Echocardiographic Parameters of Ventricular Dyssynchrony Validation in Patients With Heart Failure Using Sequential Biventricular Pacing

Pierre Bordachar, MD, Stephane Lafitte, MD, PHD, Sylvain Reuter, MD, Prashanthan Sanders, MBBS, PHD, Pierre Jaïs, MD, Michel Haïssaguerre, MD, Raymond Roudaut, MD, Stephane Garrigue, MD, PHD, Jacques Clementy, MD

Pessac, France

OBJECTIVES
We sought to evaluate the relationship between hemodynamic and ventricular dyssynchrony parameters in patients undergoing simultaneous and sequential biventricular pacing (BVP).

BACKGROUND
Various echocardiographic parameters of ventricular dyssynchrony have been proposed to screen and optimize BVP therapy.

METHODS
Forty-one patients with heart failure undergoing BVP implantation were studied. Echocardiography coupled with tissue tracking and pulsed Doppler tissue imaging (DTI) was performed before and after BVP implantation and after three months of optimized BVP. Indexes of inter- or intraventricular dyssynchrony were correlated with hemodynamic changes during simultaneous and sequential BVP (10 intervals of right ventricular [RV] or left ventricular [LV] pre-excitation).

RESULTS
Variations in intra-LV delay peak, intra-LV delay onset, and index of LV dyssynchrony measured by pulsed DTI were highly correlated with those of cardiac output ($r = 0.67$, $r = 0.64$, and $r = 0.67$, respectively; $p < 0.001$) and mitral regurgitation ($r = 0.68$, $r = 0.63$, and $r = 0.68$, respectively; $p < 0.001$), whereas variations in the extent of myocardium displaying delayed longitudinal contraction ($r = 0.48$ and $r = 0.51$, respectively; $p < 0.05$) and the variations in septal-to-posterior wall motion delay ($r = 0.41$, $p < 0.05$ and $r = 0.24$, $p = NS$, respectively) were less correlated. The changes in interventricular dyssynchrony were not significantly correlated ($p = NS$). Compared with simultaneous BVP, individually optimized sequential BVP significantly increased cardiac output ($p < 0.01$), decreased mitral regurgitation ($p < 0.05$), and improved all parameters of intra-LV dyssynchrony ($p < 0.01$). At three months, a significant reverse mechanical LV remodeling was observed with significantly decreased LV volumes ($p < 0.01$) associated with an increased LV ejection fraction ($p = 0.035$).

CONCLUSIONS
Specific echocardiographic measurements of ventricular dyssynchrony are highly correlated with hemodynamic changes and may be a useful adjunct in the selection and optimization of BVP. Individually optimized sequential BVP provided a significant early hemodynamic improvement compared with simultaneous BVP.

© 2004 by the American College of Cardiology Foundation

Cardiac resynchronization therapy (CRT) using simultaneous biventricular pacing (BVP) has an established role in the management of symptomatic drug-refractory heart failure in patients with delayed left ventricular (LV) activation (1–4). Although restoring synchronized contraction in these patients has been demonstrated to improve systolic and diastolic ventricular function (5–9), the pathophysiologic basis of these observations remains poorly defined.

Whether the hemodynamic benefits of CRT are due to improvements in inter- or intraventricular synchronization is unclear. Several echocardiographic parameters have been proposed to screen and optimize CRT (10–14). Some authors have suggested the importance of “contractile reserve recruitment,” advocating the reduction of the number of ventricular segments contracting after the aortic valve closure (15–17), whereas others have suggested the importance of improving “ventricular dysynchrony” or intraventricular delays as the mechanism for improvement using CRT (8,18). An array of parameters identifying ventricular dyssynchrony has evolved based on the onset, peak, or end-systolic motion of the different segments of the ventricular wall to optimize CRT. However, the correlation between these parameters and hemodynamic variables has not been demonstrated yet.

In this prospective clinical study, we utilized echocardiography to evaluate the relationship between the various parameters of ventricular dyssynchrony and hemodynamic status in patients undergoing CRT. In addition,
study, which was approved by the institutional clinical research and ethics committee.

**Pacemaker implantation.** All leads were positioned transvenously as previously described. The atrial lead was positioned at the right atrial appendage and the RV lead at the apex. The LV lead (Attain OTW 4193, Medtronic, Minneapolis, Minnesota) was positioned through a coronary sinus in the posterolateral (n = 23) or lateral (n = 18) cardiac vein. The pacing leads were connected to a rate-adaptive pacemaker (InSync III, Medtronic). This pacemaker has two separate ventricular channels for RV and LV pacing with a programmable interventricular stimulation delay of 0 to 80 ms.

