Left ventricular lead placement in the latest activated region guided by coronary venous electroanatomic mapping

Masih Mafi Rad1*, Yuri Blaauw1, Trang Dinh1, Laurent Pison1, Harry J. Crijns1, Frits W. Prinzen2, and Kevin Vernooy1

1Department of Cardiology, Maastricht University Medical Center, P. Debyelaan 25, PO Box 5800, 6202 AZ Maastricht, The Netherlands; and 2Department of Physiology, Cardiovascular Research Institute Maastricht, PO Box 616, Maastricht 6200 MD, The Netherlands

Received 10 October 2013; accepted after revision 22 July 2014; online publish-ahead-of-print 3 September 2014

**Aim**

Left ventricular (LV) lead placement in the latest activated region is an important determinant of response to cardiac resynchronization therapy (CRT). We investigated the feasibility of coronary venous electroanatomic mapping (EAM) to guide LV lead placement to the latest activated region.

**Methods and results**

Twenty-five consecutive CRT candidates with left bundle-branch block underwent intra-procedural coronary venous EAM using EnSite NavX. A guidewire was used to map the coronary veins during intrinsic activation, and to test for phrenic nerve stimulation (PNS). The latest activated region, defined as the region with an electrical delay >75% of total QRS duration, was located anterolaterally in 18 (basal, n = 10; mid, n = 8) and inferolaterally in 6 (basal, n = 3; mid, n = 3). In one patient, identification of the latest activated region was impeded by limited coronary venous anatomy. In patients with >1 target vein (n = 12), the anatomically targeted inferolateral vein was rarely the vein with maximal electrical delay (n = 3). A concordant LV lead position was achieved in 18 of 25 patients. In six patients, this was hampered by PNS (n = 4), lead instability (n = 1), and coronary vein stenosis (n = 1).

**Conclusion**

Coronary venous EAM can be used intraprocedurally to guide LV lead placement to the latest activated region free of PNS. This approach especially contributes to optimization of LV lead electrical delay in patients with multiple target veins. Conventional anatomical LV lead placement strategy does not target the vein with maximal electrical delay in many of these patients.

**Keywords**

Cardiac resynchronization therapy • Left ventricular lead placement • Electroanatomic mapping • Left ventricular electrical activation • Feasibility

---

**Introduction**

Cardiac resynchronization therapy (CRT) reduces morbidity and mortality and reverses left ventricular (LV) remodelling in heart failure patients with LV systolic impairment and electrical dysynchrony.1–3 Despite the striking effectiveness of CRT, a substantial proportion of apparently eligible patients fail to benefit.4 Part of this reduced benefit has been attributed to a suboptimal LV lead position.4 There is increasing evidence to suggest that positioning of the LV lead in the region of latest electrical activation provides superior clinical outcome.5,6 The conventional LV lead placement strategy involves an anatomical approach, targeting a coronary venous branch situated on the posterolateral wall.7 This strategy is based on the contention that the posterolateral wall is typically the latest activated site of the ventricle in patients with left bundle-branch block (LBBB). However, studies have shown a considerable variability in the ventricular activation pattern in LBBB, resulting in inter-individual variability in the optimal pacing site.8–10 Therefore, a more patient-specific physiological approach focused on achieving maximal LV lead electrical delay may improve CRT response. Electroanatomic mapping (EAM) is typically used in the electrophysiology lab to guide diagnostic or ablation procedures of cardiac arrhythmias.

---

*Corresponding author. Tel: +31 433871613; fax: +31 433877081. E-mail address: masih.mafirad@mumc.nl

Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2014. For permissions please email: journals.permissions@oup.com.
What’s new?
- We describe a novel method of coronary venous electroanatomic mapping that utilizes the EnSite NavX system in combination with a guidewire that allows mapping and pacing of the coronary veins.
- This technique can be used at the time of cardiac resynchronization therapy implantation to assess left ventricular (LV) electrical activation pattern and guide LV lead placement to the latest activated region.
- The technique also enables comprehensive identification of sites that are free of phrenic nerve stimulation.

