Left ventricular resynchronization therapy in a canine model of left bundle branch block

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Heart Failure (HF) affects more than 5,000,000 Americans, with more than 400,000 new cases diagnosed each year. A significant percentage of HF patients presents with an abnormally wide QRS complex, often resulting from complete or partial left fascicular block. Recent studies (3–5, 7, 8, 10, 12, 16, 22) have shown that cardiac resynchronization therapy (CRT) with biventricular (BV) or left ventricular (LV) stimulation in the New York Heart Association functional class III and IV HF patients with wide QRS and left bundle branch block (LBBB) can improve acute hemodynamic function and chronic functional status. Moreover, the acute improvements in systolic function seem to be accompanied by decreased energy consumption in the myocardium (21). It has been reported (4, 10, 16) that a wide QRS complex and the presence of LBBB may be useful clinical markers to identify those patients who may benefit from CRT.

The mechanisms suggested for the hemodynamic improvements observed with CRT include restoration of optimal atrioventricular (AV) timing and septal-LV lateral-posterior wall recoordination by LV or BV stimulation. However, the isolated role of LBBB in the acute hemodynamic improvements observed during CRT has not been previously studied. The aim of this study, therefore, is to develop an animal model of isolated LBBB, which increases the width of the QRS complex by delaying the activation of the LV lateral free wall. Second, we sought to use this LBBB model to test the hemodynamic impact of this electrical delay and whether CRT would restore cardiac function by correcting the asynchronous contraction of the LV.

METHODS

This study was conducted in nine adult greyhound dogs of either sex weighing 29.9 ± 3.9 kg. The stimulation protocol was performed in six dogs to obtain simultaneous electrophysiological and hemodynamic data. Echocardiography was performed in three more dogs to determine the relative coordination of the septal and LV free wall motion. Dogs were premedicated with 10 mg of butorphanol and 0.5 mg of acepromazine administered subcutaneously. General anesthesia was induced with 150 mg of ketamine and 7.5 mg of diazepam, and was maintained with the use of isoflurane gas and a semi-pressure-volume-regulated ventilator. Heart rate and blood pressure were monitored during the procedure to ensure that a deep level of anesthesia was maintained.

All experiments were carried out in accordance with National Institutes of Health’s Guide for the Care and Use of Laboratory Animals. The Institutional Animal Care and Use Committee of Guidant Corporation approved this protocol.

Catheterization. After the jugular veins and the right carotid artery were exposed, a 7-Fr pulmonary-wedge pressure catheter (Bard; Billerica, MA) was advanced into the ostium of the coronary sinus under fluoroscopic guidance. A coronary venogram was generated by inflation of the balloon and injecting renographin into the coronary sinus. The image was then stored and displayed on a monitor to guide stimulation

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lead placement. After the balloon catheter was removed, two 8-Fr custom-designed guiding catheters were advanced into the ostium of the coronary sinus, which served as conduits for the coronary vein leads. Two coronary vein prototype leads were implanted at the apex and base of the LV in anterior and lateral branches of the great cardiac vein, as described by Auricchio et al. (2). A unipolar lead (Sweet Tip, model 4169, Guidant; St. Paul, MN) and a bipolar lead (model 4269, Guidant) were placed in the right ventricular (RV) apex and right atrium (RA) for stimulation and sensing, respectively. Two 8-Fr dual-transducer pressure catheters (model SPC-780c, Millar Instruments; Houston, TX) were placed in RV and LV to measure RV, LV, and aortic pressures (Fig. 1). Pressure catheters and stimulation leads were connected to a custom external stimulation system (FlexStim, Guidant) to acquire hemodynamic signals and execute an acute stimulation protocol.

Creation of LBBB. To generate LBBB, an 8-Fr introducing sheath was inserted into the carotid artery. A 7-Fr ablation catheter with a 4-mm tip (model D7-DL-252-PS, Cordis Webster; Baldwin Park, CA) was then advanced retrogradely across the aortic valve into the LV and manipulated under fluoroscopic guidance until a discrete left bundle potential (LBP) was found in the distal bipole and recorded via a multichannel oscilloscope (Fig. 2). The LBP was confirmed by an atrial and ventricular amplitude ratio <1:10 and an interval of <35 ms from LBP to QRS complex. LBB ablation was performed with a radio frequency (RF) generator (model RFG 3-E, Radionics; Burlington, MA) delivering 480 kHz (±10%) unmodulated sine wave energy. RF energy of 30 W was applied for 60 s between the tip of an ablation catheter and a metal plate placed on the back of each dog (15). The impedance was continuously monitored during the energy application. A drop in impedance was considered a sign of good tissue contact and adequate heating (14). The energy application was continued for 60 s or until a sudden rise in impedance occurred, which indicated that coagulation had formed at the tip of the catheter.

A surface electrocardiogram (ECG) was recorded before and after each ablation (Cardioperfect Portable, Cardioperfect; Atlanta, GA). Reported ECG parameters (i.e., HR, PR interval, QRS duration, and axis) are those automatically calculated by Cardioperfect system. Complete LBBB was defined by a prolongation of QRS duration to >100 ms; QRS positive in leads I, II, III, and aVF with notched R wave and negative in leads aVR, aVL; absent or small Q wave in lead I (see Ref. 6), and loss of LBP electrogram.

