Clinical implication of right ventricular to left ventricular interlead sensed electrical delay in cardiac resynchronization therapy

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Aims
To evaluate the clinical implication of right ventricular (RV) to left ventricular (LV) interlead sensed electrical delay (RV-LVs) and the relation to ventricular lead position in cardiac resynchronization therapy (CRT).

Methods and Results
Eighty-five consecutive CRT patients (mean age 66 ± 11 years) received LV lead prospectively targeted to the latest mechanical activated segment (concordant), assessed by two-dimensional speckle tracking radial strain (ST-RS) echocardiography. The RV lead was randomized to RV apex (n = 43) or RV high posterior septum (n = 42). Right ventricular to left ventricular interlead sensed electrical delay was obtained during the CRT implant procedure. Intraventricular dyssynchrony was evaluated by ST-RS echocardiography. Interventricular mechanical delay (IVMD) was measured by using pulse-wave Doppler. Separated by the median RV-LVs (82 ms), a long RV-LVs demonstrated more LV end-systolic volume (LVESV) reduction than a short RV-LVs (−27 ± 20 vs. −16 ± 22%; P = 0.02), 6 months after CRT (6FU). Right ventricular to left ventricular interlead sensed electrical delay correlated to IVMD (r = 0.50; P < 0.001) and intraventricular dyssynchrony (r = 0.25; P = 0.02) at baseline. Concordant LV leads (n = 61) demonstrated superior reduction of LVESV (P = ns) in the 6FU at 6FU were concordant LV lead (odds ratio, 3.210; P = 0.029) and IVMD (odds ratio, 1.028; P = 0.026).

Conclusion
Right ventricular to left ventricular interlead sensed electrical delay was not predictive to LV reverse remodelling affected by CRT at 6FU. Concordant LV leads demonstrated superior LV reverse remodelling at 6FU. Right ventricular to left ventricular interlead sensed electrical delay was irrespective of ventricular lead position and might be insufficient to target optimal LV lead position in CRT.


Keywords
Cardiac resynchronization therapy • Electrical delay • Concordant LV lead position • Right ventricular lead position • Echocardiography • Speckle tracking

Introduction
Cardiac resynchronization therapy (CRT) can improve heart failure (HF) symptoms and prolong survival in selected patients with left ventricular (LV) dysfunction and LV conduction system disorders.1,2 The clinical implication of measuring right ventricular (RV) to LV interlead sensed electrical delay (RV-LVs) during the CRT implant procedure is unclear.3,4 Further, the influence of ventricular lead position on RV-LVs has not been investigated. Optimal LV lead position may increase the beneficial effects of CRT.5 Left ventricular leads located at the latest activated segment (concordant LV lead), assessed by two-dimensional (2D) speckle tracking...
radial strain (ST-RS) echocardiography, have demonstrated superior improvement in LV contractile function and long-term prognosis in CRT.6,7 The clinical impact of RV lead position in CRT is uncertain.8–10 To evaluate the effects of two RV lead positions in CRT, the RV lead was randomized to RV apex (RV-A) or RV high posterior septum (RV-HS). Prior to CRT implantation, a study protocol using ST-RS echocardiography was performed to identify the LV segment with latest mechanical activation, and the LV lead was targeted to this LV segment. The clinical status and echocardiographic measurements on LV reverse remodelling and LV dyssynchrony from this study have been reported previously.11

We hypothesized that RV-LVs measured during the CRT implant procedure can predict the haemodynamic response to CRT, and that LV leads positioned at the segment with latest mechanical activation would demonstrate a longer RV-LVs. Thus, the primary aim of this sub-study analysis was to investigate whether RV-LVs at baseline can be used to predict LV reverse remodelling in CRT. The second objective was to explore the RV-LVs provided by different LV lead positions in CRT.