Recently, the technique has also been applied during CRT implantation to determine the electrical activation pattern of the coronary venous system.11,12 Final LV lead position, however, depends on several factors such as cardiac venous anatomy, performance and stability of the lead, and the absence of phrenic nerve stimulation (PNS). The feasibility of coronary venous EAM to guide LV lead placement to the latest activated region during CRT implantation remains to be assessed. The aim of the present study was to assess the feasibility of positioning the LV lead in the latest activated region guided by coronary venous EAM in patients undergoing CRT implantation.

Methods

Population
From December 2012 to May 2013, consecutive patients referred for CRT device implantation, with LV ejection fraction (LVEF) < 35%, New York Heart Association (NYHA) functional class 2, 3, or ambulatory 4, and LBBB according to European Society of Cardiology (ESC) guidelines,13 were prospectively enrolled. The study complied with the Declaration of Helsinki. Our locally appointed ethics committee approved the study protocol and waived the need for informed consent.

Electroanatomic mapping and left ventricular lead placement
All patients underwent intra-procedural coronary venous three-dimensional (3D) EAM using EnSite NavX (St Jude Medical). Prior to LV lead placement, an occlusive venogram was recorded under fluoroscopy in right anterior oblique (RAO) and left anterior oblique (LAO) view for optimal visualization of the coronary veins. Subsequently, a 0.014 inch guidewire (Vision Wire, Biotronik SE & Co.KG) was inserted into the coronary sinus and connected to EnSite NavX along with the surface electrocardiogram (ECG). This guidewire has complete polyethylene isolation except for 15 mm at the distal ‘J’-shaped tip and 30 mm at the proximal end permitting unipolar sensing and pacing.14 The guidewire was used to map all target veins located on the anterolateral or inferolateral LV wall as defined by the American Heart Association (AHA) 17-segment heart model.15 A 3D electrical activation map was constructed during intrinsic ventricular activation. Electrical delay was measured in milliseconds from QRS onset on the surface ECG to the peak negative slope on the unipolar intra-cardiac electrogram (EGM) and expressed as a percentage of total QRS duration. All measurements were performed in EnSite NavX at a screen speed of 400 mm/s. The latest activated region of the LV was defined as the region with an electrical delay comprising > 75% of total QRS duration. This definition was chosen because epicardial mapping via the coronary veins is limited by coronary venous anatomy, which means that some areas cannot be mapped because they do not contain any veins. Therefore, the latest activated LV region can only be identified using coronary venous mapping by relating the electrical activation time of the anatomical region to its timepoint within the QRS complex. Candidate pacing sites were tested for PNS by 10 V unipolar pacing on the tip of the guidewire and regions with PNS or absence thereof were annotated on the EAM using EnSite NavX. After the mapping procedure, the LV lead was connected to EnSite NavX for real-time visualization and navigation of the lead to the latest activated region free of PNS in the created EAM. Standard bipolar leads of various vendors were used. Figure 1 shows an example of a coronary venous EAM together with the results of PNS testing and the final LV lead position. The latest activated region and final LV lead position were classified according to the AHA 17-segment heart model by detailed evaluation of pre-implantation venograms and post-implantation LV lead fluoroscopy images (Figure 2). The final LV lead position was described as concordant if the lead was located in the same myocardial segment as the latest activated region.

Statistical analysis
Continuous variables are expressed as mean ± SD. Categorical variables are expressed as observed number and percentage values. Continuous variables were compared using Mann–Whitney U test. Statistical significance was accepted at the 95% confidence interval (P < 0.05). Statistical analysis was performed using SPSS version 20.0 (SPSS Inc.) software.

Results

Patient characteristics
Twenty-five consecutive patients with LBBB referred for CRT device implantation were included in this study. The patient characteristics are described in Table 1.

