Reversal of left bundle branch block–induced cardiomyopathy by His bundle pacing

Fei Liu¹, Lijun Zeng², Xiaomeng Yin¹, Lianjun Gao¹, Yunlong Xia¹,* and Yingxue Dong¹,*

Abstract
A 61-year-old woman was referred to our institution for evaluation of severe nonischemic dilated cardiomyopathy and left bundle branch block (LBBB). After permanent His bundle pacing, the LBBB was immediately corrected; however, the right bundle branch was injured during the procedure. Subsequent recovery of the right bundle branch block and normalization of heart function were observed during follow-up. This case indicates that LBBB might result in the development of nonischemic cardiomyopathy and emphasizes the necessity of a temporary pacemaker during His bundle pacing for patients with LBBB.

Keywords
Left bundle branch block, cardiomyopathy, His bundle pacing, right bundle branch injury, heart failure, temporary pacemaker

Date received: 28 July 2019; accepted: 1 October 2019

Introduction
Whether left bundle branch block (LBBB) is a cause or consequence of deterioration of left ventricular function is difficult to determine because both conditions are often identified concomitantly in clinical practice. We herein describe the correction of LBBB by His bundle pacing (HBP) and...
subsequent normalization of a low left ventricular ejection fraction (LVEF) in a patient previously diagnosed with dilated cardiomyopathy and LBBB. The right bundle branch (RBB) was injured during the procedure and recovered during follow-up. The evidence of LBBB-induced cardiomyopathy and possible causes of RBB block (RBBB) are discussed.

Case report

A 61-year-old woman presented with a 4-year history of severe dyspnea and bilateral pedal edema. She had repeatedly been admitted to a local hospital for dilated cardiomyopathy with heart failure. The LVEF had fluctuated between 18% and 28%. An electrocardiogram (ECG) and the results of 24-hour Holter monitoring, recorded at admission, showed complete LBBB with a QRS duration of 172 ms (Figure 1(a)). Echocardiography revealed a left ventricular end-diastolic diameter (LVEDD) of 69 mm, severe left ventricular dysfunction (LVEF of 15%), and decreased left ventricular wall motion. Computed tomography–coronary angiography

Figure 1. Twelve-lead electrocardiogram and intracardiac electrogram recorded by the 3830 lead. (a) Electrocardiogram at admission showed sinus rhythm with complete left bundle branch block and QRS duration of 172 ms (57 beats per minute). (b) The intracardiac electrogram showed a 2:1 atrioventricular block (red arrows indicate the P wave and blue arrows indicate the QRS complex). (c) The intracardiac electrogram revealed His bundle–ventricular block.
showed no evidence of coronary artery disease. Intensive medical treatment was administered to the patient but was ineffective. Therefore, we decided to attempt HBP with an aim to recruit the left bundle branch (LBB) and restore synchronization.

A SelectSecure 3830 lead (Medtronic, Inc., Minneapolis, MN, USA) was placed through a C315 sheath (Medtronic, Inc.) and guided to the atrioventricular septum. During His bundle mapping, 2:1 atrioventricular conduction occurred (Figure 1(b)), and an intracardiac electrogram showed His bundle–ventricular (HV) block (Figure 1(c)). A temporary pacemaker was inserted into the right ventricle to avoid a progressive atrioventricular block. At a low pacing frequency of about 60 beats per minute, HBP corrected the LBBB and decreased the QRS duration to 120 ms with an acute threshold of 1.0 v/0.4 ms. However, the QRS morphology presented RBBB even with a higher voltage and pulse width during the procedure. The unipolar and bipolar impedance was 886 and 593 $\Omega$, respectively. We managed to screw the lead in the exact site (Figure 2(a), (b)). The post-procedural ECG showed RBBB (Figure 3(a)). The symptoms of heart failure had been reversed by the 1-month post-procedure follow-up. A repeat ECG showed recovery of the RBB injury with rate-dependent RBBB (Figure 3(b)), which may have existed before HBP although the results of the 24-hour Holter monitoring, recorded at admission, did not show rate-dependent RBBB. Repeat echocardiography revealed a decreased LVEDD of 53 mm and an improved LVEF of 38%. The patient showed significant improvements 3 months later: the LVEDD had decreased to 51 mm and the LVEF had increased to 50%.

