Case Report

Improvement of Left Ventricular Function by Permanent Direct His-Bundle Pacing in a Case with Dilated Cardiomyopathy

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The patient was a 67-year-old female diagnosed with dilated cardiomyopathy. She had chronic atrial fibrillation (AF) with bradycardia and low left ventricular function (left ventricular ejection fraction (LVEF) 40%). She was admitted for congestive heart failure. She remained New York Heart Association (NYHA) functional class III due to AF bradycardia. Pacemaker implantation was necessary for treatment of heart failure and administration of dose intensive β-blockers. As she had normal His-Purkinje activation, we examined the optimal pacing sites. Hemodynamics of His-bundle pacing and biventricular pacing were compared. Pulmonary capillary wedge pressure (PCWP) was significantly lower on His-bundle pacing than right ventricular (RV) apical pacing and biventricular pacing (13 mmHg, 19 mmHg, and 19 mmHg, respectively) with an almost equal cardiac index. Based on the examination we implanted a permanent pacemaker for Direct His-bundle pacing (DHBP). After the DHBP implantation, the LVEF immediately improved from 40% to 55%, and BNP level decreased from 422 pg/ml to 42 pg/ml. The number of premature ventricular complex (PVC) was decreased, and non sustained ventricular tachycardia (NSVT) disappeared. Pacing threshold for His-bundle pacing has remained at the same level. His-bundle pacing has been maintained during 27 months and her long-term DHBP can improve cardiac function and the NYHA functional class.
(J Arrhythmia 2006; 22: 245–250)

Key words: Bundle of His, Pacing, Cardiomyopathy

Introduction

Several clinical trials have evaluated that biventricular pacing can improve intraventricular conduction delays (IVCD) in heart failure patients. Biventricular pacing has been adapted for therapy-resistant heart failure patients because intraventricular conduction delay would deteriorate not only dyssynchronous left ventricular contraction but also ventricular performance. Recently Direct His-bundle pacing (DHBP) has been applied for heart failure patients with normal His-Purkinje activation instead of conventional apical pacing. Even though DHBP has still some technical problems of implantation,

Received 12, September, 2006: accepted in final form 1, December, 2006.
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DHBP is able to provide improved non-artificial synchronous ventricular depolarization and prevent reduction of cardiac function compared to apical pacing. We report a case of dilated cardiomyopathy with bradycardia, which had been clinically improved by permanent DHBP implantation for more than 2 years.

Case Report

In March 2004, a 67-year-old female with dilated cardiomyopathy was admitted to our hospital from another hospital for treatment of congestive heart failure (New York Heart Association (NYHA) functional class III) and non-sustained ventricular tachycardia (NSVT). Her cardiac catheter examination in her previous hospital showed a left ventricular ejection fraction (LVEF) of 21% and normal coronary arteries. In physical examinations, her blood pressure was 90/40 mmHg, and pulse rate was 56 beats/min with an irregular rhythm. She showed neither murmur nor rales, and she did not have leg edema. Cardiothoracic ratio (CTR) was 62% on chest radiography, and the ECG showed atrial fibrillation without IVCD (QRS = 89 msec, **Figure 1A**). Left ventricular end-diastolic dimension (LVDd) was 52 mm and left ventricular wall motion revealed diffuse severe hypokinesis (LVEF, 40%) with mild mitral regurgitation on echocardiography. Biochemistry examinations were not remarkable other than her serum B-type natriuretic peptide (BNP) level of 423 pg/ml. Holter electrocardiography showed atrial fibrillation with beadycardia (mean heart rate, 51 beats/min), and frequent premature ventricular complexes (PVCs) and NSVT (PVCs, 2116 beats/day; NSVTs, 5 times/day and maximum length 5 beats).