Methods

Patient population and study protocol

This single-centre study constituted 85 consecutive HF patients undergoing CRT between 2008 and 2010. Heart failure patients in New York Heart Association (NYHA) functional class III–IV despite optimal pharmacological treatment, LV ejection fraction (LVEF) ≤35%, and QRS duration ≥120 ms were included. The QRS morphologies included left bundle branch block (LBBB) and intraventricular conduction delay (IVCD), indicating a non-specific bundle branch block. Right bundle branch block (RBBB) was not included because no eligible CRT candidates with RBBB were referred in the inclusion period. The patients underwent echocardiographic examination prior to CRT implantation and 6 months after CRT. During the CRT implant procedure RV-LVs was obtained. In our study protocol, the LV lead was targeted to the segment with latest mechanical activation identified by ST-RS echocardiography. The RV lead was randomly assigned to RV-A or RV-HS. The regional ethics committee approved the study, and the patients were enrolled after informed and written consent.

Cardiac resynchronization therapy implantation procedure

A transvenous CRT implant procedure was performed under local anaesthesia. The coronary sinus was cannulated before inflation of a balloon catheter (Attain 6215, Medtronic, Minneapolis, MN, USA) and a venogram of the coronary vein tributaries was obtained. A coronary venous venogram by using fluoroscopic imaging in left anterior oblique (LAO), 30° view was divided into five equal segments: anterior, anterolateral, lateral, posterolateral, and posterior.5 The LV lead was targeted at the segment with latest mechanical activation, identified by ST-RS imaging prior to CRT implantation. A basal or midventricular LV lead location was used, and the apical region was avoided, confirmed by fluoroscopy in right anterior oblique view.5 The RV lead was positioned in RV-A or RV-HS according to the randomization. The RV-A lead position was located conventionally.12 A stylet-delivered technique was performed to obtain the RV-HS lead position, by using a pre-shaped curved stylet and lead pull-back from the pulmonary artery during counterclockwise rotation.13 The RV-HS lead position was confirmed by biplane fluoroscopy. The RV outflow tract was visualized in anteroposterior view, and a posterior lead direction was verified in LAO. The right atrial (RA) lead was located in RA appendage. Similar pacing leads and CRT devices were used in all patients (Medtronic, Minneapolis, MN, USA).

Right ventricular to left ventricular interlead sensed electrical delay

During the CRT implant procedure RV-LVs was obtained during intrinsic rhythm. Right ventricular to left ventricular interlead sensed electrical delay was obtained by connecting the RV lead to the atrial channel and LV lead to the ventricular channel of the programmer for device testing (Model 2090, Medtronic, Minneapolis, MN, USA).3,4 Right ventricular to left ventricular interlead sensed electrical delay was derived from the sensed electrogram signal in the RV and LV lead as the time delay (ms), measured automatically by the device-testing programmer (Figure 1). In comparison with the ventricular lead placements, RV-LVs was corrected for QRS duration and expressed as percentages. To account for beat-to-beat variations, as demonstrated in Figure 1, the average of three separate RV-LVs was used for statistical calculations.

Echocardiographic imaging

Transonic 2D echocardiography was performed by using a commercially available system (Vivid 7, General Electric Vingmed, Milwaukee, WI, USA) equipped with a 3.5-MHz transducer. The images were processed offline from digitally stored cine-loops (EchoPac 108.1.5, GE Medical Systems, Horten, Norway). Left ventricular end-diastolic volumes (LVEDV), LV end-systolic volumes (LVESV), and LVEF were calculated using biplane Simpson rule from images recorded in apical four- and two-chamber views.14 Patients with ≥15% LVESV reduction 6 months after CRT were considered as LV reverse remodelling responders.15 The relative improvements in LVEF, LVEDV, and LVESV at 6-month follow-up were evaluated as percentages. Intraventricular dyssynchrony was assessed by using ST-RS echocardiography from 2D images in mid-LV parasternal short-axis view during breath-hold.16 All images were recorded at frame rates ≥30 Hz. The LV endocard was traced in an end-systolic frame before speckle tracking software automatically generated time-strain curves of anteroseptal, anterior, lateral, posterior, inferior, and septal segments. Intraventricular dysynchrony was quantified as anteroseptal to posterior (AS-P) time delay.16 Prior to CRT implantation, the time delay of the anterior, lateral, and posterior segments from five consecutive beats was obtained and averaged. If possible, LV segments with strain <10% were excluded.17 Subsequently, the latest mechanical activation of anterior, anterolateral, lateral, posterolateral, or posterior segments was identified to target the LV lead position. Concordant LV lead was accredited if the LV lead position was located at the latest activated segment, or adjacent one. Interventricular dyssynchrony was evaluated from the interventricular mechanical delay (IVMD) by...
using pulse-wave Doppler of aortic flow velocity in apical long-axis view and pulmonic flow velocity in parasternal short-axis view. The pre-ejection time delay from QRS onset to opening of aortic and pulmonic valves were measured, and the IVMD was defined as the difference in time. Three consecutive cardiac cycles were used to measure LV volumes and LVEF, and five consecutive cardiac cycles were used to evaluate LV dyssynchrony. The mean value of the echocardiographic measurements obtained was used for statistical calculations. One single observer blinded to the lead positions performed all the echocardiographic analyses.

