Impact of interventricular lead distance and the decrease in septal-to-lateral delay on response to cardiac resynchronization therapy

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Aims To investigate the influence of interlead distance and lead positioning on success of cardiac resynchronization therapy (CRT) in patients with advanced chronic heart failure and electrical dyssynchrony. Despite application of established selection criteria, 20–40% of the patients do not respond to CRT.

Methods and results We examined consecutive patients in whom CRT was implanted. Response to CRT was defined as a decrease in the left ventricular end-systolic volume ≥10% after 6 months. A comparison was made between patients who were responders to CRT and those who were non-responders. A univariate and stepwise multivariate logistic regression was performed with regard to predictors for response. Between January 2004 and January 2008, 174 patients who were treated with CRT were classified as responders \( n = 95 \) (55%) or non-responders \( n = 79 \) (45%). Responders had a significantly larger horizontal interlead distance on the lateral thoracic X-ray \( \text{OR} = 2.8 \) (1.2–6.6), \( P = 0.01 \), a septal-to-lateral delay \( > 60 \) ms \( \text{OR} = 4.9 \) (2.0–11.4), \( P < 0.001 \), non-ischaemic cardiomyopathy \( \text{OR} = 3.0 \) (1.3–6.9), \( P = 0.009 \), a left ventricular end-diastolic diameter > 67 mm \( \text{OR} = 4.2 \) (1.8–9.9), \( P = 0.001 \), angiotensin-converting enzyme inhibitor use \( \text{OR} = 8.1 \) (1.7–38.2), \( P = 0.008 \), and no tricuspid valve insufficiency \( \text{OR} = 6.9 \) (1.3–35.5), \( P = 0.02 \). Post-implantation responders had a significantly greater decrease in the intraventricular delay (septal-to-lateral delay \( 62 \pm 62 \) vs. \( 26 \pm 65 \) ms, \( P = 0.001 \)), but not in the interventricular mechanical delay.

Conclusion Larger interlead distance on the lateral thoracic X-ray, associated with positioning of the left ventricular lead in the posterior position, is associated with response after 6 months of follow-up. Furthermore, diminishing the septal-to-lateral delay is predictive for response.

KEYWORDS
Congestive heart failure; Artificial pacing; Cardiac resynchronization therapy

Introduction
Heart failure is the fastest growing cardiovascular diagnosis; the lifetime risk is estimated at nearly 20%. According to epidemiological studies, an estimated 22.5 million people suffer from heart failure worldwide.1 Intraventricular conduction disturbances are common and are associated with an increased mortality.2,3 Several randomized clinical trials have demonstrated the beneficial effects of cardiac resynchronization therapy (CRT) in patients with pharmacological refractory heart failure.3–10 These studies have shown an improvement in clinical (symptoms, exercise capacity, quality of life, and mortality) and echocardiographic endpoints (systolic function, left ventricular (LV) size, and mitral valve regurgitation). However, depending on the criteria used for the definition of response, 20–40% of the patients do not respond to CRT.

At present, the eligibility criteria for CRT are New York Health Association (NYHA) functional class III or IV despite optimal pharmacological treatment, LV ejection fraction ≤35%, left ventricular end-diastolic diameter (LVEDD) ≥55 mm, and wide QRS complex ≥130 ms.11 According to the recent literature, evident dyssynchrony by echocardiography was taken into account in case of a QRS duration ≤130 ms.12 A decrease in the left ventricular end-systolic volume (LVESV) of ≥10% has been shown to be predictive for lower long-term mortality and heart failure events.12,13
Positioning of the LV lead is often difficult due to the characteristics of the coronary venous anatomy, unacceptable electrical parameters in the target area, phrenic nerve stimulation, and/or other technical difficulties. The aim of the present study was to investigate the independent influence of interlead distance and thus lead position on response to CRT.

Methods

Patient population

Between January 2004 and January 2008, 194 consecutive patients with congestive heart failure received CRT. Patients who died prior to 6 months of follow-up and thus did not have a follow-up cardiac ultrasound to determine the changes in LVESV were not included in the analysis (n = 20). Response was defined as a decrease of ≥10% in LVESV after 6 months of follow-up. A total of 95 (55%) patients were classified as responders and the remaining 79 (45%) patients were non-responders.