After starting amiodarone administration for NSVT, bradycardia deteriorated (mean heart rate, 41 beats/min). Pacemaker implantation and administration of dose intensive β-blockers (carvedilol 10 mg per day) was necessary for treatment of heart failure. As she had normal His-Purkinje conduction, we examined the comparative hemodynamics for right ventricular (RV) apical pacing, His-bundle pacing, and biventricular pacing (**Table 1**). For biventricular pacing, a deflectable catheter was positioned in the left ventricular lateral wall and stimulated with a RV apical temporary pacing catheter. The pulmonary capillary wedge pressure

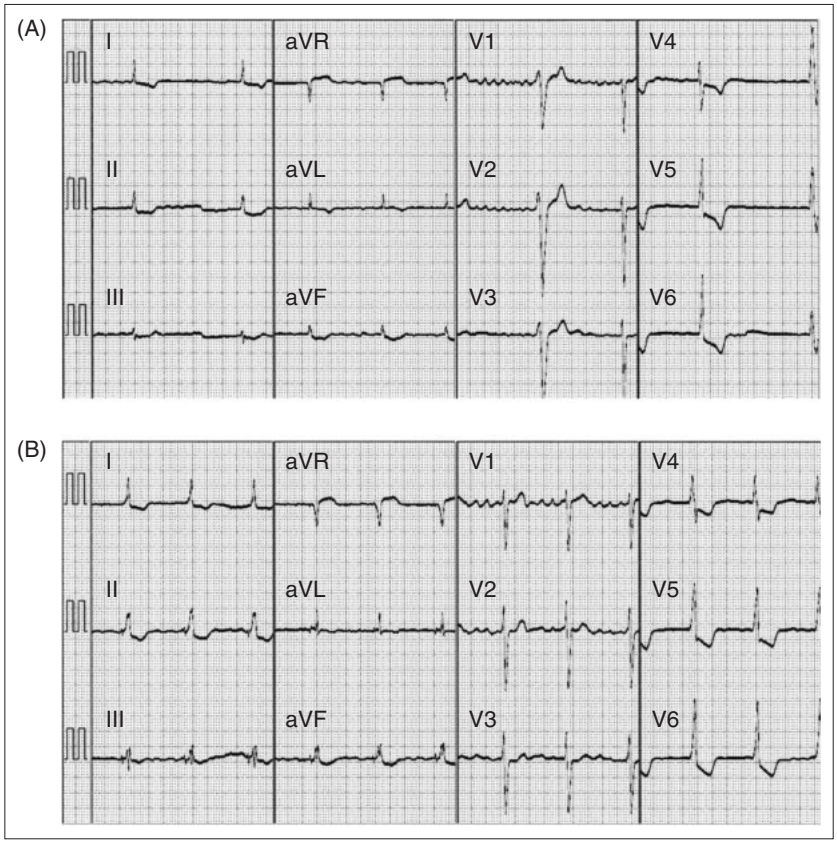

Figure 1 Surface 12-lead electrocardiogram comparing native conduction and DHBP.
QRS and T-wave of DHBP (B) are in concordance with that of native conduction (A).

was significantly lower on His-bundle pacing than RV and biventricular pacing (13 mmHg, 19 mmHg, and 19 mmHg, respectively) with a nearly equal cardiac index.

Based on the examination, we implanted a permanent DHBP-type pacemaker. The surgery took 3 hours and 10 minutes. First, a His-bundle mapping catheter was introduced via the right femoral vein. A bipolar screw-in lead (Thin Line II Sterox, model No. 430-35S, Intermedics) was introduced via the right subclavian vein and advanced to the atrioventricular septum. Iterative adjustments to the "J"-shaped stylet were required because of patient's anatomy. A lead was required to achieve optimal positioning near the mapping catheter and adjusted to obtain the largest His potential using a modified "J"-shaped stylet. The lead could capture the His bundle directly at reasonable low pacing out put (**Figure 2**). Another lead (CapSureFix NOVUS, model No. 5068, Medtronic) was positioned in the RV apical septum for back-up pacing (acute pacing threshold was 0.9 V at 0.5 msec pulse width, R-wave sensing was 10.2 mV). The lead for His-bundle pacing was connected to the atrial port, and another lead was connected to the ventricular port. The pacemaker was set at AAI mode and a fixed rate of 70 beats/min. Acute His-bundle pacing threshold was 3.2 V at 0.5 msec pulse width, and sensed potential (R-wave sensing) was 4.2 mV. Atrial far-field potentials were never observed. QRS complex of DHBP was in concordance with that of native conduction (**Figure 1**).

