Delivered Trans-Septal Activation Results in Comparable Hemodynamic Effect of Left Ventricular and Biventricular Endocardial pacing  
Insights From Electroanatomical Mapping

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Background—We sought to compare left ventricular (LVendo) and biventricular epicardial pacing (BIVepi) with LV (LVepi) and BIV endocardial pacing (BIVendo) in patients with chronic heart failure with an emphasis on the underlying electrophysiological mechanisms and hemodynamic effects.  

Methods and Results—Ten patients with chronically implanted cardiac resynchronization devices underwent temporary LVendo and BIVendo pacing with an LV endocardial roving catheter. A pressure wire and noncontact mapping array were placed to the LV cavity to measure LVP/dtmax and perform electroanatomical mapping. At the optimal endocardial position, the acute hemodynamic response (AHR) was superior to epicardial stimulation, the AHR to BIVendo pacing and LVendo pacing being comparable (21±15% versus 22±17%; P=NS). During intrinsic conduction, QRS duration was 185±30 ms, endocardial LV total activation time 92±27 ms, and trans-septal activation time 60±21 ms. With LVendo pacing, QRS duration (187±29 ms; P=NS) and endocardial LV total activation time (91±23 ms; P=NS) were comparable with intrinsic conduction. There was no significant difference in endocardial LV total activation time between LVendo and BIVendo pacing (91±23 versus 85±15 ms; P=NS). Assessment of isochronal maps identified slow trans-septal conduction with both LVendo and BIVendo pacing resulting in activation of almost the entire LV endocardium prior to septal breakout, thereby limiting any possible fusion with either pacing mode.  

Conclusions—The equivalent AHR to LVendo and BIVendo pacing may be explained by prolonged trans-septal conduction limiting fusion of electrical wavefronts. The optimal AHR was associated with predominantly LV pre-excitation and depolarization. Our results suggest that LV pacing alone may offer a viable endocardial stimulation strategy to achieve cardiac resynchronization. (Circ Arrhythm Electrophysiol. 2014;7:251-258.)

Key Words: bundle-branch block ■ cardiac resynchronization therapy ■ heart failure

The last 2 decades have seen cardiac resynchronization therapy (CRT) establish itself as a powerful tool in selected heart failure patients with evidence of electric dyssynchrony. However, when applying internationally recognized selection criteria for CRT, there is a nonresponse rate of 30% to 40%.1,2 CRT is conventionally delivered as biventricular pacing with endocardial stimulation of the right ventricle and epicardial left ventricular (LV) stimulation via the coronary sinus (BIVepi). Clinical studies have shown similar outcomes with LV and BIV pacing suggesting that epicardial LV-only pacing (LVepi) may be as beneficial as BIVepi.3 A recent canine study suggested delayed trans-septal activation results in the majority of LV depolarization occurring prior to any contribution from right ventricular (RV) stimulation, thereby limiting fusion of electric wavefronts.4 LV pacing alone has potential advantages over BIV pacing because it may preserve intrinsic conduction, avoid detrimental effects of RV pacing, and reduce the number of electrodes making implantation less technically challenging. Endocardial LV pacing (LVendo) is not limited by coronary sinus anatomy and has been shown to improve acute and medium-term CRT response5-8 due to a more physiological electric and mechanical propagation.9,10 The relative effect of endocardial pacing (LVendo) alone compared with BIVendo pacing is not well defined. We therefore sought to compare the hemodynamic effects of LVepi and BIVepi with LVendo and BIVendo pacing in patients with chronic heart failure with an emphasis on the underlying electrophysiological mechanisms that might explain the response to these different pacing modalities.

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Methods

The study was approved by the local ethics committee, and informed consent was obtained from each patient. The study population consisted of 10 patients with a chronically implanted CRT system in situ for at least 3 months (St. Jude Medical, Sylmar, CA). All patients fulfilled standard criteria for CRT (New York Heart Association [NYHA] class II-IV drug refractory heart failure, LV ejection fraction $\leq 35\%$, and QRS $\geq 120$ ms [9 patients had left bundle-branch block (LBBB), 1 patient was dependent on RV pacing]). Patients with a mechanical aortic valve or significant peripheral vascular disease were excluded. Baseline assessment included NYHA functional class, ECG, and 2-dimensional echocardiography prior to the original CRT implant. Each patient’s heart failure pathogenesis was confirmed on the basis of clinical history, coronary angiography, and/or cardiac MRI. In all cases, a temporary LV endocardial lead was used to perform LV endocardial stimulation.

