

CASE REPORT

INTERMEDIATE

CLINICAL CASE

Left Bundle Branch Conduction Recovery Following Left Bundle Branch Pacing in a Heart Failure Patient



Weiwei Zhang, MD,^a Jingjuan Huang, MD, PhD,^a Yiding Qi, MS,^a Xuerui Shi, MS,^a Fei Wang, MS,^a Xiaohong Zhou, MD,^b Ruogu Li, MD, PhD^a

ABSTRACT

This report presents the application of left bundle branch pacing as a cardiac resynchronization therapy in a patient with systolic heart failure and complete left bundle branch block. At the 3-month follow-up, the patient had significant improvement in cardiac function accompanied by the recovery of left bundle branch conduction. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2019;1:592-6) © 2019 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Left bundle branch pacing (LBBP) has recently been proposed to treat left bundle branch block (LBBB) by bypassing the blocked conduction region (1,2). With a low capture threshold, LBBP can correct LBBB and generate a narrow electrocardiogram (ECG) QRS duration and fast left ventricular activation (1-3). LBBP therefore represents a novel promising strategy for cardiac resynchronization therapy (CRT). This report presents the application of LBBP in a patient with systolic heart failure and complete LBBB. At 3-month follow-up, the patient

had significant improvement in cardiac function accompanied by the recovery of left bundle branch conduction during intrinsic rhythm.

HISTORY OF PRESENTATION

A 72-year-old man with a 1-year history of exertional shortness of breath and fatigue was hospitalized twice over a 6-month period due to worsening paroxysmal nocturnal dyspnea and lower limb edema. Coronary artery disease was excluded by using computed tomography angiography. ECGs performed during both admissions showed sinus rhythm and complete LBBB. Echocardiography revealed left ventricular dilation and reduced left ventricular ejection fraction (LVEF).

MEDICAL HISTORY

The patient's medical history included chronic bronchitis and hypertension. He had no history of

LEARNING OBJECTIVES

- LBBP that is applied at a site distal to the LBBB region can directly correct LBBB and is one type of CRT.
- Recovery of left bundle branch conduction can occur following the improvement in cardiac function and ventricular reverse remodeling in heart failure patients with LBBB.

From the ^aDepartment of Cardiology, Shanghai Chest Hospital, Shanghai Jiao Tong University, Shanghai, China; and the ^bCRHF Division, Medtronic plc, Mounds View, Minnesota. This work was supported by Science and Technology Commission of Shanghai Municipality, China (grant number 17411971000). Dr. Zhou is an employee of Medtronic, Inc. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

Informed consent was obtained for this case.

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smoking or alcohol consumption, and there was no family history of cardiac disease.

Current medications included perindopril 8 mg once daily, bisoprolol 5 mg once daily, spironolactone 20 mg once daily, and furosemide 20 mg once daily, and compound methoxyphenamine capsules (2 tablets 3 times a day).

The patient's blood pressure at admission was 112/70 mm Hg, and physical examination showed inferior lung rales and lower limb edema.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses include dilated cardiomyopathy, ischemic cardiomyopathy, valvular heart disease, and hypertensive cardiomyopathy.

INVESTIGATIONS

Echocardiography revealed an enlarged left atria and ventricle (left atrial diameter 45 mm; left ventricular end-systolic diameter 49 mm; and left ventricular end-diastolic diameter 65 mm), interventricular septal thickness of 11 mm, left ventricular posterior wall thickness of 11 mm, and reduced LVEF (38%); moderate mitral regurgitation; and congenital bicuspid aortic valve with mild aortic valve stenosis. ECG revealed sinus rhythm, with complete LBBB with a QRS duration of 180 ms (Figure 1). The patient's

B-type natriuretic peptide concentration was 1,023 pg/ml.

Based on the history of hypertension, documented cardiac hypertrophy by echocardiography, and clinical cardiac function assessment, hypertensive cardiomyopathy with New York Heart Association functional class III was diagnosed.

MANAGEMENT

With a QRS duration ≥ 150 ms, ECG morphology of complete LBBB, and LVEF of 38%, and considering that optimal drug treatment had been applied for >3 months, CRT with LBBP was recommended. Written consent was obtained from the patient.