**Study protocol.** After implantation of BVP, the study protocol was performed. For the study protocol, different predetermined pacing configurations were assessed using spontaneous atrial synchronized pacing. To ensure complete ventricular capture during the different pacing configurations, the QRS morphology and width were verified to be similar between the non-atrial synchronized (VVI mode) and atrial synchronized (VDD mode) ventricular pacing configurations. The AV delay was modified and optimized for each tested configuration, as previously reported, in order to provide the longest transmitral filling time without truncation of the A-wave from pulsed Doppler analysis of the LV filling (19).

Cardiac output, severity of MR, and parameters of interand intraventricular dyssynchrony were evaluated using echocardiography at the following predetermined configurations of ventricular pacing: 1) RV pacing; 2) LV pacing; 3) simultaneous BVP; 4) sequential BVP with RV pre-activation with interventricular intervals of 12, 20, 40, and 80 ms; and 5) sequential BVP with LV pre-activation with interventricular intervals of 12, 20, 40, and 80 ms.

These configurations were performed in random order with baseline data being determined during simultaneous BVP at the start and completion of the protocol to control for the effects of the study duration. In addition, data were collected only after 10 min of pacing in each configuration to ensure data acquisition after achieving equilibrium.

After the study protocol, patients were maintained in the individually optimized sequential BVP configuration. The optimal pacing configuration for a given patient was identified as that achieving the maximal increase in cardiac output during the study protocol.

**Echocardiography analysis.** Echocardiography was performed before and on the day after pacemaker implantation, using a 2.5- to 5.0-MHz imaging probe connected to a Vingmed-General Electric ultrasound system (System 5; Horten, Norway) and performed in accordance with the American Society of Echocardiography guidelines. To minimize variability between examinations, all echocardiographic recordings were performed by one echocardiographer. All images were recorded digitally and analyzed off-line. Each parameter was measured and averaged over three consecutive beats during sinus rhythm. The off-line
analysis was performed by a different observer and was blinded to the pacing mode.

The following hemodynamic variables were evaluated at each predetermined interval: 1) LV filling time; 2) cardiac output; and 3) severity of MR. The LV filling time was determined by pulsed wave Doppler transmitral flow as the time between the onset of the E wave and the end of the A-wave. Cardiac output was determined by the LV outflow method (20). Such evaluation has been previously standardized and demonstrated to be reproducibly applied in individuals with heart failure (21). The severity of MR was assessed by the proximal isovelocity surface area (PISA), as previously described (22–24). Color flow imaging of MR was optimized with a small color angle from the apical window. The color flow at zero baseline was shifted downward to increase hemispheric PISA. The negative aliasing velocity was adjusted from 20 to 40 cm/s to obtain satisfactory hemispheric PISA. The regurgitant volume and effective regurgitant orifice area (EROA) were calculated using standard formulae (EROA = regurgitant flow/maximal mitral regurgitant velocity).

At each predetermined interval, features of inter- and intraventricular (intra-LV) dyssynchrony were evaluated as previously described. Interventricular dyssynchrony was defined as the difference between the aortic and pulmonary pre-ejection delays and determined as the time from the onset of the QRS complex to the beginning of each respective systolic ejection by pulsed wave Doppler imaging (11).

Intra-LV dyssynchrony was determined using tissue Doppler imaging (TDI) to assess segmental wall motion, as previously described (8,18,25). In brief, TDI was applied by placing the sample volume in the middle of the basal and mid-segmental portions of the septal, lateral, inferior, anterior, posterior, and antero-septal walls. Gain and filter settings were adjusted as needed to eliminate background noises and to allow for a clear tissue signal. The TDI velocities were recorded and measured at a sweep speed of 100 mm/s using on-line callipers. The following were measured:

1. Variation in the onset of segmental LV contraction (intra-LV delay \( \text{onset} \)) was evaluated by determining the electromechanical delay \( \text{onset} \) for each segment by measuring the interval between the onset of the QRS complex and the onset of each segmental contraction. The intra-LV delay \( \text{onset} \) was then calculated as the difference between the shortest and longest of the 12 segmental electromechanical delay \( \text{onset} \) values (26–28).