Baseline assessment included patient history, physical examination, 12 lead-electrocardiogram (ECG), transthoracic echocardiography, exercise testing, radionuclide scanning, and coronary angiography. Clinical history and characteristics were retrieved using patients’ medical records. QRS duration was determined using ECG stored in medical records. A transthoracic echocardiography was made to determine whether there were signs of tricuspid valve or mitral valve regurgitation; the severity of regurgitation was graded semi-quantitatively from colour flow Doppler in the parasternal long axis and apical four-chamber images. Left ventricular end-diastolic and end-systolic diameters were determined by M-mode echocardiography in the parasternal long-axis view, according to the guidelines of the American Society of Echocardiography.14 Left ventricular end-diastolic and end-systolic volumes were measured using the modified biplane Simpson method, using the apical four-chamber and two-chamber views.15

Mechanical dyssynchrony was determined by tissue velocity imaging (TVI), using Echopac 6.1.3, General Electric Vivid 7. We evaluated the aortic pre-ejection time, interventricular mechanical delay (IVMD), and septal-to-lateral delay by TVI. An IVMD >40 ms was considered indicative of interventricular mechanical dyssynchrony.12 Intraventricular LV dyssynchrony was measured by calculating the septal-to-lateral delay. A mechanical delay >60 ms was considered indicative of intraventricular dyssynchrony.12

The positioning of right ventricular (RV) and LV lead was determined by chest X-rays in postero-anterior and lateral views, at maximal inspiration, typically on the day after device placement. The LV lead tip position was defined as posterior (within the posterior one-third of the heart shadow on the lateral radiograph), all other positions were defined as other. The interlead distance was measured on a digital radiology workstation, where the thoracic width, the cardiac width, the direct RV-LV electrode tip separation, and the horizontal and vertical components of LV-RV electrode tip separation were measured. (Figure 1) All measurements were divided by the cardiothoracic ratio to account for relative differences in cardiac and thoracic sizes between patients, thus creating the corrected interlead distance.

All available coronary sinus angiograms, in total 156 (90%), were examined to determine into which coronary vein the LV lead was positioned. Correlation with regard to lead position on thoracic X-ray was evaluated.

Follow-up

After implantation, all patients underwent echocardiography to determine the persistence of IVMD and/or septal-to-lateral delay. Thereafter, all patients were routinely seen every 6 months at the outpatient department. At all visits, patient history, medication use, physical examination, ECG, pacemaker interrogation, transthoracic echocardiography, a radionuclide scan, and exercise test were performed. At each CRT interrogation, data were stored both on computer disc and in a computerized ICD medical record database of the University Medical Center Groningen. A consistent protocol to standardize CRT and ICD programming was used. VV delay was set to 0 ms, and AV delay optimization was performed 2 weeks post-implantation. The duration of follow-up was computed from the time of CRT implantation until death or heart transplantation when applicable, or to the date when the last follow-up data were obtained. The determination of whether the patient was a responder or a non-responder was based on the LVESV measured by echocardiographic ultrasound at 6 months of follow-up.

Statistical analysis

Baseline descriptive statistics are presented as mean ± standard deviation (SD) or median (range) for continuous variables and numbers with percentages for categorical variables. Differences between variables in responders vs. non-responders were evaluated.
by Student’s t-test or Mann-Whitney U test, depending on the normality of the data, for continuous data and Fisher’s exact test or χ² test for categorical data.

We calculated adjusted odds ratios (ORs) of clinical characteristics, baseline drug therapy, and device characteristics with logistic regression, to identify predictors of CRT response. Linearity of the continuous variables with respect to the response variable was assessed by determining the quartiles of their distribution. Thereafter, ORs for each quartile were calculated. In the case of a linear trend in the estimated ORs, the variable was introduced in the model as continuous. If no linearity was shown, the variable was further categorized by taking together the quartiles with ORs.

A stepwise approach was used. The final model included all variables with P < 0.05; variables with P ≥ 0.05 in the multivariate model were excluded; interaction was investigated. In all analysis, P < 0.05 was considered statistically significant.

