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ORIGINAL ARTICLE

Feasibility of RA–LV pacing in patients with symptomatic left bundle branch block: a pilot study

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Abstract

Several studies have reported the adverse effects of right ventricular apical pacing. Permanent His bundle pacing is proved to be the most physiological. But it can be technically difficult sometimes. One recent large multicenter randomized trial showed that pacing from left ventricular apex or mid-lateral wall has the greatest potential to prevent pacing-induced reduction of cardiac pump function (by maintaining left ventricular mechanical synchrony) and, therefore, can be considered as physiological site. In our study, we have wanted to see the outcome of left ventricular pacing through coronary sinus branch with active fixation bipolar lead as a routine pacing technique in patients with symptomatic left bundle branch block. In our study we have recruited 27 patients for left ventricular pacing through coronary sinus branch (as done in cardiac resynchronization therapy) with active fixation bipolar lead and 33 patients for right ventricular apical pacing (control) and compared left ventricular pacing with right ventricular apical pacing in patients with history of syncope with left bundle branch block in baseline electrocardiography who presented with atrio-ventricular block or prolonged HV interval ($\geq$70 ms) on electrophysiology study in term of procedure and fluoroscopy time and short-term lead performance and left ventricular function. The results of our study showed that left ventricular pacing through a tributary of coronary sinus is associated with shortened QRS duration (21.10 $\pm$ 3.92 ms) and better LV function (higher left ventricular ejection fraction 64.00 $\pm$ 3.03 vs. 59.73 $\pm$ 6.73 and lower left ventricular diastolic internal diameter 4.58 $\pm$ 0.32 vs. 5.23 $\pm$ 0.40 cm) in comparison to right ventricular apical pacing. However, the total procedure time and fluoroscopy time was significantly higher (73.75 $\pm$ 11.02 vs. 63.32 $\pm$ 6.06 min and 7.08 $\pm$ 1.48 vs. 5.02 $\pm$ 1.39 min, respectively) in left ventricular pacing group. The results of this study indicate that transvenous left ventricular epicardial pacing may be an option for physiological pacing in patients with symptomatic left bundle branch block.

Keywords Left bundle branch block · Coronary sinus · Physiological pacing · Epicardial pacing

Introduction

Right ventricular (RV) apical pacing can be detrimental in some patients [1–3]. Studies have shown that baseline-wide QRS complex and left bundle branch block (LBBB) are associated with higher incidence of heart failure hospitalization and increased cardiac mortality [4]. Permanent His bundle (HB) pacing is proved to be the most physiological as it does not induce ventricular dyssynchrony (electrical as well as mechanical) (inter and intra) [5]. It corrects wide range of atrio-ventricular (AV) block including nodal and infra-nodal [6]. In patients with standard indications for cardiac resynchronization therapy, it has been seen that in about 70% cases it is possible to narrow the QRS by capturing distal to the diseased His tissue and can be screened by a HB mapping catheter [7]. HB in about one third of the patients remains deep seated in the muscular ventricular septum and reaching the HB area with conventional pacing lead in these patients may be difficult sometimes [8]. Moreover, HB pacing in some patients may fail to narrow the paced complex because of the infra-Hisian location of the conduction block [6]. Investigators have proposed RV septal pacing as a method of physiological pacing [9]. But, the results of RV septal pacing are heterogeneous [10, 11]. One recent large multicenter randomized trial showed that pacing from left ventricular (LV) apex or LV mid-lateral wall has

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the greatest potential to prevent pacing-induced reduction of cardiac pump function (by maintaining LV mechanical synchrony) and, therefore, can be considered most physiological site [12]. Studies have also shown that LV pacing as a part of cardiac resynchronization therapy (CRT) is most effective for patients with LBBB and less effective for non-LBBB group [13]. In our study, we compared LV pacing (as done in CRT) with RV apical pacing in term of procedure time and fluoroscopy time, short-term lead performance and short-term LV function in patients with symptomatic LBBB.

Method

In our hospitals, from 1st July 2016 till 31st December 2017 we had recruited 60 patients (30 in RV apical and 30 in LV epicardial group) who presented with history of syncope with baseline LBBB (as defined by American Heart Association/American College of Cardiology Foundation/Heart Rhythm Society recommendations [14]) preserved LV systolic function (LV ejection fraction > 50%) with one of the followings: (1) complete or advanced AV block, (2) QRS duration of ≥ 140 ms with HV interval ≥ 70 ms on electrophysiology (EP) study. All the patients underwent dual chamber pacing. In a prior date to pacemaker implantation we took all the patients (who are not in AV block) to our EP laboratory and put a quadripolar catheter into the His bundle area by trans-femoral venous route and measured the HV interval (distance between His deflection and the beginning of earliest surface QRS).