Cardiac resynchronization therapy implantation
In 21 patients, a de novo CRT defibrillator was implanted. Two patients were upgraded from an implantable cardioverter-defibrillator to a CRT defibrillator and two patients received a CRT pacemaker. Left ventricular lead implantation was successful in all patients. There were no procedural complications.

Coronary venous electroanatomic mapping
In all patients, occlusive coronary venography revealed at least one target vein on the anterolateral or inferolateral LV wall. In 12 of 25 patients, two target veins were available. All target veins were successfully mapped. Three-dimensional electrical activation maps were generated from an average of 46 ± 28 unique anatomical points. Mapping time was 19 ± 6 min and fluoroscopy time during the entire procedure was 20 ± 6 min. Figure 3 shows the distribution of the latest activated regions in all patients. The latest activated region was located anterolaterally in 18 (basal, n = 10; mid, n = 8) and inferolaterally in 6 (basal, n = 3; mid, n = 3). In one patient (No. 7), identification of the latest activated region was impeded by limited coronary venous anatomy.
Figure 4 shows the distribution of the final LV lead position in all patients. The LV lead was positioned anterolaterally in 16 (basal, n = 9; mid, n = 7) and inferolaterally in 9 (basal, n = 4; mid, n = 5). The final LV lead position was concordant with the latest activated region in 18 of 25 patients. In six patients, concordant LV lead placement was hampered by PNS (n = 4, one of them depicted in Figure 5), lead instability (n = 1), and coronary vein stenosis (n = 1). In the patient in whom the latest activated region could not be identified (No. 7), the LV lead was positioned at the site where maximal electrical delay was measured with coronary venous EAM. An overview of the latest activated regions and final LV lead positions of all patients together with the corresponding electrical delays at these sites is provided in Table 2. In patients with a concordant LV lead position, electrical delay was $132 \pm 15$ ms ($85 \pm 6\%$ of QRS duration) at the latest activated region and $128 \pm 13$ ms ($83 \pm 5\%$ of QRS duration) at the final lead position ($P = 0.17$). In patients with a discordant LV lead position, electrical delay was $125 \pm 10$ ms ($84 \pm 3\%$ of QRS duration) at the latest activated region and $94 \pm 15$ ms ($63 \pm 7\%$ of QRS duration) at the final lead position ($P = 0.002$). Left ventricular lead electrical delay was significantly longer in patients with a concordant compared with patients with a discordant LV lead position ($P < 0.001$).

In 9 of 12 patients with two available target veins (Nos. 1, 11, 12, 13, 14, 15, 18, 23, and 24), the latest activated region was located in a
different vein than the anatomically targeted inferolateral vein (Table 2). In these patients, maximal electrical delay was 89 $\pm$ 14 ms (58 $\pm$ 6% of QRS duration) in the anatomically targeted vein vs. 133 $\pm$ 15 ms (87 $\pm$ 5% of QRS duration) at the latest activated region ($P < 0.001$). The LV lead was positioned in the latest activated region in seven of nine patients (Nos. 1, 11, 12, 14, 15, 23, and 24). In these patients, maximal electrical delay was 89 $\pm$ 16 ms (57 $\pm$ 7% of QRS duration) in the anatomically targeted vein vs. 131 $\pm$ 14 ms.
(85 ± 4% of QRS duration) at the final LV lead position ($p = 0.001$).

In the other two patients (Nos. 13 and 18), coronary vein stenosis and PNS hampered concordant LV lead placement. Figure 6 illustrates the additional value of mapping-guided LV lead placement as compared with the conventional anatomical LV lead placement approach. In this example, the coronary venous EAM of Patient No. 24 is shown where a pure anatomical approach would have resulted in a suboptimal LV lead position in the inferolateral vein with a maximal electrical delay of only 90 ms. However, additional mapping of the small anterolateral vein resulted in a potentially far better LV lead position with an electrical delay close to 140 ms.

Discussion

Pacing at the latest activated region of the LV appears to improve the outcome of CRT. The present study demonstrates that coronary venous EAM using EnSite NavX in combination with a mapping guidewire can be used intraprocedurally to guide LV lead placement to the region of latest electrical activation. In addition, the potential of this mapping approach to facilitate comprehensive identification of regions free of PNS is demonstrated.