2. Variation in the peak of segmental LV contraction (intra-LV delay \( \text{peak} \)) was evaluated by determining the electromechanical delay \( \text{peak} \) for each segment by measuring the interval between the onset of the QRS complex and the peak of each segmental contraction. The intra-LV delay \( \text{peak} \) was then calculated as the difference between the shortest and longest of the 12 segmental electromechanical delay \( \text{peak} \) values (8,12,18).

3. The index of systolic dyssynchrony was defined as the standard deviation of the 12 segmental electromechanical delay \( \text{peak} \) values already cited (8,18).

4. Delayed longitudinal contraction (DLC) was calculated using TDI coupled with strain rate analysis. A segment was considered to present with DLC if the strain rate analysis demonstrated motion reflecting true contraction and if the end of the segmental contraction occurred after the aortic valve closure. DLC is presented as the number of segments demonstrating DLC, expressed as a percentage of the total number of segments evaluated (15–17).

In addition, the septal-posterior wall motion delay was determined as a marker of intraventricular delay. The septal-posterior wall motion delay was defined as the shortest interval between the maximal displacement of the LV septum and that of the posterior LV wall, as determined by M-mode echocardiography in the short-axis view at the papillary muscle level (14).

Clinical evaluation. All patients underwent clinical evaluation at baseline before BVP implantation and at three months after individually optimized BVP. During clinical evaluation, a 6-min hall-walk test and quality-of-life assessment using the Minnesota Living with Heart Failure were performed.

Statistical analysis. All data are presented as the mean values ± SD. Sequential data measurements were analyzed by repeated measures analysis of variance followed by Scheffé’s procedure for multiple comparisons. Proportions were compared using the Fisher exact test. Simultaneous BVP was considered as the reference pacing configuration to compare all sequential BVP configurations and to assess the correlations between the percent change in cardiac output and MR with markers of ventricular dyssynchrony. Pearson’s correlation coefficient was used to quantify correlations between quantitative variables. Statistical significance was established at \( p < 0.05 \).

RESULTS

Implantation of BVP and the study protocol were completed in all patients. In one patient, the LV lead had to be replaced one day after implantation for an acute threshold increase, but this complication did not interfere with the protocol.

The intra-observer correlation values for the TDI parameters and standard echocardiography quantification were determined in 15 patients (0.91 and 0.95, respectively), demonstrating high reproducibility. The intra-observer correlation for the cardiac output calculation was 0.94.

Simultaneous BVP. Compared with baseline evaluation, simultaneous BVP significantly increased the LV filling time from 291 ± 72 ms to 378 ± 53 ms (\( p < 0.01 \), cardiac
output from 2.2 ± 0.6 l/min to 3.0 ± 0.6 l/min (p < 0.001) and significantly decreased the regurgitant volume from 30 ± 14 ml to 19 ± 12 ml (p < 0.01) and EROA from 28 ± 13 mm² to 20 ± 8 mm² (p < 0.01) (Table 2). In addition, compared with baseline, simultaneous BVP significantly reduced interventricular dyssynchrony (p < 0.001), septal-posterior wall motion delay (p < 0.001), intra-LV delaypeak (p < 0.001), intra-LV delayonset (p < 0.001), systolic dyssynchrony index (p < 0.01), and extent of myocardium displaying DLC (p < 0.01).

**Optimized sequential BVP.** Simultaneous BVP was the optimal stimulation configuration for six patients (15%). Pre-activation of the LV lead was optimal for 25 patients (61%) with a VV interval of 12 ms for 9 patients, 20 ms for 9 patients, and 40 ms for 4 patients, and with LV pacing alone for 3 patients (Fig. 1). For the remaining 10 patients

---

**Table 2. Echocardiographic Data on Baseline, Simultaneous, and Optimized Sequential BVP**

<table>
<thead>
<tr>
<th></th>
<th>Before Implantation</th>
<th>Simultaneous BVP</th>
<th>Optimized Sequential BVP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (l/min)</td>
<td>2.2 ± 0.6</td>
<td>3.0 ± 0.6*</td>
<td>3.8 ± 0.6‡</td>
</tr>
<tr>
<td>LV filling time (ms)</td>
<td>291 ± 72</td>
<td>378 ± 53†</td>
<td>408 ± 59§</td>
</tr>
<tr>
<td>EROA (mm²)</td>
<td>28 ± 13</td>
<td>20 ± 8†</td>
<td>12 ± 6§</td>
</tr>
<tr>
<td>Interventricular dyssynchrony (ms)</td>
<td>58.1 ± 28</td>
<td>30.9 ± 18*</td>
<td>30.1 ± 16</td>
</tr>
<tr>
<td>SPWM delay (ms)</td>
<td>63.4 ± 38</td>
<td>31.5 ± 21*</td>
<td>19.2 ± 21§</td>
</tr>
<tr>
<td>Intra-LV delaypeak (ms)</td>
<td>76.4 ± 31</td>
<td>46.2 ± 21*</td>
<td>30.2 ± 17§</td>
</tr>
<tr>
<td>Intra-LV delayonset (ms)</td>
<td>67.8 ± 25</td>
<td>46.3 ± 18*</td>
<td>31.4 ± 19§</td>
</tr>
<tr>
<td>Index of LV dyssynchrony (ms)</td>
<td>44 ± 19</td>
<td>35 ± 13†</td>
<td>26 ± 14§</td>
</tr>
<tr>
<td>DLC (%)</td>
<td>48.6 ± 18</td>
<td>30.6 ± 09†</td>
<td>20.4 ± 09§</td>
</tr>
</tbody>
</table>