We assigned the patients to either of the two groups (RV apical or LV epicardial pacing through the branch of CS for ventricular pacing) (simple randomization). We took clearance from our institutional ethical committee for this study. After taking proper informed consent, under local anesthesia with the coverage of intravenous antibiotics access to the left subclavian vein was performed in all cases. Then the pacemaker pocket was prepared with blunt dissection. In control group (RV apical) for ventricular pacing we used conventional bipolar pacing lead (either active or passive fixation lead available at the time of implantation) and positioned into the RV apex with the help of a simple curved stylet. For LV pacing group, we used a deca-polar EP catheter (or AL 2 angiographic catheter) to cannulate the CS ostium through a guiding sheath in fluoroscopic left anterior oblique (LAO) view (Fig. 1a). Occlusive CS venogram was

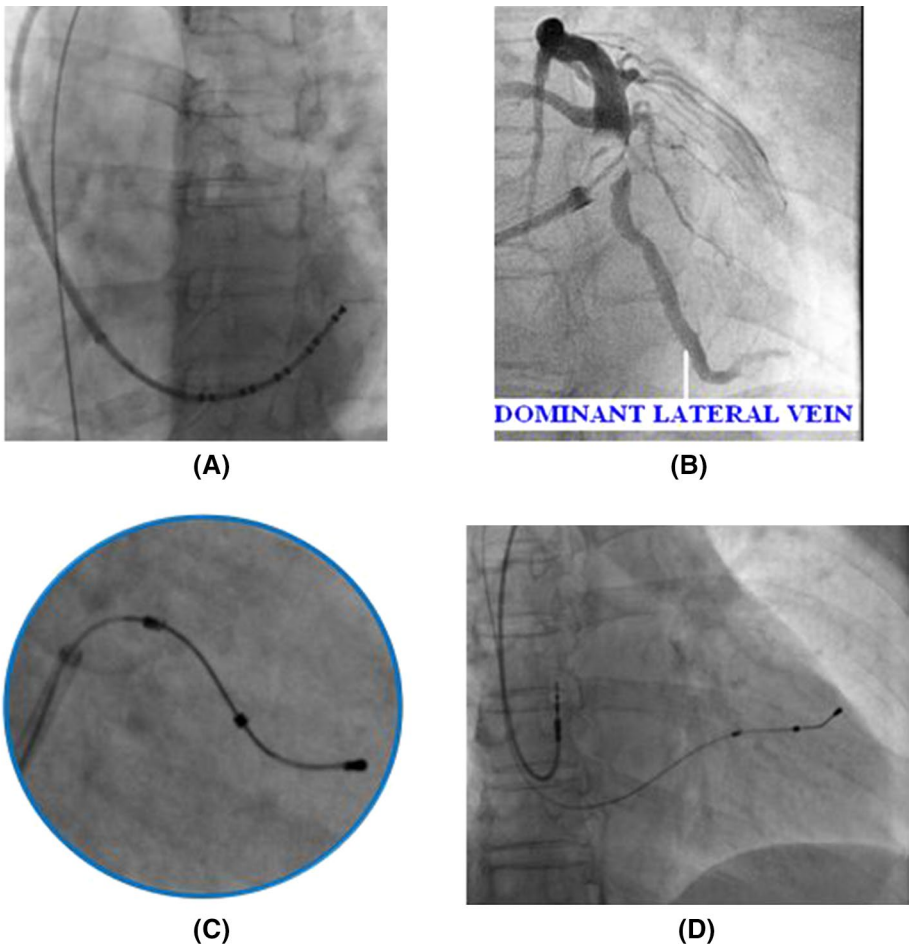
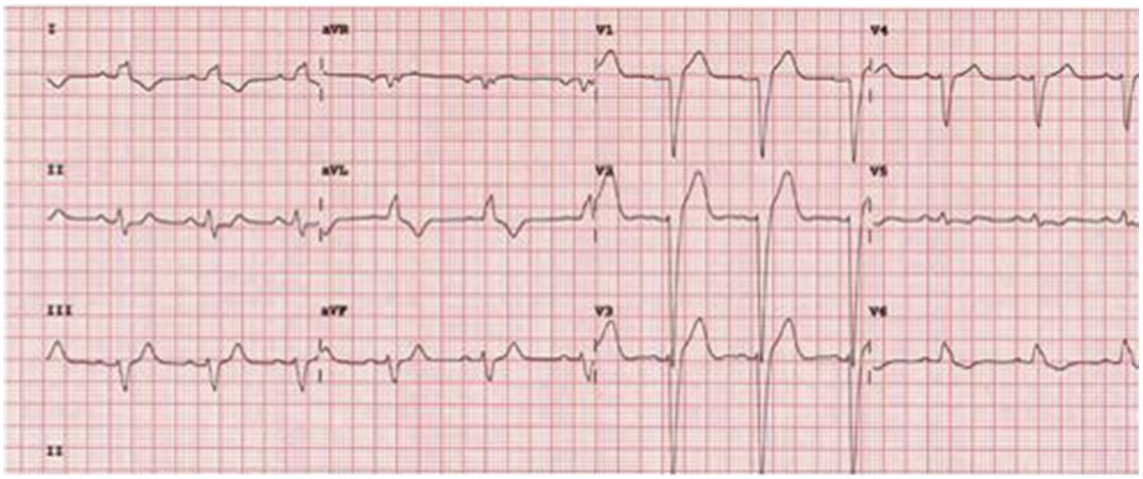