Targeted left ventricular lead placement at the latest activated left ventricular region

Several studies have investigated the relationship between pacing in the region of latest activation and the response to CRT. Studies on mechanical activation suggest superior CRT outcome when the LV lead position coincides with the region of latest mechanical contraction compared with discordant positions.16–22 Two recent prospective single-centre trials randomized a total of 407 patients to speckle-tracking echocardiography-guided LV lead placement targeting the site of latest mechanical activation or to standard unguided LV lead placement. In both trials, patients with a LV lead position concordant with the site of latest activation had a higher echocardiographic response rate, more clinical responders, and a reduced risk of mortality and heart failure hospitalization.23,24 Studies focusing on the electrical activation pattern have demonstrated that a greater delay in time from onset of the QRS complex to the local sensed LV lead EGM (Q-LV) is also associated with a greater likelihood of benefit from CRT.25,26 Recently, Zanon et al. measured the Q-LV interval of various pacing sites within patients, and evaluated the haemodynamic effect of pacing at the different sites by invasive measurement of $LVdP/dt_{max}$. Pacing the LV at the latest activated site resulted in greatest haemodynamic improvement.6 The choice between targeting the region of latest electrical or latest mechanical activation is still a matter of debate and definitive randomized trials that support the use of either approach to reliably select an optimal LV pacing site are lacking. Accordingly, current ESC
Feasibility of coronary venous electroanatomic mapping to guide left ventricular lead placement to the latest activated region

Our observation that the location of the latest activated region varies between patients agrees with previous EAM studies in patients with heart failure and LBBB,9–10 and emphasizes the need for a patient-tailored approach to optimize LV lead positioning. The present study demonstrates that coronary venous EAM using EnSite NavX in combination with a mapping guidewire is a feasible method to tailor LV lead placement to the individual patient at the time of implantation. Using this approach, we were able to position the LV lead in the latest activated region in >70% of our patients. Conventional LV lead placement strategy involves an anatomical approach, targeting a coronary vein situated on the posterolateral wall.

Table 2 Overview of the latest activated regions, final LV lead positions, and empirical target veins of all patients together with the corresponding electrical delays

