A technique for cardiac resynchronization therapy using left bundle branch area and left ventricular pacing

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Cardiac resynchronization therapy (CRT) via biventricular pacing (BVP) is known to improve clinical outcomes and decreases all-cause mortality, particularly in patients with left bundle branch block (LBBB) and reduced left ventricular (LV) function.[1] Recently, several groups have shown the feasibility of left bundle branch area pacing (LBBAP) as an alternative choice to His bundle pacing in patients with LBBB by pacing the left bundle branch (LBB) region beyond the block site with a stable threshold and a short QRS duration (QRSd).[2,3] However, it is unknown whether the clinical efficacy of LBBAP with an appropriate atrioventricular (AV) delay would be the same as or better than that of LV epicardial pacing or CRT.

We developed a technique that can be accomplished more effectively using LBBAP followed by sequential LV pacing (LBB-optimized CRT [LOT–CRT]) than using existing techniques.

In this technique, the right ventricle (RV) defibrillator electrode was first implanted in the RV to provide backup ventricular pacing in case the patient develop transient complete AV block during LBBAP lead placement. Subsequently, the LV coronary sinus (CS) lead was implanted using routine implantation techniques, targeting sites with maximal LV delay.[4] Then, LBBAP was performed using the SelectSecure pacing lead (model 3830, Medtronic Inc). All defibrillator electrodes were implanted in the RV apical position. The fluoroscopy durations for the entire procedure, LBBAP lead implantation, and LV lead implantation were separately recorded.

As previously described,[5,6] a Select Site C315 His sheath and a Select Secure 3830 pacing lead (Medtronic Inc., Minneapolis, MN, USA) were advanced to the implantation site. The RV septal location for LBBAP was identified using the anatomical location and pacing localization of the nine-grid system.[7] Once this site was identified, the pacing lead is advanced deep into the septum while the unipolar pacing impedance, electrogram characteristics, and paced QRS morphology were monitored.

Additionally, the lead orientation can be displayed in various projections. During the initial LBBAP lead fixation, if the lead twists back, this indicates that the lead and sheath are not oriented orthogonal to the RV septum. Generally, the sheath and the lead are oriented such that the lead is pointing in the 12- to 1-o'clock direction from a right anterior oblique viewing angle of 30° and the 2- to 3-o'clock direction from a left anterior oblique viewing angle of 30°.[1]

In patients undergoing CRT-defibrillator (CRTD) treatment, the LBBAP lead was connected to the pace-sensing portion of the RV port, and a bipolar LV CS lead was connected to the LV port. The pace-sensing portion of the spliced implantable cardioverter-defibrillator lead was capped. In patients undergoing CRT-pacemaker (CRTP) treatment, the LV CS lead was placed first and connected to the LV port. Then, the LBBAP lead was placed and connected to the RV port.

Patients were seen for routine clinical follow-up at standard time intervals (every 3 months). Functional status was assessed by the New York Heart Association (NYHA) classification system. Device thresholds were checked and adjusted as needed to maximize battery longevity. The pacing threshold, impedance, and R wave amplitude were measured.

Consequently, five patients were registered in the LOT–CRT cohort. Among the five included patients, three (60%) were male. All patients had cardiomyopathy (two non-ischemic and three ischemic), and two patients had

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paroxysmal atrial fibrillation. Hypertension was present in all the patients. The mean age was 71.8 ± 5.1 years, and the baseline characteristics of the patients are provided in Table 1. The baseline left ventricular ejection fraction (LVEF) and the baseline QRSd with LBBB were 32.0% ± 4.2% and 158.0 ± 13.0 ms, respectively.

All patients had at least one heart failure (HF) hospitalization 3 months before LBBAP implantation. Entresto (sacubitril/valsartan), β-blockers, and loop diuretics were prescribed to all patients.

LOT–CRT was successfully achieved in all five patients. The operation duration was 152.0 ± 31.1 min. The duration of X-ray fluoroscopy was 26.2 ± 5.9 min. CRTDs were implanted in four patients, and CRTP was implanted in the remaining one patient [Supplementary Figure 1A and B, http://links.lww.com/CM9/A849].

Both the LBBAP and LV capture thresholds remained stable during follow-up (1.3 ± 0.6 V at 0.4 ms *vs.* 1.6 ± 0.7 V at 0.4 ms). Bipolar LBBAP resulted in partial but significant narrowing of the QRSd (bundle branch block correction) in five patients.

After unipolar LBBAP, five patients demonstrated a right bundle branch block pattern with a paced QRSd of 123.0 ± 5.7 ms ($P = 0.001$ *vs.* baseline). LBB potential could be recorded in three patients from the LBB lead (60%). The LV activation time for all LBBAP patients was 72.5 ± 9.4 ms, and the R wave amplitude, pacing impedance, and unipolar pacing capture threshold were 9.9 ± 7.2 V, 678 ± 102 Ω, and 0.84 ± 0.17 V/0.4 ms, respectively, after implantation.