*p < 0.001 versus baseline. †p < 0.01 versus baseline. ‡p < 0.01 versus simultaneous BVP. §p < 0.05 versus simultaneous BVP.

Data are presented as the mean value ± SD.

BVP = biventricular pacing; DLC = delayed longitudinal contraction; EROA = effective regurgitant orifice area; LV = left ventricular; SPWM = septal-posterior wall motion.

---

**Figure 1.** Measurements of the electro-mechanical delaypeak of the basal portion of the septal and lateral walls before the implantation, in simultaneous biventricular pacing (BVP), and in optimized sequential BVP (pre-activation of the left ventricular [LV] lead of 20 ms). Important delay (110 ms) between the two segments in spontaneous rhythm, reduction of this delay (50 ms) in simultaneous BVP, and further improvement (0 ms) in optimized sequential BVP.
(24%), pre-activation of the RV lead was optimal with a VV interval of 12 ms for 5 patients and 20 ms for 5 patients. In patients with ischemic heart disease (n = 23 [56%]), the optimal pacing configuration was simultaneous BVP in 13% versus 17% in patients with primitive dilated cardiomyopathy, sequential BVP with LV pre-activation in 61% versus 17% in patients with primitive dilated cardiomyopathy or septal-posterior wall motion delay and (p < 0.05) and index of myocardium displaying DLC (p < 0.05). In contrast, there was no significant change in LV filling time to 408 ± 59 ms (p < 0.05) and the cardiac output to 3.8 ± 0.6 l/min (p < 0.01) and significantly decreased the regurgitant volume to 13 ± 8 ml (p < 0.05) and the EROA to 12 ± 6 mm² (p < 0.05). All parameters of intra-LV dyssynchrony improved significantly with optimized sequential BVP compared with simultaneous BVP (Table 2, Fig. 4). The intra-LV delay onset (p < 0.01) and intra-LV delay peak (p < 0.01) improved to a greater extent than the septal-posterior wall motion delay (p < 0.05), the index of ventricular dyssynchrony (p < 0.05), and the extent of myocardium displaying DLC (p < 0.05). In contrast, there was no significant change in interventricular dyssynchrony (p = NS).

**Relationship between ventricular dyssynchrony and hemodynamic status.** The changes between simultaneous BVP and sequential BVP, RV or LV pacing, in the intra-LV delay onset (r = -0.64, p < 0.001), intra-LV delay peak (r = -0.67, p < 0.001), and index of LV dyssynchrony (r = -0.67, p < 0.001) exhibited highly significant correlations with the changes in cardiac output, whereas the septal-posterior wall motion delay (r = -0.41, p < 0.05) and the extent of myocardial DLC changes (r = -0.48, p < 0.05) revealed a significant but lower correlation. There was no correlation observed between the changes in interventricular dyssynchrony and cardiac output (p = NS) (Table 3, Fig. 2).

Similarly, the changes in the intra-LV delay onset (r = 0.63, p < 0.001), intra-LV delay peak (r = 0.68, p < 0.001), and index of LV dyssynchrony (r = 0.68, p < 0.001) demonstrated highly significant correlations with the changes in the severity of MR (changes in EROA), whereas the extent of myocardial DLC changes revealed a significant but lower correlation (r = 0.51, p < 0.05). There was no significant correlation between the changes in interventricular dyssynchrony or septal-posterior wall motion delay and the change in severity of MR (p = NS) (Figs. 3 and 4).