Table 1 Hemodynamic data at different pacing sites.

Pacing site	control	RV	Bi-Vent	His-bundle
HR	54	70	70	70
PA (sys/dia)	39/11	39/17	41/17	42/16
PCWP (m)	10	19	19	13
CI	1.83	2.04	1.85	2.09
BP (sys/dia)	135/65	139/73	142/64	146/77

HR = heart rate (/min)
PA = pulmonary artery pressure (systolic/diastolic, mmHg)
PCWP = pulmonary capillary wedge pressure (mean, mmHg)
CI = cardiac index (L/min/m^2)
BP = blood pressure (systolic/diastolic, mmHg)

100% His-bundle pacing has been maintained during 27 months after the pacemaker implantation. Drug therapy including intensive β-blockers has been continued at the same dose. In April 2004, her echocardiograms showed a remarkable improvement of LVEF from 40% to 55%, and her mild mitral regurgitation disappeared, which has continued to the present. The CTR decreased from 62% to 56% in about one month (**Figure 3**). BNP immediately decreased from 423 pg/ml to 237 pg/ml in a week after DHBP implantation, and afterward gradually decreased to 42 pg/ml (**Figure 4**). The number of PVCs decreased from 2116 beats/day to 66 beats/day and the NSVT has disappeared. NYHA functional class is improving from class III to class I at present. Chronic His-bundle pacing threshold after 2 years was 3.2 V at a 0.5 msec pulse width, and it remains unchanged.

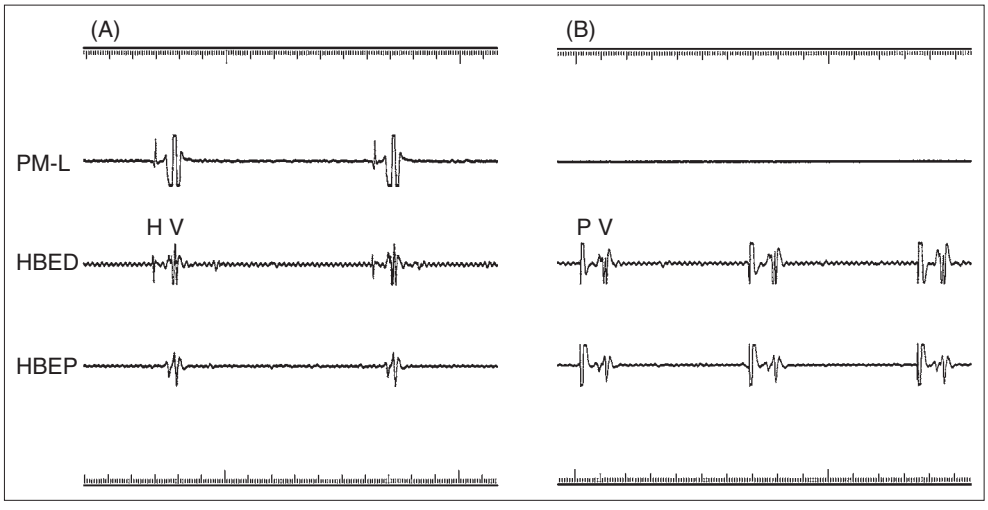

Figure 2 Intracardiac electrograms from His-bandle mapping catheter (HBED, distal and HBEP, proximal) and pacemaker lead for DHBP (PM-L).
(A) is intrinsically conducted, and (B) is His-bundle paced. HV interval (H-V) in native conduction is 70 msec. Equivalence in His-ventricular (H-V) and pace-ventricular (P-H) activation intervals provides evidence of successful DHBP.