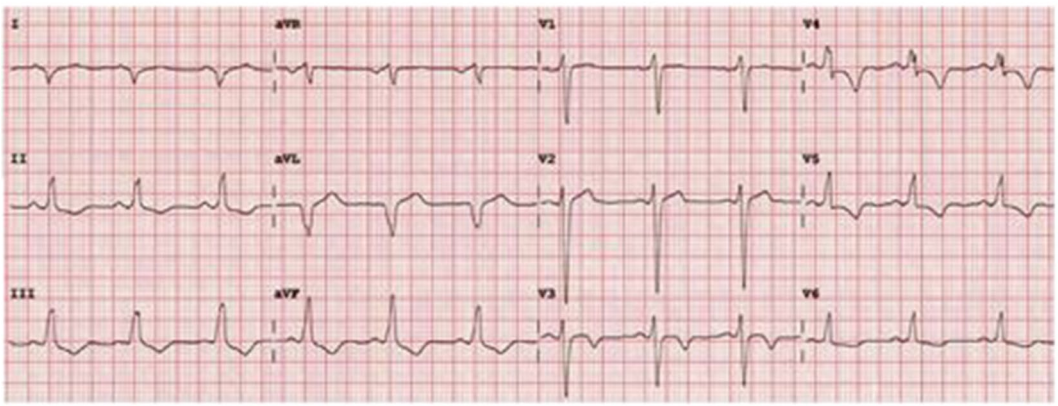
Fig. 1 **a** A deca-polar EP catheter positioned into the CS through the guiding sheath in fluoroscopic LAO view. **b** Occlusive CS venogram in fluoroscopic LAO view shows dominant large caliber lateral vein which was the target vein in this case. **c** Fluoroscopic appearance of the lead after deployment in LAO view, **d** fluoroscopic RAO appearance of the atrial and ventricular leads

performed in both LAO (Fig. 1b) and right anterior oblique (RAO) view using a Swan-Ganz catheter. The target vein (usually lateral or postero-lateral vein, antero-lateral vein was the last choice) was identified and the lead (Attain stability, Medtronic Ltd) was positioned over a 0.014″ coronary wire and active fixation screw was deployed by 6–8 clockwise rotation of the lead keeping the wire fixed (Fig. 1c). Atrial lead was positioned in right atrial appendage in all the patients using conventional J-stylet (Fig. 1d). After positioning, the leads were checked for parameters (R wave sensitivity, pacing threshold and lead impedance). Stability of the leads was checked with deep breath and mild cough maneuver. Once satisfied with the lead parameters, the guiding sheath was removed. Then the leads were connected to the pulse generator and positioned in the preformed pocket. Wound was closed after achieving proper hemostasis.