**Clinical outcome.** All patients completed a review at three months, during which they were paced in the optimized BVP configuration, as determined by the study protocol. Three months of optimized BVP significantly improved the NYHA functional class (1.7 ± 0.6 vs. 3.2 ± 0.5, p < 0.01), quality-of-life score (24 ± 14 vs. 45 ± 17, p < 0.01), and exercise capacity (6-minute walking test: 389 ± 65 m vs. 264 ± 73 m, p < 0.05). Echocardiography demonstrated a significant increase in LVEF (34 ± 6% vs. 28 ± 6%, p = 0.035) and significantly decreased the LV end-diastolic (148 ± 49 cm³ vs. 193 ± 73 cm³, p < 0.01) and LV end-systolic volumes (101 ± 30 cm³ vs. 141 ± 32 cm³, p < 0.01).

**DISCUSSION**

This study presents new information on the evaluation and optimization of BVP in patients with symptomatic drug-refractory heart failure.

First, this study demonstrates that although all echocardiographic parameters of ventricular dyssynchrony improve with simultaneous BVP, individually optimized sequential BVP results in further improvements in these parameters.

Second, these parameters of ventricular dyssynchrony were found to not all be correlated with improved hemodynamic status. Changes in the intra-LV delay onset, intra-LV delay peak, and index of LV dyssynchrony were found to be strongly correlated with changes in cardiac output and reduction in severity of MR, with the lower the value of these markers indicating an improved hemodynamic status. There was an absence of correlation between interventricular dyssynchrony and hemodynamic improvement.

**Reduced ventricular dyssynchrony with BVP.** Cardiac resynchronization therapy has been demonstrated to improve hemodynamic status in selected patients with symptomatic heart failure (1–5). Different pathophysiologic mechanisms have been proposed to explain this hemodynamic benefit; however, the optimal pre-implant parameters predictive of a “response” to BVP remain to be defined. It is increasingly recognized that identification of candidates

---

**Table 3. Correlations Between the Markers of Ventricular Dyssynchrony and Those of Hemodynamic Status**

<table>
<thead>
<tr>
<th>Markers of Ventricular Dyssynchrony</th>
<th>Cardiac Output (% Changes)</th>
<th>Mitral Regurgitation (% Changes)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p Value</td>
</tr>
<tr>
<td>Intra-LV delay peak</td>
<td>-0.67</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Intra-LV delay onset</td>
<td>-0.64</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Index of LV dyssynchrony</td>
<td>-0.67</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>SPWM delay</td>
<td>-0.41</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>%DLC</td>
<td>-0.48</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Interventricular dyssynchrony</td>
<td>-0.24</td>
<td>NS</td>
</tr>
</tbody>
</table>

NS = not significant; other abbreviations as in Table 2.
solely based on the QRS duration is inadequate, whereas inclusion of some direct measurement of ventricular dyssynchrony can assist in identifying potential responders (10,16,18). Recent data indicate that patients with improved ventricular dyssynchrony due to BVP also experience clinical improvement (16,18). However, a direct correlation between hemodynamic status and the change from asynchronous to almost simultaneous movement of the LV walls was missing. The current study demonstrates an improvement in LV hemodynamic performance in parallel with a reduction in all the studied parameters of ventricular dyssynchrony. Importantly, it demonstrates a strong correlation and a proportional change in cardiac output with the degree of reduction in ventricular dyssynchrony, suggesting its potential contribution to the mechanisms whereby CRT leads to clinical benefit.

**Markers of ventricular dyssynchrony.**

**INTERVENTRICULAR DYSSYNCHRONY.** The clinical benefits of CRT were initially attributed to the theoretical reduction of electromechanical interventricular delay associated with synchronous BVP (1–3). Although an attractive postulate, particularly in patients with delayed LV activation, the current study demonstrates the absence of a correlation between interventricular dyssynchrony and hemodynamic status. Sequential BVP can provide an optimization of the interventricular dyssynchrony, but this optimization was not correlated with the optimization of cardiac output, MR, or intra-LV dyssynchrony. These findings illustrate the limitation of patient selection according to their degree of interventricular dyssynchrony.

**INTRA-LV DYSSYNCHRONY.** In contrast, in the current study, we observed a strong correlation between the changes in intra-LV electromechanical delay and the hemodynamic status, in particular, measures of intra-LV electromechanical delays calculated at the onset (intra-LV delay_{onset}), peak (intra-LV delay_{peak}), and index of LV dyssynchrony. Indeed, decreasing the electromechanical dispersion within the left ventricle could be expected to increase efficiency of ventricular contraction, thereby increasing cardiac output. Improvements in these parameters not only correlated with improved cardiac output but also a reduction in the severity of MR. These features could potentially contribute to the observed reverse remodeling of the LV with long-term BVP, which is a major phenotypic component of the pathophysiologic mechanisms of heart failure.