**Discussion**

The present case suggests that HBP offers a more physiological means and can significantly improve the long-term prognosis and quality of life in patients with heart failure, which is in accordance with the results of several clinical trials.\(^1\,^2\) In addition, the severe heart dysfunction in our patient recovered to absolute normality after the correction of LBBB by HBP, which implies the presence of LBBB-induced cardiomyopathy instead of primary cardiomyopathy in this patient. Similarly, Vaillant et al.\(^3\) reported that six patients with isolated LBBB developed nonischemic cardiomyopathy characterized by a

![Figure 2](image_url). Lead position determined by fluoroscopy in the (a) left anterior oblique (LAO) view and (b) anteroposterior (AP) view. The red arrows show the 3830 His lead.
decreased LVEF and left ventricular enlargement after 11 years of follow-up, and all patients were hyper-responders to cardiac resynchronization therapy, indicating that LBBB can cause severe heart failure. Moreover, Auffret et al.4 reviewed the evidence of LBBB-induced cardiomyopathy and implied that LBBB might be the best explanation for the subsequent progression to cardiomyopathy in patients who exhibit an excellent response to cardiac resynchronization therapy. However, a large-cohort study with prospective follow-up is required.

In the present case, 2:1 atrioventricular conduction occurred during His bundle mapping, and the intracardiac electrogram showed HV block. After HBP, the LBBB was corrected; however, RBBB occurred with a QRS duration of 120 ms. We inferred that the RBB had been injured by the lead. The HBP procedure runs the risk of damaging the normal conduction system of the heart. Vijayaram et al.5 reported the incidence of His bundle injury during permanent HBP in patients with no evidence of His-Purkinje disease. In total, 28 of 358 patients had His bundle injury in the form of third-degree atrioventricular block (n = 4), RBBB (n = 21), and LBBB (n = 3). All patients with third-degree atrioventricular block, all patients with LBBB, and 12 of 21 patients with RBBB completely recovered from their injury within 1 minute to 1 hour. However, the remaining 9 of the 21 patients with RBBB did not recover, even during the follow-up period. Most cases of RBBB occurred during fixation of the His bundle electrode, and a few occurred during the process of electrical mapping. In the current case, RBBB occurred during the process of electrical mapping to identify the appropriate site of the His bundle. The RBB had not recovered from this injury within 72 hours, whereas it had finally recovered by the 1-month follow-up. Because the ventricular escape beats occurred before the RBBB occurred, we inferred that the cause of the RBBB may be related to mechanical injury of the distal RBB during mapping, suggesting that a temporary pacemaker is necessary during the HBP procedure for patients with LBBB.

In conclusion, we have herein reported a case in which a patient, initially diagnosed with dilated cardiomyopathy, presented with heart failure and LBBB that were eventually reversed by HBP. This case
provides evidence for the clinical presence of LBBB-induced cardiomyopathy and shows that the use of temporary pacemakers is essential during HBP procedures in patients with LBBB. Moreover, the recovery period for the intraoperative mechanical injury to RBB conduction may be delayed.

Acknowledgment
The authors thank all participants of the study. The authors also thank all colleagues for providing guidance and help.

Ethics statement
The study was approved by the institutional review board of the First Affiliated Hospital of Dalian Medical University, Dalian, Liaoning, China. The patient provided written informed consent. The study was conducted according to the Guidelines of the Declaration of Helsinki. All protocols described here were performed in accordance with the approved guidelines.

Declaration of conflicting interest
The authors declare that there is no conflict of interest.

Funding
This study was supported by a grant from the Natural Science Foundation of Liaoning Province of China (20170540258).

ORCID iDs
Fei Liu https://orcid.org/0000-0002-1753-5445
Yunlong Xia https://orcid.org/0000-0003-4791-9666
Yingxue Dong https://orcid.org/0000-0002-1342-9012

References
1. Vijayaraman P, Dandamudi G, Zanon F, et al. Permanent His bundle pacing: recommendations from a Multicenter His Bundle Pacing Collaborative Working Group for standardization of definitions, implant measurements, and follow-up. Heart Rhythm 2018; 15: 460–468. doi: 10.1016/j.hrthm.2017.10.039
2. Zanon F, Ellenbogen KA, Dandamudi G, et al. Permanent His-bundle pacing: a systematic literature review and meta-analysis. Europace 2018; 20: 1819–1826. doi: 10.1093/europace/euy058
3. Vaillant C, Martins RP, Donal E, et al. Resolution of left bundle branch block-induced cardiomyopathy by cardiac resynchronization therapy. J Am Coll Cardiol 2013; 61: 1089–1095. doi: 10.1016/j.jacc.2012.10.053
4. Auffret V, Martins RP, Daubert C, et al. Idiopathic/Iatrogenic Left Bundle Branch Block-induced reversible left ventricle dysfunction: JACC state-of-the-art review. J Am Coll Cardiol 2018; 72: 3177–3188. doi: 10.1016/j.jacc.2018.09.069
5. Vijayaraman P, Dandamudi G and Ellenbogen KA. Electrophysiological observations of acute His bundle injury during permanent His bundle pacing. J Electrocadiol 2016; 49: 664–669. doi: 10.1016/j.jelectrocard.2016.07.006