Statistical analysis

The current study was powered at 80% to detect a difference of 30% in number of patients demonstrating ≥50% AS-P delay reduction between RV-A and RV-HS, using a two-sided t-test at a significance level of 5%. Assuming 10% to be lost at follow-up, this would require 85 patients. All statistical analyses were performed by using SPSS software version 18.0 (SPSS Inc., Chicago, IL, USA). Continuous variables are presented as mean ± SD and were compared by using paired or unpaired Student’s t-test as appropriate. One-way analysis of variance (ANOVA) was used to compare more than two groups of continuous variables. Two-way ANOVA for repeated measurements were performed to assess subgroup interactions. Categorical data were listed as frequencies and percentages and were compared by using Pearson χ² or Fisher’s exact test. Linear regression analysis was conducted to identify correlation between continuous variables. Univariable and multivariable logistic regression analyses were performed to assess the relationship of seven independent variables to predict LV reverse remodelling 6 months after CRT. A P value of < 0.05 was considered statistical significant.

Results

Patient population

The baseline characteristics of the 85 HF patients in the study population [74 men (87%); mean age 66 ± 11 years] are presented in Table 1. All patients had advanced HF symptoms [65 in NYHA functional class III (76%) and 20 in NYHA functional class IV (24%)], wide QRS duration (169 ± 25 ms), and severely depressed LVEF (24 ± 4%). The patients were treated with angiotensin-converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers, β-blockers, or diuretics at maximum tolerated dosages.

Right ventricular to left ventricular interlead sensed electrical delay and response to cardiac resynchronization therapy

The median value of RV-LVs in the study population was 82 ms with interquartile range of 49–112 ms. Patients were separated by the RV-LVs median value and the clinical and haemodynamic effects of CRT were assessed. The improvement according to NYHA functional class (P < 0.001) at 6-month follow-up was greater in patients with long RV-LVs (3.3 ± 0.4 to 2.2 ± 0.4 vs. 3.2 ± 0.4 to 2.4 ± 0.5; P = 0.045). Similarly, the LV reverse remodelling induced by CRT was more pronounced in patients demonstrating long RV-LVs than short RV-LVs, for LVEDV (−20 ± 18 vs. −10 ± 20%; P = 0.02) and for LVESV (−27 ± 20 vs. −16 ± 22%; P = 0.02), but improvements in LVEF were not different (33 ± 25 vs. 28 ± 27%; P = 0.41). The relation between RV-LVs quartiles and the extent of reverse remodelling at 6-month follow-up are
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ARB, angiotensin receptor blockers. Interventricular mechanical delay; ACEI, angiotensin-converting enzyme inhibitors; peak radial strain measured by speckle tracking echocardiography; IVMD, LVEF, LV ejection fraction; LVEDV, LV end-diastolic volumes; LVESV, LV right ventricular (RV) to left ventricular (LV) interlead sensed electrical delay; NYHA, New York Heart Association; LBBB, left bundle branch block; RV-LVs, right ventricular to left ventricular (LV) interlead sensed electrical delay; LVEF, LV ejection fraction; LVEDV, LV end-diastolic volumes; LVESV, LV end-systolic volumes; AS-P delay, LV anteroseptal to posterior difference in time in peak radial strain measured by speckle tracking echocardiography; IVMD, interventricular mechanical delay; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers.