Invasive Hemodynamic and Electroanatomic Study

The protocol used has previously been described. Patients were lightly sedated using diazepam (5–10 mg). A steerable 6Fr Livewire decapolar catheter (St Jude Medical, Sylmar, CA) was passed from the left femoral artery retrogradely to the LV cavity to stimulate multiple sites within the LV. A noncontact mapping array was placed retrogradely into the LV cavity and an 0.014 inch diameter high-fidelity Certus PressureWire and PhysioMon software (RADI Medical Systems, Uppsala, Sweden) with a 50 Hz frequency response and 50 Hz filter bandwidth were used to assess real-time mean peak LVdP/dtmax.$^7$

Acute Hemodynamic Measurement

LVdP/dtmax was recorded for at least 20 s to ensure steady-state conditions during any pacing modality. LVdP/dtmax during atrial pacing (AAI) or RV pacing (if the patient was in atrial fibrillation) at 5 to 10 beats above intrinsic rate was used as baseline. A waiting period of at least 20 s was respected after any change in pacing settings or lead position to achieve hemodynamic stabilization. These methods have previously been shown to reliably measure LVdP/dtmax. Results at each pacing site were expressed as a percentage change from baseline. To minimize baseline drift in acute hemodynamic response (AHR) the baseline was reassessed prior to and after every change in pacing modality and comparisons were made to a mean of these 2 readings. Data from premature ventricular complexes were discarded.

Pacing Protocol

A pacing protocol was performed (5 to 10 bpm above intrinsic rate, paced and sensed atrio-ventricular delay 100 ms, AAI pacing as baseline, and repeated after each pacing mode):

1. DDD-RV only.
2. DDD-LV only from the chronic epicardial coronary sinus lead (LVepi).
3. DDD-BIV from the chronically implanted epicardial LV lead plus RV pacing with 0 ms V-V delay (BIVepi).
4. LV-only pacing from the endocardial catheter at multiple endocardial sites (LVendo).
5. LV endocardial and RV endocardial pacing from multiple LV endocardial sites with 0 ms V-V delay (BIVendo).

BIVendo and LVendo stimulation was repeated with the LV roving catheter in a random order in up to 4 different LV endocardial positions including anterior, lateral, and posterior sites. Capture was verified for each pacing modality by looking for a change in QRS morphology at a paper speed of 200 mm/s. This was also validated with reference to LV pacing by analysis of the activation wave front on noncontact mapping. For the purposes of this study, the endocardial position producing the best AHR with BIVendo pacing was chosen as the position used for analysis.

Electroanatomic Mapping

Analysis of the noncontact mapping data was performed by 2 independent observers blinded to the hemodynamic results. Endocardial maps were obtained at baseline and in each pacing configuration. The virtual unipolar electrograms recorded at 1200 Hz (temporal resolution of 0.83 ms) from the endocardial surface were used to measure the endocardial LV total activation time (LVTAT). The high-pass filter was set at 8 Hz. The onset of LV activation was defined as the first peak negative dV/dt at any point in the LV, and the end of LV activation was defined as the time of the latest peak negative unipolar electrogram on the virtual endocardial surface. The trans-septal activation time (TSAT) was defined as the time from the onset of the earliest QRS complex to the time of the first detected LV endocardial breakthrough as described previously by Auricchio et al.$^{19}$

Statistical Analysis

Statistical analysis was performed on PASW Statistics 20 (SPSS Inc, Chicago, IL). Data were analyzed using generalized estimating equations using an exchangeable correlation structure to explore the extent of differences between pacing methods. Pacing methods were compared with each other, and to avoid type 1 errors, $P$ values were corrected using the Bonferroni adjustment. Descriptive results are expressed as mean±SD.

Results

Patient Demographics

Baseline characteristics are shown in Table 1. All patients were men with a mean age of 62±8 years. The mean LV ejection fraction was 26±8%, and there were equal numbers of ischemic and nonischemics (5/5). The mean QRS duration (QRSd) was 186±27 ms, and morphology was LBBB in 9 and RV-paced patients.

Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>62±8</td>
</tr>
<tr>
<td>Sex n (%)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>10 (100)</td>
</tr>
<tr>
<td>Female</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Etiology n (%)</td>
<td></td>
</tr>
<tr>
<td>Ischemic</td>
<td>5 (50)</td>
</tr>
<tr>
<td>Nonischemic</td>
<td>5 (50)</td>
</tr>
<tr>
<td>NYHA class n (%)</td>
<td></td>
</tr>
<tr>
<td>Class II</td>
<td>4 (40)</td>
</tr>
<tr>
<td>Class III</td>
<td>6 (60)</td>
</tr>
<tr>
<td>LV ejection fraction* (%)</td>
<td>26±8</td>
</tr>
<tr>
<td>Intrinsic PR interval (ms)</td>
<td>182±19</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>185±30</td>
</tr>
<tr>
<td>QRS morphology n (%)</td>
<td>9 (90)</td>
</tr>
<tr>
<td>LBBB</td>
<td></td>
</tr>
<tr>
<td>RV paced</td>
<td>1 (10)</td>
</tr>
<tr>
<td>Epicardial LV lead position n (%)</td>
<td></td>
</tr>
<tr>
<td>Lateral</td>
<td>4 (40)</td>
</tr>
<tr>
<td>Posterolateral</td>
<td>4 (40)</td>
</tr>
<tr>
<td>Anterior</td>
<td>2 (20)</td>
</tr>
</tbody>
</table>

Continuous variables are expressed as mean±SD. LBBB indicates left bundle-branch block; LV, left ventricular; and NYHA, New York Heart Association. *Derived from 2-dimensional echocardiography using the modified Simpson’s biplane method.
rhythm in the remaining patient. The epicardial LV lead was in a lateral or posterolateral position in 8 (80%) patients and in an anterior position in 2 (20%) patients. Table 2 displays the epicardial LV lead positions for each patient alongside the optimal LV endocardial pacing position in each case. There was concordance in 4 cases (Tables 1 and 2).

**Acute Hemodynamic Response**

LV dP/dt_max while AAi pacing 5 to 10 beats above intrinsic rate was taken as the baseline to which all pacing measurements were compared in each patient. Table 3 shows the mean (SD) percentage increase in LV dP/dt_max for each pacing mode over baseline. Compared with baseline, LV_epi pacing from the chronically implanted epicardial lead gave a 10±14% increase in LV dP/dt_max, BIV_epi pacing increased LV dP/dt_max over baseline by 15±15% while LV_endo and BIV_endo pacing increased LV dP/dt_max over baseline by 22±17% and 21±15%, respectively. The P value for the overall test for variability across all pacing modalities was significant at 0.001 (Figure 1). BIV_epi increased LV dP/dt_max by a mean of 4.1% (95% confidence interval [CI], 1.2–7.0). However, the AHR to BIV_endo pacing and LV_endo pacing were comparable with a mean difference of 1.1% (95% CI, −5.5 to 7.8). At the optimal endocardial position, hemodynamics were superior to epicardial stimulation with a mean difference of 6.2% (95% CI, 0.5–11.8 for BIV_endo versus BIV_epi pacing; Figure 1; Table 3).

**QRSd, LVTAT, and Trans-septal Activation Times**

Intrinsic QRSd was 185±30 ms, and the intrinsic endocardial LVTAT was 92±27 ms. Four patients exhibited a type I pattern of endocardial activation (homogenous spread from the septum to lateral wall) while 6 patients exhibited a type II pattern of activation (characterized by a line of functional block between the septum and lateral wall). TSAT in intrinsic rhythm was 60±21 ms and lengthened with RV pacing to 71±13 ms (mean difference, 10.4; 95% CI, 0.6–20.2). Figure 2 demonstrates the delay in endocardial breakthrough on the left side of the interventricular septum following RV pacing. LV_epi pacing resulted in a similar QRSd to intrinsic (187±25 versus 185±30 ms) and a similar endocardial LVTAT (93±17). BIV_epi, however, significantly reduced QRSd to 142±12 ms (mean difference, −42.3; 95% CI, −61.0 to −23.6) and shortened endocardial LVTAT to 72±15 ms (mean difference, −20.1; 95% CI, −41.4 to 1.2), suggesting a degree of fusion of RV and LV wave fronts. The mean QRSd and endocardial LVTAT with LV_endo pacing were comparable with intrinsic rhythm (187±29 and 91±23 ms, respectively). BIV_endo pacing from the same position as LV_endo reduced QRSd over intrinsic rhythm to 155±28 ms; however, there was no significant difference between the endocardial LVTAT between LV_endo and BIV_endo pacing (91±23 versus 85±15 ms; mean difference, −5.9; 95% CI, −14.6 to 2.8; Figure 1; Table 3).