A CRT-pacemaker (C5TR01, Medtronic Inc., Minneapolis, Minnesota) was implanted with no procedural complications. The atrial pacing lead (model 5076, Medtronic Inc.) and the left ventricular pacing lead (model 4196, Medtronic Inc.) were placed in the right atrium and left ventricular epicardial lateral vein, respectively. A SelectSecure lead (model 3830, Medtronic Inc.) was then implanted into the left bundle branch area via the trans-septal approach under fluoroscopic imaging, right anterior oblique 30° (4,5) (Figure 2A). The unipolar pacing at 1 V/0.4 ms

ABBREVIATIONS AND ACRONYMS

- BNP** = B-type natriuretic peptide
- CRT** = cardiac resynchronization therapy
- ECG** = electrocardiogram
- LBBB** = left bundle branch block
- LBBP** = left bundle branch pacing
- LVAT** = left ventricular activation time
- LVEF** = left ventricular ejection fraction

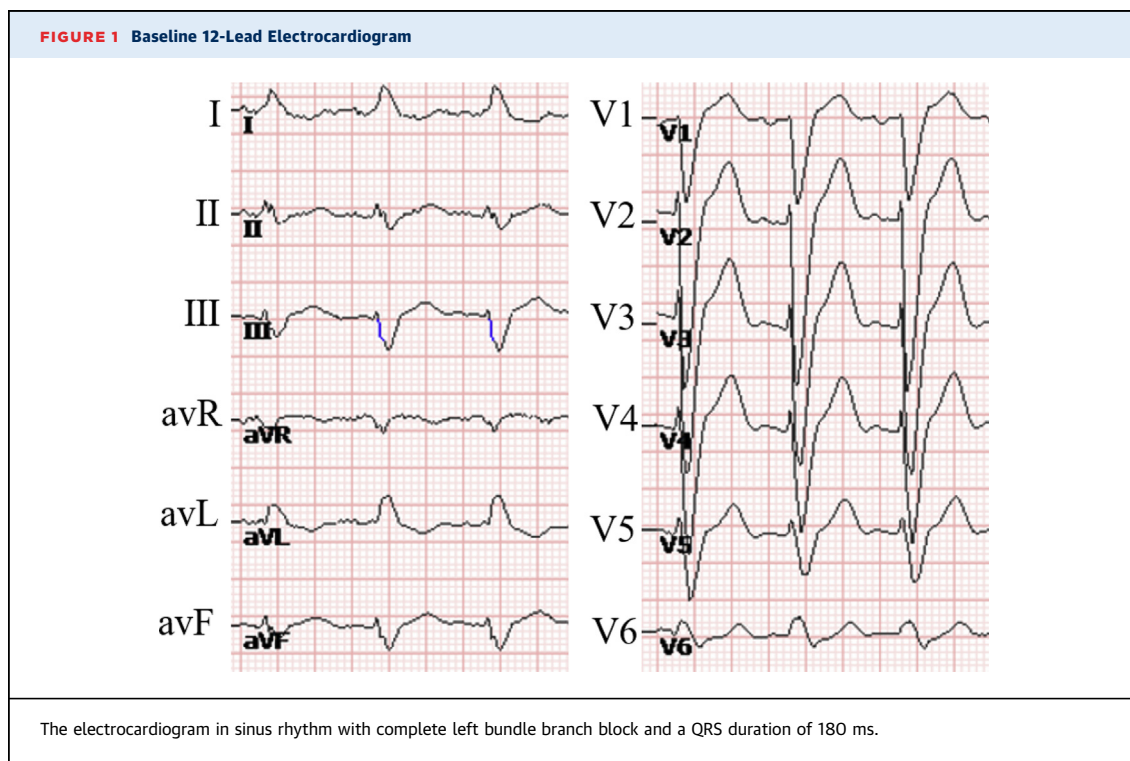
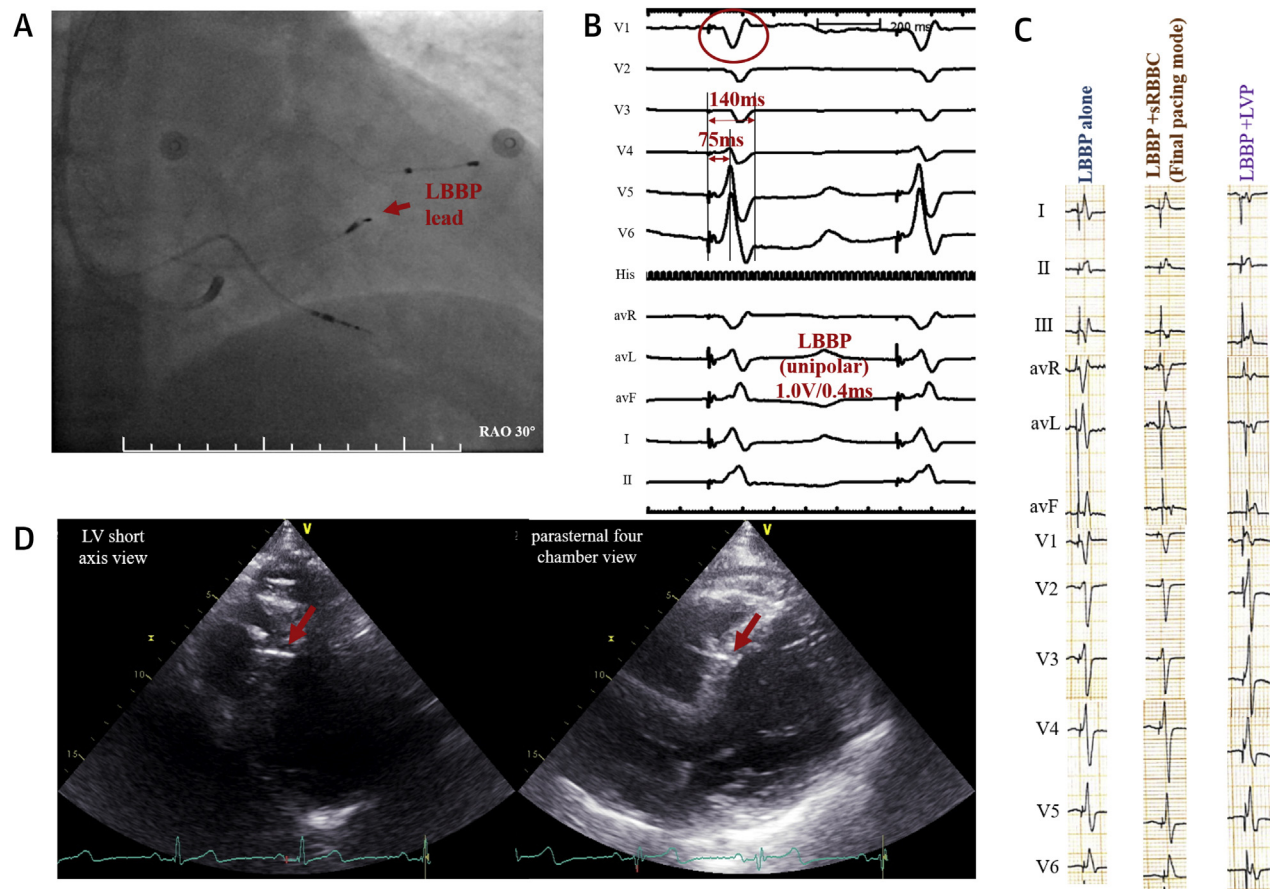