Table 1 Baseline characteristics of the study population

<table>
<thead>
<tr>
<th>Clinical variables</th>
<th>All (n = 85)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>66 ± 11</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>74 (87)</td>
</tr>
<tr>
<td>NYHA class III, n (%)</td>
<td>65 (76)</td>
</tr>
<tr>
<td>Ischaemic cardiomyopathy, n (%)</td>
<td>51 (60)</td>
</tr>
<tr>
<td>Electric delay</td>
<td></td>
</tr>
<tr>
<td>Sinus rhythm, n (%)</td>
<td>67 (79)</td>
</tr>
<tr>
<td>QRS (ms)</td>
<td>169 ± 25</td>
</tr>
<tr>
<td>LBBB, n (%)</td>
<td>73 (86)</td>
</tr>
<tr>
<td>RV-LVs, ms</td>
<td>84 ± 40</td>
</tr>
<tr>
<td>Echocardiographic variables</td>
<td></td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>24 ± 4</td>
</tr>
<tr>
<td>LVEDV (mL)</td>
<td>236 ± 63</td>
</tr>
<tr>
<td>LVESV (mL)</td>
<td>179 ± 50</td>
</tr>
<tr>
<td>AS-P delay (ms)</td>
<td>227 ± 95</td>
</tr>
<tr>
<td>IVMD (ms)</td>
<td>38 ± 23</td>
</tr>
<tr>
<td>Medical therapy</td>
<td></td>
</tr>
<tr>
<td>ACEI or ARB, n (%)</td>
<td>84 (99)</td>
</tr>
<tr>
<td>Beta-blocker, n (%)</td>
<td>81 (95)</td>
</tr>
<tr>
<td>Loop diuretics, n (%)</td>
<td>64 (75)</td>
</tr>
<tr>
<td>Spironolactone, n (%)</td>
<td>27 (32)</td>
</tr>
</tbody>
</table>

NYHA, New York Heart Association; LBBB, left bundle branch block; RV-LVs, right ventricular (RV) to left ventricular (LV) interlead sensed electrical delay; LVEF, LV ejection fraction; LVEDV, LV end-diastolic volumes; LVESV, LV end-systolic volumes; AS-P delay, LV anteroseptal to posterior difference in time in peak radial strain measured by speckle tracking echocardiography; IVMD, interventricular mechanical delay; ACEI, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers.

Right ventricular to left ventricular interlead sensed electrical delay and ventricular lead position

The distribution of the targeted LV segments from pre-operative ST-RS echocardiography was anterior (11%), anterolateral (6%), lateral (32%), posterolateral (31%), and posterior (21%). Concordant LV lead was achieved in 72% of the study population. In these, the LV lead was located at the segment with latest mechanical activation, or the adjacent one, of the five targeted LV segments. Baseline characteristics and RV-LVs according to LV and RV lead position are presented in Table 2. As demonstrated, RV-LVs was irrespective of LV lead concordance and RV lead position. In concordant LV leads, RV-LVs was similar in RV-A as compared with RV-HS (78 ± 38 ms; P = 0.08). Similarly, in discordant LV leads, RV-LVs was comparable in RV-A and RV-HS (81 ± 56 vs. 82 ± 33 ms; P = 0.94). The distributions of concordant LV leads in the RV-LVs quartiles were similar, first quartile (n = 14; 70%), second quartile (n = 17; 74%), third quartile (n = 15; 68%), and fourth quartile (n = 15; 75%; P = 0.96). Right ventricular to left ventricular interlead sensed electrical delay measured in concordant LV leads and LV leads located in the subsequent LV segments is presented in Figure 3. Left ventricular leads located at the segment next to concordant LV lead (n = 17) and LV leads located two segments from concordant LV lead (n = 7) demonstrated comparable RV-LVs as concordant LV leads (n = 61; P = 0.55).