Assessment of the isochronal activation maps of the LV during the various pacing modes demonstrated fusion of electric wave fronts with BIV_epi pacing (Figure 3). LV_epi pacing resulted in minimal fusion of RV and LV activation wave fronts. No significant fusion was seen with BIV_endo pacing, and the activation maps were strikingly similar to LV_endo pacing (Figure 3). The noncontact maps identified slow trans-septal conduction during both LV_endo and BIV_endo pacing. This resulted in activation of almost the entire LV endocardium prior to septal breakthrough, thereby limiting any possible fusion with either pacing mode. With BIV_epi pacing, endocardial breakthrough from the epicardial LV lead was sufficiently delayed to allow fusion of electric wave fronts.

**Discussion**

The present study provides new insights into the electric mechanisms of epicardial and endocardial CRT. The principal findings of our study are as follows: (1) trans-septal LV activation was prolonged during both intrinsic rhythm and RV pacing (equivalent to ≈33% of the QRSd); (2) there was no significant difference between the AHR derived from LV_endo and BIV_endo at the optimal site, whereas BIV_epi appeared superior to LV_epi pacing; (3) endocardial LVTAT and LV endocardial activation patterns were comparable between LV_endo and BIV_endo pacing, suggesting minimal fusion of LV and RV wave fronts; and (4) LV_endo pacing may be equivalent to BIV_endo pacing.

**Endocardial Pacing Hemodynamics and Electric Changes**

BIV_endo stimulation at the optimal site gave a superior AHR to epicardial pacing, which was site specific. LV_endo stimulation at a short AV delay produced a similar AHR to BIV_endo pacing (22±17% versus 21±15%; P=NS), suggesting no incremental benefit in BIV_endo over LV_endo pacing (when pacing at a short AV delay) in comparison with epicardial stimulation. Our findings may be explained on the basis of the long TSATs seen in our patients with LBBB, so that by the time the RV pacing impulse had broken through the septum into the LV cavity (≈70 ms), the majority of LV endocardial activation had already occurred. This is supported by examination of the isochronal activation maps of BIV_endo and LV_endo pacing, which were almost identical, suggesting an absence of fusion (Figure 3). The short AV interval of 100 ms used in our study may explain the apparent lack of fusion seen with LV_endo pacing, and the PR interval in our group was 182±19 ms; therefore, at an AV delay of 100 ms, one would not expect...
any fusion from intrinsic activation via the right bundle branch. A reduction in QRSd was noted with BIV endo pacing, but endocardial LVTAT remained similar between LV and BIV endo pacing. The surface QRSd represents a summation of LV and RV activation times, and therefore, the QRS reduction seen with BIV pacing may reflect a reduction in RV (and not LV) activation time. We measured only LV endocardial activation time (and not epicardial) and the reduction may be related to changes occurring in the epicardium as has been shown in the canine model. We saw divergent findings for LV epi and BIV epi pacing with an improvement in AHR with BIV epi pacing over LV epi accompanied by a reduction in both QRSd and endocardial LVTAT. The electric findings may be accounted for by the fact that pacing from the epicardium

![Overall test of variability across all pacing modes: P<0.001](http://circep.ahajournals.org/)

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**Table 3.** Hemodynamic and Electric Parameters Compared Across Pacing Modalities