Results

Patients’ characteristics

We included 174 patients, of which 95 (55%) were responders to CRT after 6 months of follow-up. The baseline characteristics are shown in Table 1. Responders significantly more frequently suffered from non-ischaemic cardiomyopathy (NICMP), more frequently had an IVMD >40 ms, a

<table>
<thead>
<tr>
<th>Table 1 Baseline characteristics of responders vs. non-responders</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total population (n = 174)</strong></td>
</tr>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Male sex</td>
</tr>
<tr>
<td>History of AF</td>
</tr>
<tr>
<td>AF baseline</td>
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<tr>
<td>NYHA class</td>
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<tr>
<td>III</td>
</tr>
<tr>
<td>IV</td>
</tr>
<tr>
<td>Coronary artery disease</td>
</tr>
<tr>
<td>Previous MI</td>
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<tr>
<td>Non-ischaemic cardiomyopathy</td>
</tr>
<tr>
<td>DM</td>
</tr>
<tr>
<td>Hypertension</td>
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<tr>
<td>Systolic blood pressure</td>
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<tr>
<td>Diastolic blood pressure</td>
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<tr>
<td>eGFR (mL/min/1.73 m²)</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
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<tr>
<td>QRS &gt; 150</td>
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<tr>
<td>QRS configuration</td>
</tr>
<tr>
<td>LBBB</td>
</tr>
<tr>
<td>RBBB</td>
</tr>
<tr>
<td>Other</td>
</tr>
<tr>
<td>LVEF</td>
</tr>
<tr>
<td>Left atrial, long-axis (mm)</td>
</tr>
<tr>
<td>LVEDD (mm)</td>
</tr>
<tr>
<td>LVESD (mm)</td>
</tr>
<tr>
<td>LVEDV (mL)</td>
</tr>
<tr>
<td>LVESV (mL)</td>
</tr>
<tr>
<td>RV-TAPSE (mm)</td>
</tr>
<tr>
<td>MV regurgitationa</td>
</tr>
<tr>
<td>TI regurgitationa</td>
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<tr>
<td>QRS AV opening (ms)</td>
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<tr>
<td>QRS PV opening (ms)</td>
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<tr>
<td>IVMD (ms)</td>
</tr>
<tr>
<td>IVMD &gt; 40 ms</td>
</tr>
<tr>
<td>SL delay (ms)</td>
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<tr>
<td>SL &gt; 60 ms</td>
</tr>
<tr>
<td>Medication</td>
</tr>
<tr>
<td>ACE/ARB</td>
</tr>
<tr>
<td>β-blocker</td>
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<tr>
<td>Diuretics</td>
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<tr>
<td>Digoxin</td>
</tr>
</tbody>
</table>

ACE, angiotensin-converting enzyme; AF, atrial fibrillation; ARB, angiotensin receptor blocker; AV, aortic valve; DM, diabetes mellitus; eGFR, estimated glomerular filtration rate; LBBB, left bundle branch block; LVEDD, left ventricular end-diastolic diameter; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVESV, left ventricular end-systolic volume; IVMD, interventricular mechanical delay; MI, myocardial infarction; MV, mitral valve; NYHA, New York Health Association; PV, pulmonary valve; RBBB, right bundle branch block; RV, right ventricle; SL, septal to lateral; TV, tricuspidalis valve.

aModerate or severe.
The desired LV lead position during implantation was the postero-lateral vein. This was thought to be achieved in 138 patients (79%), whereas in 17 patients (10%), the LV lead was positioned in the antero-lateral vein and in 1 patient (1%) in the mid cardiac vein.

Analysis of the thoracic X-rays proved that the RV lead most frequently was placed in the apex and the LV lead in the posterior position. Responders significantly had a larger corrected horizontal interlead LV–RV distance, on the lateral X-ray. (Table 2) This coincided with the LV lead more frequently being positioned in the posterior position (Figure 2).

There was no correlation between coronary sinus vein positioning and response to CRT [79 (57%) responders vs. 59 (43%) non-responders, in whom the LV lead was positioned in the postero-lateral vein, and 7 (39%) vs. 11 (61%) positioned in other veins, respectively, \( P = 0.39 \)]. Analysis with regard to vein positioning and thoracic X-ray localization of the LV lead proved that a total of 32 patients (23%), in whom LV lead was thought to be positioned in the postero-lateral vein, eventually had the LV lead positioned in a different position after evaluation by thoracic X-ray, being anterior. A total of two patients (11%) in whom the LV lead was thought to be positioned in the antero-lateral vein, analysis of the thoracic X-ray proved that the LV lead was positioned in a posterior position.