Right ventricular to left ventricular interlead sensed electrical delay and left ventricular dyssynchrony

To further explore the interlead sensed electrical delay, the relation between RV-LVs and the baseline echocardiographic mechanical dyssynchrony was investigated. Right ventricular to left ventricular interlead sensed electrical delay correlated moderately to IVMD (r = 0.50; P < 0.001) and also weakly to AS-P delay (r = 0.25; P = 0.02). As expected, RV-LVs was associated with the intrinsic QRS-duration (r = 0.40; P < 0.001), but no strong correlation was found. The RV-LVs obtained in patients with LBBB morphology was significantly different as compared with the non-specific IVCD pattern (90 ± 39 vs. 48 ± 33 ms; P = 0.001). Right ventricular to left ventricular interlead sensed electrical delay was similar in ischaemic cardiomyopathy and non-ischaemic cardiomyopathy (83 ± 37 vs. 86 ± 45 ms; P = 0.70). Patients in sinus rhythm demonstrated comparable RV-LVs as those with permanent atrial fibrillation (AF; 85 ± 42 vs. 80 ± 34 ms; P = 0.65). Left ventricular dyssynchrony in patients with LBBB morphology compared with non-specific IVCD was presented in Figure 2 demonstrating a successive increase in LV reverse remodelling from the shortest quartile to the longest quartile. There was a weak but statistical significant correlation between RV-LVs and the relative changes in LVESV 6 months after CRT (r = −0.29; P = 0.009), with similar findings for LVEDV (r = −0.31; P = 0.006). Right ventricular to left ventricular interlead sensed electrical delay was not associated with the relative changes in LVEF at 6-month follow-up (r = 0.07; P = 0.56).

Figure 2 Relationship between intraoperative right ventricular to left ventricular interlead sensed electrical delay quartiles and reverse remodelling 6 months after cardiac resynchronization therapy. LVESV, left ventricular end-systolic volume.
Reverse remodelling according to ventricular lead position

The relative improvement in LV contractile function and reverse remodelling 6 months after CRT were evaluated according to LV and RV lead positions. Concordant LV leads demonstrated superior response to CRT as compared with discordant LV leads in LVEF (35 ± 25 vs. 21 ± 26%; P = 0.039), LVEDV (−18 ± 21 vs. −6 ± 15%; P = 0.011), and LVESV (−26 ± 22 vs. −11 ± 17%; P = 0.005). However, RV-A leads were comparable with RV-HS leads in the LV improvements effected by CRT in LVEF (30 ± 23 vs. 32 ± 29%; P = 0.64), LVEDV (−15 ± 19 vs. −15 ± 20%; P = 0.94), and LVESV (−21 ± 21 vs. −22 ± 23%; P = 0.90).

Logistic regression analyses

Univariable and multivariable logistic regression analyses were performed to assess seven different parameters to predict reverse remodelling defined as ≥15% reduction of LVESV at 6-month follow-up. The parameters assessed to predict LV reverse remodelling were QRS-duration, LBBB, RV-LVs, AS-P delay, IVMD, RV-A, and discordant LV lead position (Table 3). Interventricular mechanical delay and concordant LV leads were positively associated with LV reverse remodelling in univariable analysis. In multivariable analysis, discordant LV lead and IVMD were both independent predictors to LV reverse remodelling 6 months after CRT.

Discussion

The main findings in this prospective evaluation of RV-LVs in CRT were: (i) longer RV-LVs was associated with progressive increasing LV reverse remodelling, but the present study could not demonstrate that RV-LVs was predictive to the LV reverse remodelling as compared with discordant LV leads; (ii) RV-LVs was irrespective to LV lead concordance and RV lead position; (iii) a moderate-to-weak association was found between RV-LVs and baseline LV mechanical dyssynchrony, and RV-LVs was dependent on QRS morphology; (iv) discordant LV leads demonstrated superior improvements in LVEF and LV reverse remodelling as compared with discordant LV leads; however, similar haemodynamic effects were observed in both RV lead positions.