We observed total procedure time (skin to skin) and fluoroscopy time in all patients. Lead parameters were noted during implantation and at 6 months post-procedure follow-up. ECG-gated AV optimization was done in patients in both the group before discharge. A series of AV delays were tested for both the group ranging from 80 to 240 ms in 20 ms increment until intrinsic conduction appears. AV delay was considered optimum where the QRS duration was minimum. Figure 2 demonstrates an example of AV optimization in a patient. Routine electrocardiography and echocardiography were performed in all patients before discharge and at 6 months during follow-up. We observed change in QRS duration and QRS shortening if any in both control and study groups. We also observed LV function in form of ejection fraction (EF) and diastolic internal diameter (LVIDd) before implantation and at follow-up after 6 months in all cases. We have also observed for evidence of AV dyssynchrony, interventricular dyssynchrony and intraventricular dyssynchrony. AV dyssynchrony was defined as the reduced total diastolic filling time (E plus A wave duration on pulsed wave Doppler at mitral valve level) (< 45% of the corresponding cardiac cycle). Interventricular dyssynchrony was said to be



(A) Base-line ECG showing QRS duration of 150 msec.



(B) ECG after AV delay optimization (AV delay 120 msec) shows QRS duration of 110 mse.

Fig. 2 AV optimization in a patient

present if the interventricular mechanical delay (measured by standard pulsed wave Doppler echocardiography as the interval between the onset of the QRS and the onset of the aortic and ejection) was more than 40 ms. intraventricular dyssynchrony was defined as the septal to posterior wall motion delay (the distance between the first maximum systolic inward motion of the septum and the maximum inward motion of the posterior wall measured in M-mode) of more than 130 ms. We have also noted for changes in the grade of mitral regurgitation. The severity of MR was assessed according to PISA method. Collected data were analyzed using SPSS 21 software. Bivariate comparisons between two groups were performed.

Results

In LV pacing group we successfully implanted the ventricular pacing lead into the target vein of the CS in 27/30 (90%) patients. In three patients we failed to position the lead in the target vein because of their small caliber and these three patients were enrolled in control group. There was no procedural complication.

We recruited 27 patients in study group (LV epicardial pacing) and 33 patients in control group (RV apical pacing) with similar demographic profile (age was 61.0 ± 8.22 vs. 59.64 ± 10.83 years and male:female was 7:2 vs. 8:3). Table 1 provides important statistical information of the two groups. Clinical presentations of the patients in both the group were similar with similar QRS duration at presentation (152.80 ± 4.37 vs. 152.64 ± 5.10 ms). There was no significant difference in measured HV interval (in study group was 77.60 ± 3.78 ms, whereas in control group was 77.11 ± 4.81 ms). Pre-implant LV function was similar in both the groups (LVEF in study group was 61.15 ± 4.04% and in control group was 62.50 ± 4.00% and LVIDd was 4.75 ± 0.33 and 4.99 ± 0.35 cm, respectively).

Total procedure time and fluoroscopy time were higher in LV epicardial pacing group (73.75 ± 11.02 vs. 63.32 ± 6.06 min and 7.08 ± 1.48 vs. 5.02 ± 1.39 min, respectively). R wave amplitude and pacing threshold in study group were similar with the control group (14.55 ± 3.78 and 0.81 ± 0.17 V vs. 12.64 ± 3.23 and 0.80 ± 0.18 V). However, lead impedance was much higher in study group during implantation (787.45 ± 140.96 vs. 672.86 ± 64.72 ohms). Post-implant QRS duration was shorter in study group in comparison to control group (124.70 ± 3.74 vs. 154.36 ± 6.13 ms with p value of < 0.001).

Pacing LV lead in study group was positioned in lateral vein in 12 patients, in postero-lateral vein in nine patients and in antero-lateral vein in six patients. Mean QRS shortening in study group was 21.10 (± 3.92) ms. The QRS shortening in lateral vein group was 28.67 ± 4.80 ms, while in postero-lateral and antero-lateral group were 28.57 ± 1.51 and 26.00 ± 4.90 ms, respectively. However, no significant correlation of QRS shortening was noted in relation to a

Table 1 Comparison of LV epicardial (study group) and RV apical (control group)