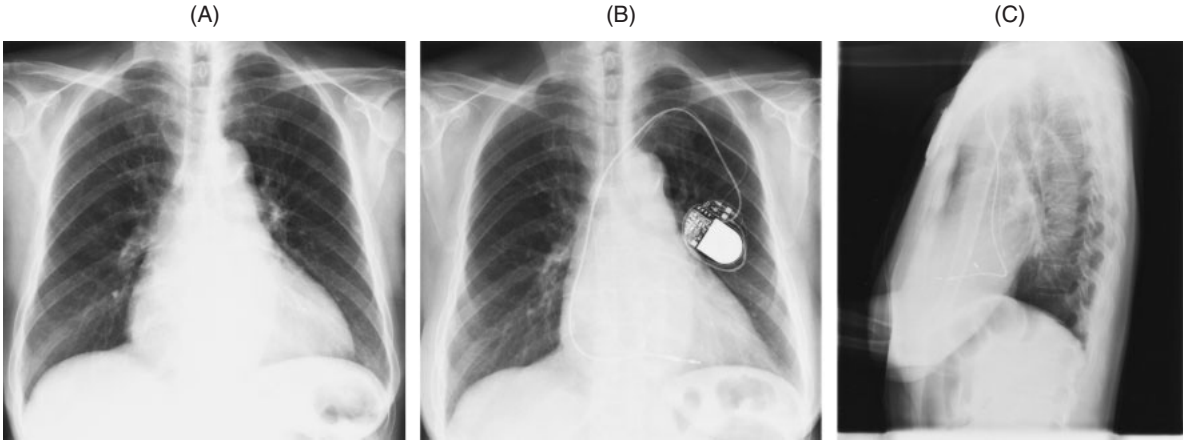

Figure 3 Radiographic images showing reduction in heart size, before (A) and after sustained DHBP in about one month (B), (C).
The CTR decreased from 62% to 56% in about one month.

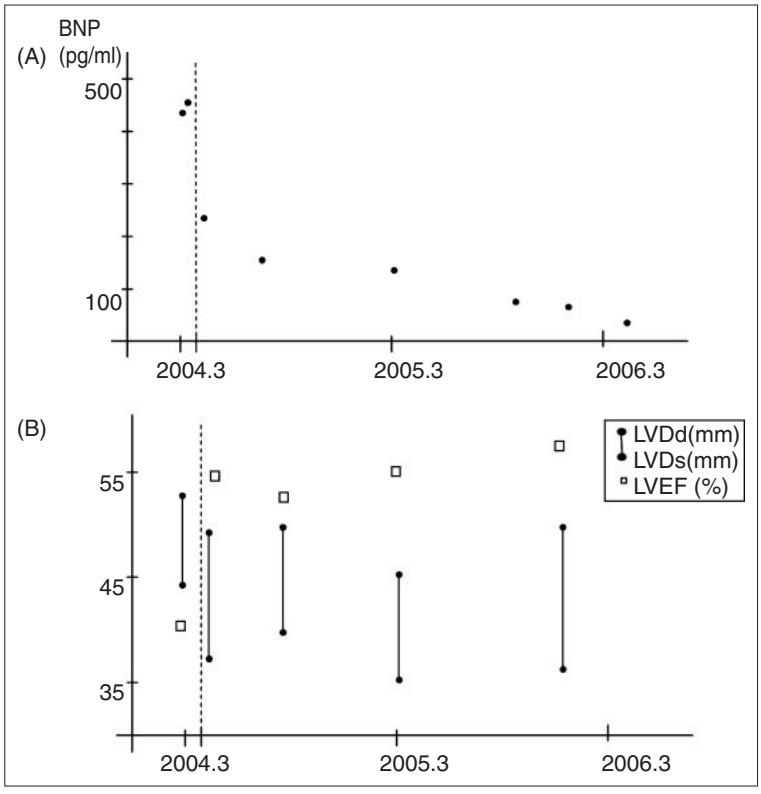

Figure 4 Changes of serum BNP level (A) and echocardiographic data (B).
BNP immediately decreased from 423 pg/ml to 237 pg/ml in a week after DHBP implantation, and afterward gradually decreased to 42 pg/ml. Echocardiographic data showed remarkable improvement of LVEF from 40% to 55%. Dotted line is the day of DHBP implantation.
LVDd = Left ventricular diastolic dimension
LVDs = Left ventricular systolic dimension

