interval under the pacing output of 8 V / 0.5 ms and 1 V / 0.5 ms during unipolar LBBP at the lead fixation site: 35 vs 53 ms.

ms, whereas loss of capture occurred at 0.75 V / 0.4 ms. The Stim-LVAT at 1 V / 0.4 ms was longer than that at 2 V / 0.4 ms: 105 ms vs 86 ms.

Discussion

In both case 1 and case 2, retrograde His bundle potential was observed and recorded during simultaneous transseptal placement of the pacing lead. In the first case, the initial retrograde His bundle activation delay and the gradual shortening of S-H interval can be attributed to complete RBBB suffered by the patient. His bundle activation delay at the RV septum occurred because His bundle was activated only after transseptal conduction and retrograde capture of LBB. As the pacing lead advanced into the LBB region, the electrical wavefront captured septal tissues that were in closer proximity to LBB and eventually captured LBB directly, resulting in gradual shortening of S-H interval (Figure 1A). Similarly, the lengthening of S-H interval in the second case can be attributed to LBBB. Unlike in the first case, His bundle potential was initially not observed in this patient. We hypothesize that the His bundle potential was initially hidden in the pacing spike, as the S-H interval for direct His bundle–right bundle branch (RBB) capture was extremely short. The first observed His bundle potential had S-H interval of 24 ms, indicating immediate His bundle–RBB capture (Figure 2A). As the pacing lead approached the LBB, His bundle activation occurred via retrograde capture of RBB, resulting in longer S-H interval (Figure 2A). Retrograde His bundle activation occurs either transseptally or via peripheral activation of the Purkinje system far from the actual pacing site.[3,4] The 2 cases we report demonstrated transseptal activation of His bundle as S-H interval changed gradually as the pacing lead advanced from the right to left side of the septum. Moreover, S-H interval differed under high and low pacing outputs. S-H interval was shorter under high pacing outputs because the electrical wavefront captured bundle branches or septal tissues that were in closer proximity to bundle branches (Figure 1B and Figure 2B), indicating transseptal activation of His bundle.

In the second case, output-dependent changes in QRS morphology with RBBB pattern occurred during threshold