FIGURE 2 12-Lead ECG and Imaging Records at Implantation and Post-Implantation

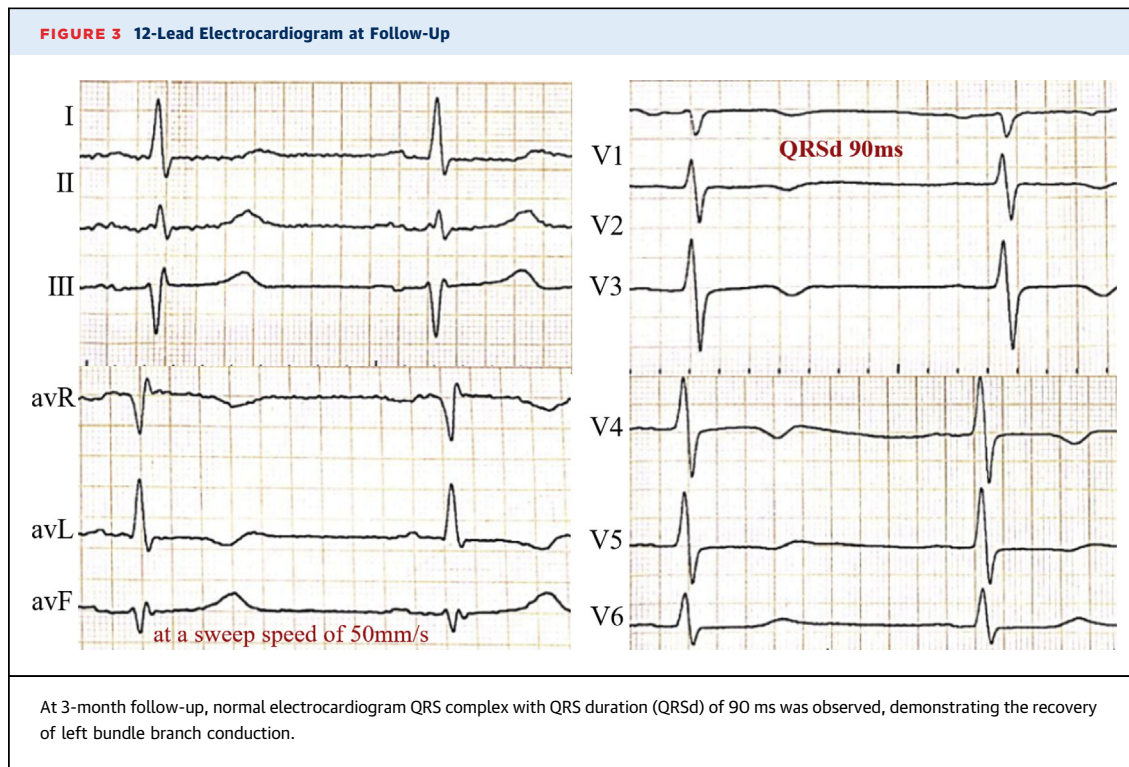
(A) Location of left bundle branch pacing (LBBP) lead under the right anterior oblique (RAO) 30° fluoroscopic image (arrow for the pacing lead tip). **(B)** Twelve-lead electrocardiogram (ECG) during unipolar LBBP (1 V/0.4 ms), right bundle branch block morphology (circle in ECG lead V₁) was observed with a left ventricular activation time (LVAT) of 75 ms in ECG leads V₅ and V₆ and a QRS duration (QRSd) of 140 ms. **(C)** Post-implantation, 12-lead ECG was performed in different pacing modes, including LBBP alone (QRSd, 140 ms; LVAT, 75 ms), LBBP synchronized with intrinsic right bundle branch conduction (LBBP + sRBBC) with atrioventricular delay 100/130 ms (QRSd, 130 ms; LVAT, 75 ms), and LBBP combined with left ventricular epicardial pacing (LBBAP + LVP) with VV delay 0 ms (QRSd, 130 ms; LVAT, 75 ms). **(D)** Post-implantation, echocardiography identified the pacing lead tip on the left side of the ventricular septum near the left ventricular subendocardium (arrows).