---

**Figure 2.** Correlation between the changes in the parameters of ventricular dyssynchrony and those of cardiac output (CO). A high correlation is shown between the changes in the index of dyssynchrony, the intraventricular delay_{peak}, and the changes in CO; lower correlation for the extent of myocardium displaying delayed longitudinal contraction (DLC); no correlation for the interventricular delay (intra-LV delay). LV = left ventricular.
To further improve the correlation with hemodynamic parameters and probably to better predict the response with BVP, new parameters, including both the delays between the peaks and also the directions of those peaks, might be considered to distinguish between dyskinetic and asynchronous patterns.

This study also observed a significant correlation between variations in the hemodynamic status and variations in the extent of myocardium displaying DLC. However, this correlation in the current study appeared to be less. It may be explained by the fact that DLC represents the proportion of LV segments (among the 12 studied LV segments) contracting after the aortic valve closure. Accordingly, for each segment, the DLC analysis is dichotomic (presence or absence of DLC) without calculating the time spent in DLC for each LV segment.

**Individually optimized sequential BVP.** Most studies have described 20% to 30% of patients as “nonresponders” to simultaneous BVP (6,18,20). Individually optimized sequential BVP may increase the utility of BVP by reducing the number of “nonresponders.” (17,29). Indeed, the benefit of sequential CRT with an individually optimized interventricular delay has been previously demonstrated by Sogaard et al. (17) to increase the diastolic filling time with a reduction in intra-LV dysynchrony compared with simultaneous BVP.

The findings of the current study suggest that the optimal sequence of CRT might be difficult to predict between patients. Although in some individuals, BVP with LV pre-activation results in a significant cardiac output increase (compared with simultaneous BVP), in others such improvements were observed with RV pre-activation. Unfortunately, we could not identify pre-implant parameters predictive of the best sequential BVP configuration. Interestingly, in only 15% of patients was simultaneous BVP found to be the optimal configuration. These observations underscore the importance of individually tailored optimized sequential BVP in CRT at pre-discharge with the aim of best optimizing the cardiac output.

**Study limitations.** The current study demonstrates an acute benefit of optimized sequential BVP over simultaneous BVP in terms of the markers of ventricular dyssynchrony and hemodynamic status. However, the long-term effects on these parameters of each pacing mode were not evaluated and no acute hemodynamic parameter has been
proven to predict the long-term response to cardiac resynchronization therapy. In addition, although this study demonstrates the strong correlation between the acute changes in the parameters of dyssynchrony and hemodynamic status, it has not evaluated their usefulness in the selection of patients most likely to benefit from BVP. The next step will be to define the cut-off value of these parameters of intra-LV dyssynchrony to separate between responders and nonresponders.

The sample size (n = 41) was rather small, but the protocol with 11 different echocardiographic evaluations provided pertinent data for a reliable statistical analysis. The described echocardiographic protocol appears to be too time-consuming for the daily practice but the analysis of all the parameters of dyssynchrony is probably not mandatory to determine the optimal interventricular delay.

The employment of cardiac output is underused in clinical practice or in recent clinical studies. In this study the intra-observer variation for this parameter was 0.94. More than the value of the cardiac output, we analyzed the variations of the cardiac output among the different pacing configuration in a same patient. When we modified the program of the VV delay, the heart rate remained constant and there was a very low variation of the aortic valve diameter. The variations of cardiac output depended on the variations of aortic velocity-time integral. The measurement of this parameter has been demonstrated to be reliable when the sample size is placed at the same site.

**Conclusions.** In patients with symptomatic drug-refractory heart failure, optimization of the ventriculo-ventricular pacing timing results in a substantial reduction of ventricular dyssynchrony that is associated with an improvement in hemodynamic status compared with simultaneous BVP. Some echocardiographic parameters of intra-ventricular dyssynchrony were demonstrated to be strongly correlated with hemodynamic variables and may be a useful adjunct in the selection and optimization of CRT.

Reprint requests and correspondence: Dr. Stephane Garrigue, Hôpital Cardiologique du Haut Lévèque, 19 Avenue de Magellan, Pessac Cedex 33604, France. E-mail: stephane.garrigue@chu-bordeaux.fr.

**REFERENCES**


Bordachar et al.

BVP and Ventricular Dysynchrony

December 7, 2004:2157–65