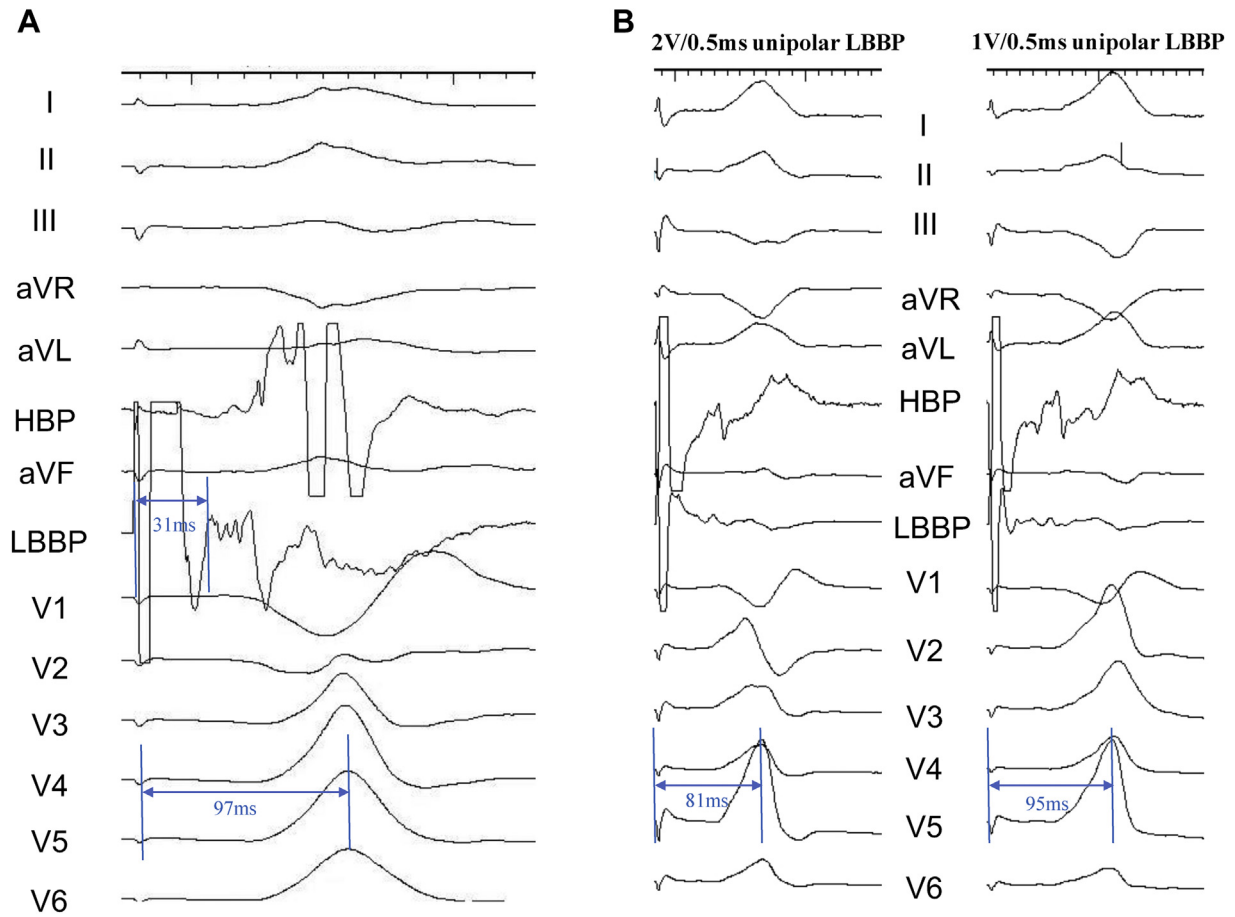

Figure 3 Selective and nonselective left bundle branch pacing (LBBP). **A:** Intracardiac isoelectric stimulus-QRS interval (S-V interval) at 0.6 V / 0.5 ms during unipolar LBBP at the lead fixation site. S-V interval was 31 ms. Stimulus to left ventricular activation time (Stim-LVAT) was 97 ms. **B:** Unipolar LBBP under 1 V / 0.5 ms and 2 V / 0.5 ms exhibited significant QRS morphology difference. Moreover, unipolar LBBP under 2 V / 0.5 ms at the lead fixation site revealed Stim-LVAT of 81 ms, while unipolar LBBP under 1 V / 0.5 ms revealed Stim-LVAT of 95 ms.

testing. Distinct isoelectric stimulus-QRS interval (S-V interval) at low pacing output is one of the characteristics of selective LBB capture.[1] In this case, S-V interval was observed in intracardiac electrogram under the pacing output of 0.6 V / 0.5 ms. (Figure 3A), therefore suggesting that the capture threshold of LBB is lower than that of myocardium. Interestingly, Stim-LVAT differed significantly between NS-LBBP and S-LBBP: Stim-LVAT of NS-LBBP was shorter than that of S-LBBP. The longer Stim-LVAT during S-LBBP can be attributed to the isoelectric stimulus-QRS interval, which represents the recruitment of LBB/Purkinje fibers. Specifically, the intracardiac S-V interval observed during S-LBBP was 31 ms, while the interval was not present during NS-LBBP. Delta wave was instead observed in electrocardiogram during NS-LBBP, indicating fusion capture of local septal myocardium and the LBB. Therefore, the difference between Stim-LVAT during S-LBBP and NS-LBBP can be largely explained by S-V interval. Abrupt shortening of Stim-LVAT at high pacing output or short and constant Stim-LVAT at both low and high outputs indicates LBB capture.[1] However, our case proposes that distinct isoelectric stimulus-QRS interval and lengthening of Stim-LVAT at low pacing output may suggest selective LBBP.

Upadhyay and colleagues[5] have reported that while the distal-to-proximal activation of the ventricular component was preserved during selective HPB, the physiological ventricular activation was not observed during nonselective HBP. Therefore, the short Stim-LVAT during NS-LBBP may similarly suggest nonphysiological ventricular activation. In the case we reported, S-LBBP occurred only at low pacing outputs below the capture threshold of myocardium, and slightly higher voltages resulted in simultaneous capture of both LBB and myocardium. Therefore, the case raises the question whether left ventricular hemodynamics will be perturbed under outputs above the thresholds of both LBB and myocardium, since the higher voltages result in nonphysiological fusion capture.

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