corrected LBBB with ECG QRS duration of 140 ms (from the stimulus to the end of the QRS complex) and left ventricular activation time (LVAT) of 75 ms in ECG leads V₅ and V₆ (Figure 2B). The pacing lead was then fixed and inserted to the right ventricular IS-1 connector port. Post-implantation, a 12-lead ECG was performed to choose the final pacing mode. The atrioventricular delay was set at 100/130 ms to achieve LBBP synchronized with intrinsic right bundle branch conduction with a paced QRS duration of 130 ms and paced LVAT of 75 ms. Because paced QRS duration and LVAT could not be shortened by LBBP combined with left ventricular epicardial pacing, LBBP synchronized with intrinsic right bundle branch

conduction was selected as the final pacing mode (Figure 2C). In post-implantation echocardiographic assessment, the pacing lead tip was identified on the left side of the basal interventricular septum and near the left ventricular subendocardium (Figure 2D).

DISCUSSION

As shown in this case, LBBP was able to bypass the blocked region and correct LBBB. Ventricular activation, especially left ventricular activation, was also better synchronized, with a QRS duration of 140 ms and an LVAT of 75 ms. Moreover, LBBP in fusion with intrinsic right ventricular activation by programming



atrioventricular delay 100/130 ms yielded a further narrowed QRS duration (130 ms).

Another finding of this case is that CRT via LBBP led to significant improvement in cardiac function and reverse ventricular remodeling. During LBBP synchronized with intrinsic right bundle branch conduction, QRS morphology was approximately normal according to ECG recording. The battery energy of the device is also saved by avoiding left ventricular epicardial pacing whose capture threshold is 1.5 V/0.4 ms.

LBBB is common in systolic heart failure, and ventricular dyssynchrony caused by LBBB is believed to exacerbate left ventricular impairment. It is the major finding of this case that the conduction of previously blocked left bundle branch was fully recovered after 3-month LBBP, suggesting functional rather than organic injury in the left bundle branch. One possible mechanism is that because heart failure was alleviated and left ventricular end-diastolic diameter was reduced, mechanical stretch in the left bundle branch region was relieved, resulting in the recovery of left bundle branch conduction function.

FOLLOW-UP

At the 3-month follow-up, the patient's cardiac function had improved significantly, with New York

Heart Association functional class improving from III to I and B-type natriuretic peptide levels decreasing from 1,023 pg/ml to 95 pg/ml. Echocardiography also revealed an improved LVEF, from 38% to 58%, along with reduced left ventricular end-diastolic diameter from 65 mm to 48 mm. When CRT-pacing was suspended during the device interrogation, 12-lead ECG showed normal QRS morphology with a QRS duration of 90 ms during intrinsic rhythm (Figure 3), demonstrating full recovery of native activation conduction via the left bundle branch.

CONCLUSIONS

This case showed that LBBP with a low capture threshold was able to correct LBBB and restore cardiac synchronization. Three months after LBBP, significant alleviation of heart failure, reversed ventricular remodeling, and recovered left bundle branch conduction were observed. LBBP is a promising novel approach for CRT.

ADDRESS FOR CORRESPONDENCE: Dr. Ruogu Li, Department of Cardiology, Shanghai Chest Hospital, Shanghai Jiao Tong University, No. 241 West Huaihai Road, Xuhui District, Shanghai 200030, China. E-mail: 13564565961@163.com.

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KEY WORDS cardiac pacemaker, cardiac resynchronization therapy, systolic heart failure