Dysynchrony after implantation

The septal-to-lateral delay was significantly more reduced in responders after implantation (62 ± 62 vs. 26 ± 65, \( P = 0.001 \)) (Figure 3). Responders significantly less frequently had a septal-to-lateral delay >60 ms after implantation of the CRT device [11 (12%) vs. 28 (35%), \( P = < 0.0001 \)].

There was no significant difference in the reduction of IVMD between both groups (14 ± 38 vs. 13 ± 39, \( P = 0.39 \)).

Evaluation of the lead positioning showed that the septal-to-lateral delay significantly more diminishes in patients in whom the LV lead is positioned posteriorly. (Figure 3).

Response to therapy

Median follow-up was 21 ± 12 months. After 6 months of follow-up, 95 (55%) patients were defined as responders. The LVESV had decreased significantly in responders, whereas non-responders showed a significant increase in the LVESV (Figure 4A). The LVEF significantly increased in responders, whereas in the non-responders, it decreased slightly (Figure 4B). After 6 months of follow-up, 128 patients were responders with a decrease of one or more points in the NYHA class. Of the echocardiographic responders, 73 (78%) patients were NYHA responders, whereas

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Table 2  Lead positioning

<table>
<thead>
<tr>
<th>Localization LV lead</th>
<th>Total population (n = 174)</th>
<th>Responder (n = 95)</th>
<th>Non-responder (n = 79)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posterior</td>
<td>122 (70%)</td>
<td>73 (77%)</td>
<td>49 (62%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Other</td>
<td>49 (28%)</td>
<td>21 (22%)</td>
<td>28 (35%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Localization RV lead</th>
<th>Total population (n = 174)</th>
<th>Responder (n = 95)</th>
<th>Non-responder (n = 79)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apex</td>
<td>159 (91%)</td>
<td>91 (96%)</td>
<td>68 (86%)</td>
<td>0.01</td>
</tr>
<tr>
<td>RVOT</td>
<td>11 (6%)</td>
<td>2 (2%)</td>
<td>9 (11%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Measurements X-ray</th>
<th>Total population (n = 174)</th>
<th>Responder (n = 95)</th>
<th>Non-responder (n = 79)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CTR</td>
<td>0.56 ± 0.07</td>
<td>0.55 ± 0.06</td>
<td>0.57 ± 0.07</td>
<td>0.02</td>
</tr>
<tr>
<td>H-frontal X-ray (mm)</td>
<td>48 ± 32</td>
<td>50 ± 33</td>
<td>45 ± 32</td>
<td>0.29</td>
</tr>
<tr>
<td>V-frontal X-ray (mm)</td>
<td>82 ± 45</td>
<td>83 ± 42</td>
<td>81 ± 49</td>
<td>0.87</td>
</tr>
<tr>
<td>D-frontal X-ray (mm)</td>
<td>102 ± 40</td>
<td>103 ± 38</td>
<td>101 ± 41</td>
<td>0.75</td>
</tr>
<tr>
<td>H-lateral X-ray (mm)</td>
<td>127 ± 64</td>
<td>139 ± 62</td>
<td>112 ± 65</td>
<td>0.007</td>
</tr>
<tr>
<td>V-lateral X-ray (mm)</td>
<td>81 ± 45</td>
<td>81 ± 42</td>
<td>81 ± 48</td>
<td>0.99</td>
</tr>
<tr>
<td>D-lateral X-ray (mm)</td>
<td>161 ± 52</td>
<td>170 ± 51</td>
<td>151 ± 52</td>
<td>0.02</td>
</tr>
</tbody>
</table>

CTR, cardiothoracic ratio; D, direct interlead distance; H, horizontal; LV, left ventricular; RV, right ventricular; RVOT, right ventricular outflow tract; V, vertical.

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Figure 2  Interlead distance and lead position. *\(P < 0.0001\).
the remaining 21 (22%) echocardiographic responders did not improve in NYHA class. Of the echocardiographic non-responders, 55 (70%) patients improved one or more points in the NYHA class.

The multivariate analysis revealed that a horizontal interlead distance on the lateral X-ray >127 mm [OR 2.8 (1.2–6.6), \(P = 0.01\)], a septal-to-lateral delay >60 ms [OR 4.9 (2.0–11.4), \(P < 0.0001\)], NICMP [OR 3.0 (1.3–6.9), \(P = 0.009\)], LVEDD <67 mm [OR 4.2 (1.8–9.9), \(P = 0.001\)], angiotensin-converting enzyme (ACE) inhibitor use [OR 8.1 (1.7–38.2), \(P = 0.008\)], and no tricuspid valve regurgitation [OR 6.9 (1.3–35.5), \(P = 0.02\)] were related to response (Table 3).