	Group				p value	Significance
	Control		Treatment			
	Mean	Std. deviation	Mean	Std. deviation		
Age	59.64	10.83	61.00	8.22	0.651	Not significant
QRS width pre op	152.64	5.10	152.80	4.37	0.912	Not significant
HV interval	77.11	4.81	77.60	3.78	0.728	Not significant
LVEF pre op	62.50	4.00	61.15	4.04	0.283	Not significant
LVIDD—pre op	4.99	0.35	4.75	0.33	0.027	Significant
Total time (min)	64.32	6.06	73.75	11.02	0.001	Significant
Fluro time (min)	5.02	1.39	7.08	1.48	< 0.001	Significant
R AMP (R wave amplitude)—pre op	12.64	3.23	14.55	3.78	0.084	Not significant
TH (threshold)—pre op	0.80	0.18	0.81	0.17	0.993	Not significant
IMP (impedence)—pre op	672.86	64.72	787.45	140.96	0.001	Significant
QRS width post op	154.36	6.13	124.70	3.74	< 0.001	Significant
Net QRS duration shortening	− 1.09	6.36	28.10	3.92	< 0.001	Significant
LVEF follow-up	59.73	6.73	64.00	3.03	0.013	Significant
LVIDD—follow-up	5.23	0.40	4.58	0.32	< 0.001	Significant
R AMP (R wave amplitude)—follow-up	12.91	2.45	14.40	3.05	0.087	Not significant
TH (threshold)—follow-up	1.00	0.16	1.36	0.29	< 0.001	Significant
IMP (impedence)—follow-up	710.41	69.40	814.55	137.09	0.003	Significant

specific territory (lateral vs. postero-lateral vs. antero-lateral) (p value 0.51). During follow-up pacing threshold and lead impedance were higher in study group (1.36 ± 0.29 vs. 1.00 ± 0.16 V and 814.55 ± 137.09 vs. 710.41 ± 69.40 ohms, respectively). However, the parameters were within acceptable limit. The optimized AV delay in LV epicardial pacing group was 112 ± 17.65 ms and in RV apical pacing group was little longer (157.27 ± 19.80 ms). In comparison to control group, the patients in study group have a significantly higher LVEF (64.00 ± 3.03 vs. 59.73 ± 6.73 v) (p value 0.013) and lower LVIDd (4.58 ± 0.32 vs. 5.23 ± 0.40 cm) (p value < 0.001). Total diastolic filling time preoperatively was similar in both case and control group (48.3 ± 7.27% vs. 46.95 ± 7.03, respectively, with p value 0.54). During follow-up AV synchrony was better in study group (total diastolic filling time of 53.30 ± 4.99% vs. 43.18 ± 6.91% p value < 0.001). Inter-ventricular delay pre-procedure was similar in both the groups (40.85 ± 16.58 ms vs. 44.91 ± 10.88 ms, p value is 0.193). Interventricular dyssynchrony was minimal in study group while it was significant in control group (29.20 ± 4.12 ms vs. 42.91 ± 12.58, p value is < 0.001). Intraventricular delay as expressed by septal to posterior wall motion delay was comparable in both the group (82.60 ± 47.54 vs. 79.73 ± 40.40, p value is 0.53). Intraventricular delay was less in control group (48.00 ± 23.49 vs. 104.45 ± 44.42 ms, p value is < 0.001). There was no significant increase in the severity of mitral regurgitation. Only one patient developed Grade 3 MR in control group.

Discussion

Retrospective analysis of the mode selection trial (MOST) suggests that in patients with permanent pacemaker, the risks of heart failure hospitalization and atrial fibrillation can be directly linked to RV pacing burden (cumulative percent ventricular pacing) regardless of pacing mode [3]. Patients with pre-implant lower LVEF, coronary artery disease (prior myocardial infarction), LBBB, and unfavorable NYHA functional class are at greater risk of these adverse events.

Classical LBBB was defined according to American Heart Association/American College of Cardiology Foundation/Heart Rhythm Society recommendations: native QRS duration ≥ 120 ms; broad (frequently notched or slurred) R waves in leads I, aVL, V5, or V6; absent q waves in lead I, aVL, V5 and V6; R peak time > 60 ms in leads V5 and V6 but normal in V1, V2 and V3, when small initial r waves can be discerned in the above leads [13]. Strict LBBB was defined according to the criteria proposed by Strauss et al.: QRS duration ≥ 140 ms for men and ≥ 130 ms for women, QS or rS in V1-V2, mid-QRS notching or slurring in at least 2 contiguous leads (V1, V2, V5, V6, I and aVL,) [15]. The study by Martí-Almor et al. reported 249 patients with bifascicular block and confirmed that a history of syncope is predictive of complete AV block. The association of structural heart disease, renal failure and a wide QRS complex (≥ 140 ms) increased the incidence of complete AV block [16]. The definition of a maximum "normal" HV interval using a ROC curve with an optimal sensitivity and specificity relationship is welcome: a maximum value of 64 ms may be more effective than 70 ms in this respect. However, we used 70 ms as the highest normal value of HV interval.