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>No. of target veins</th>
<th>Latest activated region</th>
<th>Electrical delay (ms)</th>
<th>Final LV lead position</th>
<th>Electrical delay (ms)</th>
<th>Reason for discordance</th>
<th>Empirical target vein for LV lead</th>
<th>Maximal electrical delay (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>B-AL</td>
<td>139 (93%)</td>
<td>B-AL</td>
<td>131 (87%)</td>
<td>–</td>
<td>IL vein</td>
<td>90 (60%)</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>B-AL</td>
<td>136 (86%)</td>
<td>B-AL</td>
<td>126 (80%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>M-IL</td>
<td>122 (79%)</td>
<td>M-IL</td>
<td>122 (79%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
<td>M-AL</td>
<td>131 (84%)</td>
<td>M-AL</td>
<td>128 (82%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>5</td>
<td>1</td>
<td>B-IL</td>
<td>131 (85%)</td>
<td>B-IL</td>
<td>128 (83%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>6</td>
<td>1</td>
<td>M-AL</td>
<td>110 (85%)</td>
<td>M-IL</td>
<td>72 (56%)</td>
<td>PNS</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>7</td>
<td>1</td>
<td>Not identified</td>
<td>–</td>
<td>M-IL</td>
<td>103 (63%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>8</td>
<td>2</td>
<td>M-AL</td>
<td>126 (87%)</td>
<td>M-AL</td>
<td>126 (87%)</td>
<td>–</td>
<td>AL vein</td>
<td>–</td>
</tr>
<tr>
<td>9</td>
<td>1</td>
<td>B-AL</td>
<td>137 (84%)</td>
<td>M-AL</td>
<td>103 (63%)</td>
<td>Lead instability</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>10</td>
<td>1</td>
<td>B-AL</td>
<td>156 (96%)</td>
<td>B-AL</td>
<td>149 (92%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>11</td>
<td>2</td>
<td>B-AL</td>
<td>130 (85%)</td>
<td>B-AL</td>
<td>128 (83%)</td>
<td>–</td>
<td>IL vein</td>
<td>70 (45%)</td>
</tr>
<tr>
<td>12</td>
<td>2</td>
<td>B-AL</td>
<td>119 (91%)</td>
<td>B-AL</td>
<td>119 (91%)</td>
<td>–</td>
<td>IL vein</td>
<td>72 (55%)</td>
</tr>
<tr>
<td>13</td>
<td>2</td>
<td>M-AL</td>
<td>124 (86%)</td>
<td>B-IL</td>
<td>94 (65%)</td>
<td>Coronary vein stenosis</td>
<td>IL vein</td>
<td>94 (65%)</td>
</tr>
<tr>
<td>14</td>
<td>2</td>
<td>B-AL</td>
<td>149 (93%)</td>
<td>B-AL</td>
<td>138 (86%)</td>
<td>–</td>
<td>IL vein</td>
<td>106 (66%)</td>
</tr>
<tr>
<td>15</td>
<td>2</td>
<td>M-AL</td>
<td>116 (91%)</td>
<td>M-AL</td>
<td>111 (87%)</td>
<td>–</td>
<td>IL vein</td>
<td>80 (63%)</td>
</tr>
<tr>
<td>16</td>
<td>1</td>
<td>M-IL</td>
<td>131 (78%)</td>
<td>M-IL</td>
<td>131 (78%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>17</td>
<td>1</td>
<td>M-AL</td>
<td>111 (76%)</td>
<td>M-AL</td>
<td>111 (78%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>18</td>
<td>2</td>
<td>M-AL</td>
<td>118 (79%)</td>
<td>M-IL</td>
<td>94 (63%)</td>
<td>PNS</td>
<td>IL vein</td>
<td>94 (63%)</td>
</tr>
<tr>
<td>19</td>
<td>2</td>
<td>M-IL</td>
<td>130 (83%)</td>
<td>B-AL</td>
<td>116 (74%)</td>
<td>PNS</td>
<td>IL vein</td>
<td>–</td>
</tr>
<tr>
<td>20</td>
<td>1</td>
<td>B-AL</td>
<td>128 (83%)</td>
<td>B-AL</td>
<td>127 (82%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>21</td>
<td>1</td>
<td>B-IL</td>
<td>143 (80%)</td>
<td>B-IL</td>
<td>141 (79%)</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>22</td>
<td>1</td>
<td>B-AL</td>
<td>128 (85%)</td>
<td>M-AL</td>
<td>87 (57%)</td>
<td>PNS</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>23</td>
<td>2</td>
<td>M-AL</td>
<td>160 (84%)</td>
<td>M-AL</td>
<td>155 (81%)</td>
<td>–</td>
<td>IL vein</td>
<td>113 (59%)</td>
</tr>
<tr>
<td>24</td>
<td>2</td>
<td>B-AL</td>
<td>140 (83%)</td>
<td>B-AL</td>
<td>133 (79%)</td>
<td>–</td>
<td>IL vein</td>
<td>90 (54%)</td>
</tr>
<tr>
<td>25</td>
<td>2</td>
<td>B-IL</td>
<td>103 (80%)</td>
<td>B-IL</td>
<td>101 (78%)</td>
<td>–</td>
<td>IL vein</td>
<td>–</td>
</tr>
</tbody>
</table>