Discussion

This study demonstrates that the interlead distance, in particular, the horizontal interlead distance on the lateral thoracic X-ray, is associated with response to CRT after 6 months of follow-up. The horizontal interlead distance correlated best with a posterior positioning of the LV lead. None of the other interlead distances were of influence on response. This suggests that the currently desired lateral placement of the LV lead is of less importance for response to CRT. Furthermore, response to CRT was associated with the diminishing of the septal-to-lateral delay during follow-up. This reduction of the septal-to-lateral delay was significantly more when the LV lead was positioned in the posterior position. This strengthens the body of evidence that relieving mechanical dyssynchrony is the basis for the efficacy of biventricular stimulation.

Response to cardiac resynchronization therapy

In the present study, 55% of the patients were responders to CRT, defined as a decrease in the LVESV of >10%. This is in accordance with the results found by Yu et al. The response rate was significantly higher when improvement in one or more NYHA class was evaluated. In accordance with our data, other studies found a poor correlation between clinical response and echocardiographic response to CRT.

Influence of lead positioning

In our analysis, a larger horizontal interlead distance on the lateral thoracic X-ray correlated with response. This coincided with a posterior positioning of the LV lead and offers an advantage over other lead positions. Our analysis proved that the desired coronary vein (postero-lateral) is sometimes difficult to predict with the current fluoroscopic views. In our cohort, in a large percentage (22%), the LV lead was placed in a different location than predicted from the coronary venogram. This is most likely due to variable coronary venous anatomy, which does not always follow strict rules such as coronary arteries. By changing the current fluoroscopic views used for the implantation of the LV lead left anterior oblique (LAO) 30° to 40° to a fluoroscopic view LAO 90°, frequently used in coronary angiography, the best posterior vein can be selected. Hereby, it should be taken into account that the choice of LV electrode site for best resynchronization is often difficult because of the characteristics of the coronary venous anatomy, unacceptable electrical parameters in the target area, phrenic nerve stimulation, or other technical difficulties. Earlier studies

Figure 3 Changes in septal-to-lateral delay between responders and non-responders (A) and with regard to lead positioning (B). \(*P = 0.001\) and \(**P = 0.02\).

Figure 4 Changes in haemodynamic parameters during follow-up. Changes in left ventricular end-systolic volume (A) and in left ventricular ejection fraction (B) in responders and non-responders. \(*P < 0.01\).
have shown the lateral or postero-lateral part of the left ventricle as the site that provides greatest acute haemodynamic benefit, particularly for dP/dt.\textsuperscript{17–20} Butter \textit{et al.}\textsuperscript{19} found that the acute haemodynamic effects were significantly better when the LV lead was positioned in the free wall compared with an anterior positioning of the LV lead. Along the same lines, Rossillo \textit{et al.}\textsuperscript{21} showed that positioning of the LV lead in the lateral and postero-lateral coronary veins was associated with significant improvement in the functional capacity and greater improvement in the LV function when compared with the anterior coronary vein location. Heist \textit{et al.}\textsuperscript{20} analysed the effect of a larger horizontal interlead distance, whereby especially the interlead distance on the lateral X-ray influenced acute haemodynamics. No information, however, over objective long-term response is available. We could now demonstrate that this simple parameter can be used to predict response to biventricular stimulation. The vertical interlead difference between RV and LV, neither frontal nor lateral X-ray, was of significance with regard to response rate. This coincides with the acute haemodynamic results found by Gold \textit{et al.}\textsuperscript{22}

Bax \textit{et al.}\textsuperscript{23,24} were the first to determine that diminishing of the septal-to-lateral delay by biventricular pacing was associated with response to CRT. In analogy with our results, they found that the septal-to-lateral delay significantly decreased with CRT. In contrast to our results, Gasparini \textit{et al.}\textsuperscript{25} could not find a significant difference in lead positioning and amount of clinical responders. It should be noted, however, that they found a significant correlation with an increase in LVEF, which, in several studies,\textsuperscript{13} was correlated with LV remodelling and patient outcome (in contrast to clinical parameters).