HB pacing (selective and non-selective) produces ventricular contraction through native conduction system. So, it does not produce ventricular dyssynchrony (inter and intra) or does not trigger the myocardial perfusion disorder [5]. It has been seen that in patients with dilated cardiomyopathy with LBBB, HB pacing can normalize QRS in about 72% cases [7]. However, infra-Hisian LBBB is a complex electrical disease and can result from block or conduction delays (fixed or functional) in any of several sites of the left-sided intraventricular conduction system, including the main left bundle branch or its subdivisions. The electrical impulse is conducted essentially through the working myocardium with slow conduction properties, rather than through the rapid specialized conduction system. As a result, during abnormal electrical conduction the time required for complete activation of the ventricular muscle is much longer than during physiologic conduction and furthermore a pathologic and asynchronous activation pattern occurs. Despite similar surface electrocardiographic appearances, left ventricular electrical activation patterns in LBBB are highly heterogeneous and unpredictable [17]. HB pacing may not be always possible due to anatomical reason or non-availability of EP system.

RV septal (outflow- or mid-) pacing emerged as one of the most preferred physiological site of pacing. Achieving true septal pacing site requires use of multiple fluoroscopic projections and can be a tedious job sometimes. Study results of RV septal pacing are also heterogeneous and the beneficial effects of ventricular septal pacing are yet to be proved by large multicenter studies [10, 11]. One recent large multicenter randomized trial showed that pacing from LV apex or LV mid-lateral wall has the greatest potential to prevent pacing induced reduction of cardiac pump function (by maintaining LV mechanical synchrony) in patients with high ventricular pacing burden (high cum% VP) [12]. In this study LV pacing was performed with epicardial lead. Several other studies had also supported this concept [18, 19]. So, we have selected LV epicardial pacing through the branch of CS in our current study as the mode of physiological pacing in patients with baseline LBBB. Use of pacing lead trans-venously through CS is a routine practice for cardiac resynchronization therapy. However, use of routine LV lead for ventricular pacing is limited by its little higher procedural time and higher rate of dislodgement (about 4%)

probably due to lack of strong fixation mechanism [20]. Active fixation LV lead is now available which has a lower dislodgement rate in small studies [21]. The results of our study showed that LV epicardial pacing causes significant shortening of paced QRS duration and results in better LV function in comparison to RV apical pacing at the cost of higher procedural time and higher fluoroscopy time.

LV pacing from a correctly positioned lead in the CS (lateral or posterolateral veins) invariably produces an RBBB pattern in lead V1 with a rightward axis, similar to maximal ventricular pre-excitation over a left-sided accessory pathway. With apical sites, leads V4–V6 are typically negative. With basal locations, leads V4–V6 are usually positive as with the concordant positive R waves during overt pre-excitation in left-sided accessory pathway conduction in the Wolff–Parkinson–White syndrome.

Faris et al. [22] evaluated the importance of AV optimization noting the transition from dyssynchronous to synchronous LV activation pattern by constructing activation time maps for both endo- and epicardial surfaces. They showed that during epicardial LV pacing, LV activation pattern remained asynchronous in both the endocardial and epicardial surface without possibility of merging with the intrinsic conduction which was traveling through the right bundle branch at both longer and shorter AV delay. At optimum AV delay, the electrical activation pattern becomes synchronous in both the epicardial and the endocardial surfaces, due to increasing degree of fusion between the wavefront propagating from the LV epicardial pacing site and the intrinsic wavefront originating from the right bundle branch (Fig. 2). This activation pattern led to a significant reduction of both inter- and intraventricular electrical asynchrony. Moreover, PR interval may vary in a patient time to time as a result of variation in autonomic tone. However, keeping AV delay to shorter side ensures RBBB morphology of paced QRS complex which is more acceptable than LBBB.

Conclusion

The results of our small study indicate that RA–LV transvenous pacing results in narrow paced QRS complex and can be an option for preserving LV function in patients with symptomatic LBBB at the cost of higher total procedure and fluoroscopic time.

Study limitations

The major limitation of the present study was the relatively small sample size. So, the results may not be generalized. We followed up the patients for short term (for only 6 months). Moreover, we presented single operator experience. So large multicenter study with longer term follow-up is needed for a conclusive result.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

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