There are few data available on the clinical implication of measuring RV-LVs during the CRT implant procedure. Sassone et al.3 studied 52 CRT recipients and found that RV-LVs could not predict reverse remodelling defined as ≥15% reduction of LVESV.
Recent studies have demonstrated the superior clinical outcome when LBBB patients were the longer RV-LVs found in the patients with LBBB better predictor to CRT response than RV-LVs. An interesting observation was the longer RV-LVs corresponding to LV reverse remodelling,19 also confirmed by the current study, but a frequency used cut-off IVMD ≥ 40 ms demonstrates insensitivity to select CRT candidates. However, IVMD was not extensive in our study population (38 ms), the IVMD was a better predictor to CRT response than RV-LVs. An interesting observation was the longer RV-LVs found in the patients with LBBB as compared with those with the non-specific IVCD morphology. Recent studies have demonstrated the superior clinical outcome in patients with LBBB undergoing CRT.20 Left bundle branch block constituted 86% of our study population with similar distribution according to ventricular lead placement. However, LBBB was not predictive to reverse remodelling in the present study.

A similar approach to assess LV electrical dyssynchrony during the CRT procedure is the delay from the Q-wave on a surface ECG to intrinsic activation at the LV stimulation site (QLV).21 Recently, Gold et al.22 demonstrated a progressive increase in LV reverse remodelling with longer QLV in 426 CRT recipients, from the shortest quartile to the longest quartile (odds ratio, 3.21; P = 0.001). In addition, QLV was longer in LBBB than in non-LBBB patients (100 ± 35 vs. 73 ± 30 ms; P < 0.001), and similar findings were demonstrated in the current study. QLV might be different from RV-LVs because of variations in the sensing time delay on the RV lead relative to the Q-wave. However, a similar progressive trend was found on LV reverse remodelling with longer RV-LVs in the present study, and RV-LVs could be comparable with QLV.

There is increasing evidence that concordant LV lead location is favourable in CRT. Ypenburg et al.6 studied 257 CRT recipients retrospectively and demonstrated superior LV reverse remodelling and survival in patients with concordant LV leads as compared with those with discordant LV leads. In the present study, the LV lead was prospectively targeted to the latest activated segment by using ST-RS echocardiography. Patients with concordant LV lead demonstrated superior haemodynamic benefits of CRT, consistent with the previous empirical findings.6,7,10,17 To our surprise, RV-LVs obtained in the concordant LV lead group were similar to RV-LVs in the discordant LV lead group. Furthermore, RV-LVs was also comparable in LV leads located near the targeted LV segments. This could reflect that the pattern of electrical delay present in LV dysfunction with conduction abnormalities does not always correspond to the pattern of mechanical LV contraction. Previous studies have described the diversity of electrical dispersion in patients undergoing CRT.23 Moreover, the mechanical activation pattern in CRT recipients evaluated by ST-RS echocardiography has demonstrated a broad heterogeneity of the latest activated segment.24 Future investigations are needed to elucidate the relation between mechanical and electrical dyssynchrony in patients eligible for CRT.

To our knowledge, this is the first randomized study to explore the clinical impact of a single alternate RV lead position in CRT. Previous retrospective non-randomized studies have found contradicting effect of different RV lead positions in CRT.8–10 Our findings are consistent with previous reports that did not demonstrate differences in haemodynamic response provided by alternate RV lead location in CRT.