\textbf{Influence of right ventricular lead localization}

In our population, non-responders more frequently had the RV lead localized in the RV outflow tract (RVOT); this observation is in accordance with the results found by Van Gelder \textit{et al.},\textsuperscript{26} who found a significant difference in LV dP/dt max in 82 patients with biventricular pacing with placement of the RV lead in the apex being superior to placement in the septal wall. Therefore, it should be taken into account that this difference disappeared after V-V optimization. Possibly, a larger interlead separation is created by placing the RV lead in the apex.\textsuperscript{20}

\textbf{Dyssynchrony after implantation}

Responders had a significantly larger decrease in the intraventricular mechanical delay after implantation of the CRT. Even so, positioning of the LV lead in the posterior position led to a greater decrease of the septal-to-lateral delay. The finding that the intraventricular mechanical delay decreases in responders in contrast to the non-responders is in accordance with results found by others.\textsuperscript{23,24} The fact, however, that positioning of the LV lead in the posterior position more frequently was associated with a decrease of intraventricular mechanical delay has never been described previously.

\textbf{Predictors for response}

In the present study, we found that predictors for response were in addition to a large horizontal interlead distance on the lateral thoracic X-ray: a septal-to-lateral delay >60 ms, NICMP, a smaller LVEDD, the use of an ACE inhibitor, and no tricuspid valve regurgitation. These results are in accordance with results found by others.\textsuperscript{10,12,27} Several studies have proven that patients with NICMP have a better chance for response to CRT than those with ischaemic cardiomyopathy.\textsuperscript{28–31} Sogaard \textit{et al.}\textsuperscript{26} found a difference in contraction delay between ischaemic and non-ischaemic patients with the greatest contraction delay in the lateral and posterior LV walls for NICMP and in the septum and inferior walls for those with ischaemic cardiomyopathy.\textsuperscript{28} Possibly, this difference in conduction delay could explain the influence of lead positioning. Further studies have shown that positioning of the LV lead in previous scar tissue is associated with non-response.\textsuperscript{29,30} In accordance with our results, Richardson \textit{et al.}\textsuperscript{27} found that patients with non-ischaemic cardiomyopathy responded more frequently to CRT. Bax \textit{et al.}\textsuperscript{12} found that a septal-to-lateral delay of >60 ms is predictive for response to CRT, which we could confirm this in our study.

\textbf{Limitations}

The main limitation is the relatively small number of patients, partly due to missing data due to relocation of patients or death prior to 6 months of follow-up and therefore not included in the analysis. This limits the power to adjust for multiple variables.

\begin{table}
\centering
\caption{Univariate and stepwise multivariate logistic regression analyses with regard to predictor responder}
\begin{tabular}{llllll}
\hline
Variable & Univariate analysis & & Multivariate analysis\textsuperscript{a} & & \\
 & Odds ratio (95\% CI) & P-value & Odds ratio (95\% CI) & P-value & \\
\hline
H-lateral X-ray >127 mm & 2.6 (1.4–5.8) & 0.003 & 2.8 (1.2–6.6) & 0.01 & \\
SL-delay >60 ms & 4.0 (2.0–8.0) & <0.0001 & 4.9 (2.0–11.4) & <0.0001 & \\
NICMP & 3.0 (1.6–5.6) & <0.0001 & 3.0 (1.3–6.9) & 0.009 & \\
LVEDD <67 mm & 2.4 (1.3–4.4) & 0.006 & 4.2 (1.8–9.9) & 0.001 & \\
ACE-inhibitor & 5.5 (1.5–20.2) & 0.01 & 8.1 (1.7–38.2) & 0.008 & \\
No TV regurgitation & 4.2 (1.1–15.7) & 0.04 & 6.9 (1.3–35.5) & 0.02 & \\
\hline
\end{tabular}
\textsuperscript{a}There is no significant interaction between parameters in the end model.
\end{table}
Conclusion
The present study highlights that lead positioning is predictive for response to CRT. Larger interlead distance on the lateral thoracic X-ray, associated with the positioning of the LV lead in the posterior position, is associated with response after 6 months of follow-up. Furthermore, diminishing the septal-to-lateral delay is predictive for response.

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Conflict of interest: D.J.V.V. is currently conducting research with Medtronic and has received consultancy fees from Medtronic. I.C.V.G. has received research grants from Medtronic, Biotronik, Boston Scientific and St Jude Medical.

References