Discussion

Antibradicardia pacemaker therapy is considered to be a somewhat well-established method. Many studies have actually shown the effectiveness of physiologic pacing therapy in the past. The DANISH trial[1] proved the beneficial effect of physiologic pacing. The trial showed that atrial pacing was associated with a significantly higher survival, less atrial fibrillation and heart failure than ventricular pacing. However, CTOPP,[2] MOST,[3] PACE,[4] and UKPACE[5] reported that differences between physiologic pacing and ventricular pacing were not significant in the rates of heart failure and survival except for atrial fibrillation. The DAVID trial[6] reported that dual-chamber pacing compared with

ventricular backup pacing was associated with an increase of the combined end point of death or hospitalization for heart failure for patients with both standard indications for ICD therapy and a LVEF of less than 40%. Sweeny et al.[7] reported that a 10% increase of cumulative ventricular pacing was correlated with a 54% increase in risk of heart failure hospitalization even though AV synchrony was preserved. These studies suggest that right ventricular apical pacing produces a left ventricular electrical activation sequence resembling left bundle-branch block, and consequently can induce heart failure.

On the other hand, recent studies showed that biventricular pacing therapy is efficient for severe heart failure patients with IVCD.[8–10] Approximately 15% to 30% of advanced heart failure patients have been associated with IVCD. The biventricular pacing strategy is based on the fact that most patients with IVCD have a dyssynchronous left ventricular contraction, which results in a reduction in ventricular performance. It remains unclear whether biventricular pacing therapy provides the most benefit for non-IVCD heart failure patients.

The patient described in this study had a severe ventricular dysfunction (LVEF, 40%) with dilated cardiomyopathy and chronic atrial fibrillation, and demonstrated NYHA functional class III. Though she was treated with maximal tolerated medical therapy for heart failure, including agiotensin II type 1 receptor antagonists and β-blockers, she remained NYHA functional class III and her bradicardia worsened. Because she had a narrow QRS complex, we examined her hemodynamic function during His-bundle pacing as well as biventricular pacing. His-bundle pacing increased her cardiac index and decreased her pulmonary capillary wedge pressure more effectively than biventricular pacing. We thought that His-bundle pacing was favorable for its acute hemodynamic effects, and she underwent a permanent DHBP implantation. Her permanent DHBP resulted in dramatic improvements in LVEF, BNP and NYHA functional class, and improved levels have been maintained during 27 months after pacemaker implantation.

Few studies have shown that DHBP improved cardiac function for dilated cardiomyopathy with atrial fibrillation.[11] This case suggests that a permanent DHBP can achieve sustainable improvements of cardiac function and NYHA class. These effects could be derived from the regularity of the pacemaker rate and maintaining physiologic contraction without ventricular dyssynchrony. This agrees with a recent study.[12] On the one hand, pacing therapy enabled us to continue administration β-blockers. It is not clear if the documented benefit can be related, at lease in part, to continued administration of β-blockers rather than to pacing modality. In any case, DHBP can provide the maximum therapeutic benefit in bradycardia patients with chronic atrial fibrillation, dilated cardiomyopathy, and LV dysfunction with preserved ventricular activation. DHBP would be one of the most effective methods, especially for chronic heart failure patients without IVCD who have difficulty taking β-blockers because of bradycardia.

His-bundle pacing may be an appropriate pacing site for antibradicardia pacemaker therapy. There is a problem with regard to the difficulty of placing a pacing lead at the His-bundle. It is useful to place a standard multipolar mapping catheter as a guide at the His bundle, and this enables the placement of a screw-in pacing lead to be embedded in the membranous septum or on near the His bundle. Although acute and chronic pacing thresholds for His-bundle pacing were relatively higher than for RV pacing in the present case, the pacing thresholds remained clinically acceptable. However, it is capable of appearing with IVCD, with deterioration of the threshold according to disease progression, and sometimes happens the His-bundle pacing lead is dislocated. We positioned another lead to the RV septum for back-up pacing, as we considered options.

Permanent DHBP can provide more benefit for both heart failure patients without IVCD and patients with indications for antibradicardia pacing therapy.

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