The relative low number of patients included from one single centre may limit the results in the present study. The study was designed to evaluate RV-LVs in two predefined LV and RV lead position in CRT. Only RV-LVs at the final ventricular lead position

### Table 3 Univariable and multivariable logistic regression analyses to identify predictors of left ventricular reverse remodelling

<table>
<thead>
<tr>
<th>Variables</th>
<th>Univariable OR (95% CI)</th>
<th>P value</th>
<th>Multivariable OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>QRS (per 1 ms)</td>
<td>1.014 (0.999–1.034)</td>
<td>0.169</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>LBBB</td>
<td>1.958 (0.450–8.522)</td>
<td>0.370</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>RV-LVs (per 1 ms)</td>
<td>1.013 (1.000–1.026)</td>
<td>0.059</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>AS-P delay (per 1 ms)</td>
<td>1.002 (0.997–1.007)</td>
<td>0.378</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>IVMD (per 1 ms)</td>
<td>1.030 (1.006–1.054)</td>
<td>0.014</td>
<td>1.028 (1.003–1.054)</td>
<td>0.026</td>
</tr>
<tr>
<td>RV-A</td>
<td>1.040 (0.414–2.615)</td>
<td>0.934</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Concordant LV lead</td>
<td>3.553 (1.288–9.801)</td>
<td>0.014</td>
<td>3.210 (1.125–9.157)</td>
<td>0.029</td>
</tr>
</tbody>
</table>

OR, odds ratio; CI, confidence interval; LBBB, left bundle branch block; RV-LVs, right ventricular (RV) to left ventricular (LV) interlead sensed electrical delay; AS-P delay, LV anterosetal to posterior time difference in peak radial strain measured by speckle tracking radial strain (ST-RS) echocardiography; IVMD, interventricular mechanical delay; RV-A, RV apical lead position; concordant LV lead, LV lead located at the segment with latest mechanical activation, or adjacent one, of the five targeted LV segments, identified by ST-RS echocardiography.
was obtained, and intra-patient variation was not investigated. Subsequently, some caution is needed in the interpretation of the subgroup interactions. However, the prospective trial design and the automaticity of measuring RV-LV differences strengthen the results in the present study.

Clinical implications
Conventionally, the LV lead is preferentially positioned in a lateral or posterolateral coronary tributary vein.6,7,10,17 By using ST-RS echocardiography prior to CRT implantation, the segment with latest contraction can be identified to target the LV lead placement. Subsequently, we advocate a preoperative evaluation of the LV activation pattern to guide an optimal LV lead position in CRT. However, the limitations of selective site LV lead placement are the available coronary vein anatomy, LV lead stability, and phrenic nerve stimulation.

Right ventricular to left ventricular interlead sensed electrical delay can readily be obtained during the CRT implant procedure by connecting the RV and LV lead to the device-programmer. However, RV-LVs might just indicate the intrinsic electrical LV dysynchrony present in HF patients with conduction abnormalities, in which a large electrical dispersion is favorable in CRT recipients. It has been proposed that measuring the LV electrical delay during the CRT implant procedure could be used to target an optimal LV lead position.22 The current study demonstrated similar RV-LVs in LV lead position according to mechanical activation delay, and thus might not be feasible for targeting an optimal LV lead placement in CRT.

Conclusion
Right ventricular to left ventricular interlead sensed electrical delay could not predict LV reverse remodelling 6 months after CRT and was dependent on QRS morphology. Concordant LV leads, evaluated by ST-RS echocardiography, provided superior improvement in LV contractile function and LV reverse remodelling. Randomized RV lead position to RV-A and RV-HS demonstrated comparable haemodynamic benefits of CRT. Right ventricular to left ventricular interlead sensed electrical delay was irrespective of LV lead position and thus might not be sufficient to target optimal LV lead position in CRT.

Conflict of interest: none declared.

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References

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A concealed left free wall accessory pathway in a patient with palpitations showed only intermittent conduction. Isoprenaline infusion and left ventricular pacing from a catheter placed in the posterolateral branch of the coronary sinus (panels A and B) resulted in more consistent pathway conduction. During mapping by a transseptal approach (panel B), bipolar signal from the ablation catheter showed an apparently single electrogram (panel C, beat 1). However, in beats with conduction block in the accessory pathway, a distinct atrial electrogram could be seen (panel C, beat 2). This ‘pseudodisappearance’ of the atrial electrogram is an excellent sign of ablation success.¹ Radiofrequency ablation in this location resulted in immediate loss of ventriculo—atrial conduction.

**Conflicts of interest:** none declared.

**Reference**


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