Patient numbers correspond with the numbers provided in Figures 3 and 4. Electrical delay is reported in milliseconds (percentage of total QRS duration). In Patient No. 6, the anterolateral vein anastomosed with the inferior inter-ventricular vein, which made it possible to position the LV lead inferolaterally (via the anastomosis) despite the patient having only one target vein on the anterolateral LV wall. In Patient No. 7, identification of the latest activated region was impeded by limited coronary venous anatomy. Patient No. 8 had two target veins on the anterolateral wall. For patients with two target veins, the second column from the right reports the target vein for the LV lead if the conventional anatomical lead placement approach would be used. The right-most column provides the maximal electrical delay measured within that vein. B, basal; M, mid; AL, anterolateral; IL, inferolateral; PNS, phrenic nerve stimulation.
Approximately half the patients in our study cohort had more than one target vein available on the lateral LV free wall. In most of these patients, the empirically targeted posterolateral vein was not the vein with maximal electrical delay, which is consistent with the findings of a previous study that also used coronary venous EAM to assess LV epicardial activation. In the present study, coronary venous EAM resulted in targeting of an alternative vein in many of these patients, significantly increasing LV lead electrical delay from an average of 57–85% of total QRS duration. In addition, in patients with a single target vein, coronary venous EAM enabled targeting of the vein segment with maximal electrical delay. These results demonstrate the additional value of coronary venous EAM-guided LV lead placement as compared with the conventional anatomical LV lead placement approach for optimization of LV lead electrical delay.

The use of EnSite NavX to create an EAM of the coronary veins at the time of CRT implantation has previously been described by Ryu et al. In addition, Del Greco et al. demonstrated that lead placement guided by the 3D navigation system of EnSite NavX is beneficial in terms of reducing X-ray exposure during CRT implantation. Recently, Fujiwara et al. compared electrical and mechanical dyssynchrony using coronary venous EAM and speckle-tracking echocardiography. However, sample sizes of these studies were small, LV lead placement was not targeted at the latest activated region, and constraints imposed by coronary venous anatomy for identifying the latest activated LV region were not taken into account. Therefore, the present study is the first to demonstrate, in a larger and representative CRT population, the feasibility of positioning the LV lead in the latest activated region guided by coronary venous EAM.

Unfortunately, concordant LV lead placement was still hampered in one-quarter of patients due to PNS, lead instability, and unfavourable coronary venous anatomy. The use of additional technologies

Figure 6  Coronary venous EAM of Patient No. 24, illustrating the additional value of LV lead placement guided by coronary venous mapping. Pre-implantation coronary venograms in LAO (A) and RAO (B) view revealed a large inferolateral and a small anterolateral vein. The conventional anatomical LV lead placement approach would target the inferolateral vein. However, coronary venous mapping during intrinsic ventricular activation (C) revealed a maximal electrical delay of only 90 ms in the basal inferolateral vein, which further decreased in the more distal segments, whereas a maximal electrical delay of 140 ms could be measured in the anterolateral vein. Based on the outcome of coronary venous mapping, the LV lead was positioned in the anterolateral vein, thereby achieving a clear benefit in LV lead electrical delay. (D) X-ray of final LV lead position in LAO view. (E) X-ray of final LV lead position in RAO view. AIV, anterior inter-ventricular vein; ALV, anterolateral vein; IIV, inferior inter-ventricular vein; ILV, inferolateral vein.
Avoiding phrenic nerve stimulation

Phrenic nerve stimulation accounts for failure to deliver proper CRT or urgent need for LV lead relocation in a substantial number of patients. Studies addressing the issue have reported PNS at implantation in 7.6–37% of patients, resulting in failure to pace the target site in 10–14%.40–42 PNS at follow-up has been reported in 11–33% of patients, requiring LV lead replacement in a substantial proportion of patients and eventual turn-off of CRT in some.43–45 In the present study, PNS was assessed by systematic pacing of the coronary veins with the tip of the mapping guidewire. The usefulness of this approach for detecting PNS has previously been demonstrated by de Cock et al.14 In addition, the EAM system used in our study allowed comprehensive annotation of PNS-free areas on the created coronary venous geometry, consequently providing 3D visualization of the anatomical location of these areas with respect to the target LV pacing site and the position of the LV lead tip. Subsequently, real-time navigation of the LV lead in the geometry allowed targeting of a PNS-free zone with optimal electrical delay. This approach proved to be effective to avoid PNS at medium-term follow-up (4 ± 1 months) in all study patients.