BVP resulted in a significant reduction of the QRSd from 158.0 ± 13.0 ms at baseline to 132.0 ± 4.5 ms ($P = 0.019$). Compared with BVP, unipolar LBBAP resulted in further reduction of the QRSd to 123.0 ± 5.7 ms ($P = 0.006$ *vs.* baseline and $P = 0.021$ *vs.* BVP). However, LOT–CRT resulted in a significantly greater reduction of the QRSd to 119.0 ± 7.6 ms ($P < 0.010$ *vs.* baseline, BVP, or bipolar LBBAP).

The mean follow-up time was 296 ± 201 days. Overall, the LBBAP capture threshold, R-wave amplitude, and lead impedance were 0.74 ± 0.25 V, 13.36 ± 5.23 mV, and 533.73 ± 32.31 Ω during the 1-month follow-up ($P > 0.050$, respectively, between the time of device implantation and the follow-up visit). During LOT–CRT, the QRSd was stable, and no significant difference was observed between the time of device implantation and the 3-month follow-up visit ($P > 0.050$). The ventricular pacing rate was 99%. The latest success rate was 100%. No patients showed signs of dislodgement, loss of capture, infections, embolism, or stroke associated with the implantation.

Transthoracic echocardiogram evaluation data at baseline and the 1- and 3-month follow-ups were available in all five patients [Supplementary Figure 1C, http://links.lww.com/CM9/A849]. As shown in Table 1, the left ventricular end-diastolic dimension (68.2 ± 12.3 mm *vs.* 62.2 ± 11.3 mm, $P = 0.017$) and LVEF (32.8% ± 5.2% *vs.* 45.0% ± 5.1%, $P = 0.008$) were improved at the 3-month follow-up visit. The symptoms and the median NYHA classification score improved significantly, with the latter decreasing from 3.2 ± 0.5 to 2.4 ± 0.6 ($P = 0.016$).

The reasons for BVP–CRT non-response are many but include LV scar burden and distribution, a sub-optimal LV stimulation site, sex, and limited electrical or mechanical dyssynchrony.[8] Patients with ischemic cardiomyopathy experience a similar BVP–CRT response rate to their non-ischemic counterparts. However, a higher overall scar burden, a larger number of severely scarred segments, and a greater scar density near the LV lead tip portend an unfavorable response to BVP–CRT in ICM patients. There is evidence that CRT is not salutary in patients with posterolateral scarring.

Permanent LBBAP is an effective form of physiologic pacing with high success rates in patients with intact His-Purkinje conduction.[3] LBBAP can serve as a new CRT technique to correct LBBB, provide ventricular synchrony, and improve clinical symptoms with the reverse remodeling of the LV.[9]

However, in patients with intraventricular block or higher overall scar burden, success rates are somewhat limited depending on the site of block and the scar burden and distribution of the interventricular septum.[2] Intra or interventricular dyssynchrony cannot be reduced through LBBAP. LBBAP achieved only partial reduction of the QRSd in those patients with a baseline surface ECG of atypical LBBB morphology.[2] LOT–CRT offers the advantage of using the LV lead in addition to LBBAP in patients with intraventricular block and higher overall LV scar burden.

The limitations of this technique include the following: First, LOT–CRT was time-consuming. The duration of the operation was 152 ± 31 min, and the duration of X-ray

Table 1: Comparison of the pre- and post-operative TTE parameters.

Parameters	Before procedure	1 month after the procedure	3 months after the procedure	P^*
NYHA score	3.2 ± 0.5	2.6 ± 0.6	2.4 ± 0.6	0.016
LVEDD (mm)	68.2 ± 12.3	64.4 ± 12.6	62.2 ± 11.3	0.017
LVEF (%)	32.0 ± 4.2	41.6 ± 7.5	45.0 ± 5.1	0.008
QRSd (ms)	158.0 ± 13.0	119.0 ± 7.6	120.0 ± 7.7	0.006

Data were presented as mean ± SD. *Before procedure *vs.* 3 months after the procedure. LVEDD: Left ventricular end-diastolic dimension; LVEF: Left ventricular ejection fraction; NYHA: New York Heart Association; QRSd: QRS duration; TTE: Transthoracic echocardiogram.

fluoroscopy was 26.2 ± 5.9 min; both were longer than stated in a previous report (117 ± 48 min and 16.4 ± 12.3 min).[3] Second, this study included only a small sample at a single center. Third, this study had a short follow-up interval, although we expect favorable long-term clinical benefits. Furthermore, this study enrolled only three ischemic patients. As a newly developed technique, clinical or simulative trials are needed to validate the practical relevance of the LOT–CRT. Although with limited data, we observed significant echocardiographic and clinical improvement in these HF patients treated with LOT–CRT. So, as a conclusion, LOT–CRT was clinically feasible in patients with systolic HF and LBBB, and associated with significant reduction of QRSd and improvement in LV function, especially in patients with ICM.

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Conflicts of interest

None.

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