<table>
<thead>
<tr>
<th>Modality</th>
<th>% Change in dP/dt max Over Baseline</th>
<th>Mean Change in QRSd Over Baseline</th>
<th>Mean Change in TSAT Over Baseline</th>
<th>Mean Change in LVAT Over Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>n/a 185±30</td>
<td>60±21</td>
<td>92±27</td>
<td></td>
</tr>
<tr>
<td>RV</td>
<td>1±3</td>
<td>195±30</td>
<td>60±21</td>
<td>92±27</td>
</tr>
<tr>
<td>LV ep</td>
<td>10±3</td>
<td>187±25</td>
<td>2.3 ±11.9 to 16.5; P=0.723</td>
<td>93±26</td>
</tr>
<tr>
<td>BIV ep</td>
<td>15±1</td>
<td>142±12</td>
<td>−42.3 ±61.0 to −23.8; P=0.001</td>
<td>72±15</td>
</tr>
<tr>
<td>LV endo</td>
<td>22±17</td>
<td>187±29</td>
<td>2.8 ±10.2 to 15.8; P=0.639</td>
<td>91±23</td>
</tr>
<tr>
<td>BIV endo</td>
<td>21±15</td>
<td>155±28</td>
<td>−30.1 ±47.0 to −13.2; P=0.003</td>
<td>85±15</td>
</tr>
</tbody>
</table>

LV ep indicates AV synchronous left ventricular pacing catheter for LV stimulation; BIV ep, AV synchronous biventricular pacing using the chronically implanted CRT system; LVAT, endocardial LV activation time; LV epi, AV synchronous LV pacing from the temporary endocardial LV pacing catheter; LV endo, AV synchronous LV pacing from the epicardial left ventricular lead; QRSd, QRS duration; and TSAT, trans-septal activation time.

**Figure 1.** Mean change from baseline in dP/dt max, QRS duration, and left ventricular (LV) activation time for each pacing modality. This is displayed as the percentage mean change each variable resulting from pacing in each pacing configuration, compared with baseline (AAI pacing). BIV ep indicates AV synchronous biventricular pacing using the chronically implanted cardiac resynchronization therapy system; BIV endo, AV synchronous biventricular stimulation using the temporary endocardial LV pacing catheter for LV stimulation; LV ep, AV synchronous LV pacing from the temporary endocardial LV pacing catheter; and LV endo, AV synchronous left ventricular pacing from the epicardial LV lead.
resulted in a sufficient delay to compensate for prolonged trans-septal conduction, thereby allowing fusion of electric wave fronts from RV and LV stimulation. A reduction in RV activation time (which was not measured) may have also contributed to the reduction in QRSd with BIVepi pacing (the reduction in QRSd between LVepi and BIVepi was greater than the reduction between LVendo and BIVendo). Our results would suggest a mismatch between hemodynamic response and electric parameters, with the best hemodynamic response being relatively equal between LVendo and BIVendo pacing; however, with epicardial pacing, this differed. This may suggest that optimal pacing LV endocardially may only require a single lead. The long TSAT seen in our group would suggest that most LV depolarization occurs via the LV pacing impulse in both LVendo and BIVendo modes.

**Comparison With Previous Studies**

Our findings of improved hemodynamic response with endocardial CRT are in keeping with other groups. Strik et al.\(^2\) demonstrated in a chronic canine heart failure model improved hemodynamics as a result of LV endocardial pacing compared with conventional epicardial CRT, which could be explained by the shorter path length and more rapid conduction resulting from endocardial LV pacing. Derval et al.\(^6\) found better hemodynamic effects with LVendo versus LVepi, and in keeping with our study showed that there is not 1 pacing site (or combination of pacing sites) that is best for all patients. The benefits of LV endocardial pacing seen in our patients are likely to be due to a reduction in total LV activation time (endocardial and epicardial activation) with the reduction predominantly in epicardial LV activation time, which we did not specifically measure in this study. Our findings of slow trans-septal conduction are strikingly similar to those recently found in canines.\(^4\) Strik et al.\(^4\) found a TSAT of \(67\pm9\) ms in a canine model of LBBB and pacing induced dyssynchrony compared with \(60\pm21\) ms in our patients with LBBB and heart failure. In the same study, there was no incremental benefit in BIV compared with LV-only pacing, and this was due to predominant LV stimulation in both modes due to long TSATs. This is in keeping with our findings with endocardial stimulation where we saw no incremental benefit with RV and LV pacing due to apparent lack of fusion with both modalities. Our results do however differ in that we found an incremental hemodynamic benefit with BIVepi compared with LVepi associated with a reduction in the LVTAT and a degree of fusion of wave fronts, which may be explained by much longer endocardial LVTAT than in the dogs. In addition, we saw that there was a delay between epicardial stimulation and endocardial breakthrough (as seen in Figure 3), thereby allowing for a delay in trans-septal conduction and resulting in fusion of RV and LV wave fronts. We saw minimal fusion with LVepi pacing, and this may have been as a result of the short AV delay used in the study (100 ms) versus the longer mean PR interval (182 ms) in intrinsic rhythm. The short AV delay when the RV was paced with BIVepi pacing may have provided a sufficient head start and therefore earlier breakthrough into the LV endocardium. The difference may also be explained by the differences between the animal model in our patients with chronic heart failure, significantly dilated ventricles, and the presence of scar (50% of our patients were ischemic). Also, the PR interval in our patients of over 180 ms is longer in comparison with the canine model and likely explains the difference on the basis of fusion with BIVepi pacing.