Limitations

The sample size of this study was relatively small. However, the study was performed on consecutive patients and the study population resembled the typical population of patients with LBBB and heart failure referred for CRT implantation. Only standard bipolar LV leads were used. The use of new lead technologies, such as multipolar leads and active-fixation leads, may increase the success rate of targeted LV lead placement at the latest activated LV region by improving lead stability and avoiding PNS. Unfortunately, most device vendors did not have multipolar LV leads available at the time the current study was conducted. Cardiac magnetic resonance imaging was not performed in this study. Therefore, we could not assess the distribution and extent of scar and its potential effect on electrical activation. Also, the mapping method used in the current study did not allow for reliable differentiation between scar and healthy myocardium. Thus, the position of the LV lead with respect to regions of myocardial scar was not taken into account during LV lead placement. The current study demonstrates a feasible method to guide LV lead placement to the region of latest electrical activation. However, the clinical value of this lead placement strategy was not proven by acute haemodynamic measurements or long-term echocardiographic or clinical follow-up. Subsequent larger and long-term follow-up studies are therefore required to evaluate the impact of coronary venous mapping-guided LV lead placement on CRT outcome.

Conclusion

Coronary venous EAM using EnSite NavX in combination with a mapping guidewire can be used at the time of CRT implantation to guide LV lead placement to the latest activated region free of PNS. This approach especially contributes to optimization of LV lead electrical delay in patients with multiple target veins. The conventional anatomical LV lead placement strategy does not target the vein...
with maximal electrical delay in many of these patients. The clinical value of coronary venous mapping-guided LV lead placement needs prospective evaluation.

Acknowledgements

We gratefully thank Peter Bakker and Arjan Bennink from St Jude Medical for their technical support during the mapping procedures.

Conflict of interest: Y.B. is a consultant for Medtronic. L.P. is a consultant for Attricure. F.W.P. received research grants from Medtronic, Boston Scientific, EBR Systems, Biological Delivery System Cordis, MSD, and Protesus Medical. H.J.C. received consulting fees from Boehringer Ingelheim, Sanofi-Aventis, and AstraZeneca, grant support from St Jude Medical, Boston Scientific, Boehringer Ingelheim, Sanofi-Aventis, Medapharma, and Merck, and honoraria from Medtronic, Sanofi-Aventis, Medapharma, Merck, Boehringer Ingelheim, and Biosense Webster. K.V. received research grants from Medtronic and is a consultant for Medtronic. All other authors declare no conflict of interest.

References


**Images in Electrophysiology**

**Bilateral confined pulmonary vein fibrillation**

Shinsuke Miyazaki*, Horoshi Taniguchi, and Yoshito Iesaka

Cardiology Division, Cardiovascular Center, Tsuchiura Kyodo Hospital, 11-7 Manabeshin-machi, Tsuchiura, Ibaraki 300-0053, Japan

* Corresponding author. Tel: +81 29 823 3111; fax: +81 29 826 2411. E-mail address: mshinsuke@k3.dion.ne.jp

Confined pulmonary vein (PV) fibrillation is a rare finding, which suggests that fibrillation can sustain in the isolated tissue independently. A 76-year-old woman underwent second catheter ablation procedure for recurrent atrial fibrillation (AF). The starting rhythm was AF. During ipsilateral right PV re-isolation, sinus rhythm was restored despite sustained fibrillation inside (panel A). Following the re-isolation of right PVs, confined PV fibrillation was also observed in isolated left superior PV (panel B). To the best of our knowledge, this is the first case presenting simultaneous confined bilateral PV fibrillation. The case highlighted the strong association with the mechanism of sustaining AF and bilateral PVS.

**Conflict of interest:** none declared.

Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2014. For permissions please email: journals.permissions@oup.com.