Human studies comparing LVepi pacing with BIVepi pacing would also seem to suggest that there is little difference in terms of acute hemodynamics or medium-term clinical response between the two.\(^2\) We found a small (but
statistically significant) benefit in AHR with BIVepi pacing over LVepi pacing. The reason behind this may lie behind the fact that we used a short AV delay of 100 ms compared with a long mean intrinsic PR interval of 182 ms. This meant that we saw minimal fusion with LVepi pacing (Figure 3), and this, in turn, may have attenuated the AHR. Auricchio et al also studied the differences in LVdP/dt max between LVepi and BIVepi pacing and found LVepi to be associated with a slightly greater AHR. Crucially, pacing was performed in VDD mode (atrial sensing) and so did not control for heart rate, which is one of the key determinants that affects absolute values of LVdP/dt max. In addition, most LV leads were positioned apically, and this has been shown to attenuate the response to BIV stimulation. These differences may also explain why our results vary in this respect.

Study Limitations
This study is limited by its small sample size; however, given the highly invasive and time-consuming nature of the study, the size was limited to the smallest size needed to provide significant results in this proof of principle study. Ideally, a range of atrio-ventricular delays and ventriculo-ventricular delays would have been assessed, but this was not possible because of time constraints and will need to be assessed in future studies. Our study used a short AV delay of 100 ms, and this may, in some part, explain the results. With such a short AV delay there was likely to be little intrinsic fusion in the LV-only modes (intrinsic PR interval was >180 ms). At a short AV delay, LV and BIV endocardial pacing appear equivalent, although we cannot rule out the possibility that at different AV delays, this may have differed due to LV fusion pacing. Our measures of LV activation were endocardial, and we did not study epicardial activation specifically. We do, however, have data on QRSd, which is a measure of total electric activation of the heart. We did not specifically look at areas of myocardial scar in relation to lead positioning in our patients, which may be important in determining both the optimal epicardial and endocardial stimulation sites. There was concordance between epicardial and endocardial lead position in only 4 cases, meaning that comparisons between endocardial and epicardial CRT should be made with caution, and the benefits of LV endocardial pacing seen in this study may be related to the lack of being constrained by coronary venous anatomy and so being able to pace from a more...
optimal position. Our assessment of CRT response using AHR may not necessarily translate into chronic response; however, previous work from our institution has suggested a good correlation with chronic response albeit predominantly in nonischemic patients.17

Clinical Implications
Our findings may have important implications for the practice of endocardial CRT. This type of CRT may become more prevalent in an attempt to improve CRT response. Our results would suggest that in such patients, LVendo pacing may be a viable treatment strategy and that in patients not requiring an RV lead (for potential defibrillation), it may be reasonable to implant an LV lead alone.

Conclusions
Endocardial LV pacing from the optimal site is hemodynamically superior to conventional epicardial CRT, but BIV endocardial pacing did not produce an incremental hemodynamic benefit over endocardial LV pacing alone. This may be due to long TSATs, resulting in lack of fusion of LV and RV wave fronts. Indeed, the optimal hemodynamic response would seem to be associated with predominantly LV pre-excitation and depolarization. Our results suggesting that LV pacing alone may offer a viable endocardial stimulation strategy to achieve CRT.

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References
Endocardial left ventricular pacing allows for improved hemodynamics in patients with heart failure. Improved cardiac remodeling in patients undergoing cardiac resynchronization therapy is due to atrioventricular delay and long-term biventricular pacing. The increased lateral ventricular activation during cardiac resynchronization therapy is due to atrioventricular delay and long-term biventricular pacing. The increased lateral ventricular activation during cardiac resynchronization therapy is due to atrioventricular delay and long-term biventricular pacing.
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