Stimulation protocol. Before ablation and 30 min after successful LBB ablation, a software-controlled stimulation system (FlexStim, Guidant) was connected to the animal and an acute stimulation protocol was executed. The protocol was designed to measure the immediate hemodynamic effects of CRT while accounting for local baseline shifts. This allowed statistical comparison of multiple stimulation combinations within individuals as described previously (4). Briefly, RV, LV, or BV stimulation was performed in a VDD mode (i.e., atrial sense followed by ventricular stimulation) for six beats after 14 sinus beats at 1 of 4 preset AV delays. The AV delays were determined by equally dividing the interval between 8 ms and the intrinsic AV interval −30 ms into four parts. Each combination of stimulation chamber and AV delay was repeated four times in a random order. Intracardiac electrogram (EGM) and pressure signals were simultaneously recorded to the computer hard disk for off-line analysis. Off-line analysis was performed with custom software that automatically calculated the following: aortic diastolic pressure (ADP), aortic systolic pressure (ASP), pulse pressure (PP = ASP − ADP), LV maximum pressure derivative over time (LV dP/dt max), minimum dP/dt (LV −dP/dt), and LV end-diastolic pressure (LVEDP).

Echocardiography. Because positive hemodynamic response during LV or BV stimulation after LBB ablation was observed in all six dogs, echocardiography was adopted to qualitatively look at the LV and septal wall motion to prove the hypothesis that preexcitation of LV in the presence of LBBB improves cardiac function by coordinating the LV
contraction. M-mode echocardiograms (Sonos 2500, Hewlett-Packard) were obtained in three dogs by placing a 2.5-MHz transducer on the right side of the canine thorax. The probe was positioned to track the interventricular septum and LV posterior wall motion, at the level of the tips of the papillary muscles. Echo recordings were obtained during normal sinus rhythm at baseline, after LBB ablation, and during LV stimulation at an optimum AV delay selected by the FlexStim stimulation protocol in the presence of LBBB. Because LV and BV stimulation improved hemodynamics to a similar extent, LV stimulation was applied during Echo examination in all three dogs. Echocardiograms from each experiment were recorded on videocassette for off-line review.

Gross necropsy. After each experiment, the heart was excised and rinsed with saline. The left ventricle was carefully dissected and the endocardial surface was wiped with Lugol’s solution to expose the structure of the conduction system (27). Each necropsy was archived by photography and the location of all ablation lesions was determined by visual inspection.

Statistical analysis. Hemodynamic response to stimulation was determined by following a previously published method (4). The percentage change for a given hemodynamic parameter was calculated from the value of the parameter during stimulation compared with the average value during the immediately preceding six nonpaced beats (i.e., local baseline) for each tested AV delay. The first two paced beats were ignored in the analysis because their hemodynamic response is biased by the diastolic behavior of the preceding nonpaced beat and the transitory ventricular V-to-V decrease caused by the switch to a short AV delay (4). In addition, sequences containing ectopic beats were automatically repeated by the system and ignored during the off-line processing phase. A two-way ANOVA was applied to analyze differences between stimulation chambers (RV, LV, and BV) before and after LBB ablation. The AV delay, LBBB, and stimulation chamber were considered the treatment variables. A paired t-test was used to compare the effects of two group data. A P value of < 0.05 was considered statistically significant. Average data are shown as means ± SD.

RESULTS

Creation of LBBB. A median of one RF ablation (range 1–3) was performed to create block of LBB in nine dogs. A successful ablation was confirmed by prolonged QRS duration (range 123–160 ms), LBBB morphology (see the description in METHODS), and a constant PR interval on the surface ECG (Fig. 3). The mean interval from LBP to ventricular electrogram at the successful ablation site was 33.4 ± 2.5 ms (range 29–35 ms). Successful LBB ablation doubled QRS duration (P < 0.001) and shifted QRS axis (P < 0.05) with no substantial changes in HR and PR interval (Table 1).

The left ventricle was dissected along its free wall after each experiment. The left conduction fibers, treated with Lugol’s solution, divided into fascicles immediately after the bundle branch penetrated the septum from right side to left side underneath the right aortic semilunar valve. There were no common LBBs seen on the left side of the septum. The ablation site was located where the fascicles emerged from the myocardium.

Interventricular synchrony. Table 2 reports changes of interventricular delay as defined by either the interval between RV and LV deflection on the EGM or the onset of systole on the RV and LV pressure curves for preablation (baseline), postablation (LBBB), and during BV or LV stimulation. The value of LV pressure onset during stimulation reported in Table 2 was taken from the stimulation configuration (either LV or BV) with the percentage of LV dP/dt max response in each dog. At baseline (sinus rhythm with intact conduction system), RV and LV started to activate and contract almost simultaneously (~1 ms). After the LBB was ablated, however, both LV activation and contraction were significantly delayed (~30 ms) relative to RV (P < 0.001 for both). In the presence of LBBB, either BV or LV stimulation significantly shortened RV-LV contraction delay and improved interventricular asynchrony (P < 0.001 vs. LBBB), although it did not completely restore the normal contraction sequence (P < 0.05 vs. baseline).

The effects of LBB ablation and RV, LV, or BV stimulation on LV and aortic hemodynamics are summarized in Table 3. LBB ablation significantly decreased LV contraction and relaxation velocity (P < 0.001 vs. baseline), indicating remarkable impairment of both inotropic and lusitropic function of the LV. LBBB also increased the LVEDP by >50% and reduced aortic PP by >10%, affecting both AoSP and AoDP. LV or BV stimulation improved both LV systolic and diastolic function and increased PP. Figure 4, A and B, shows the average percentage change of LV dP/dt max and aortic PP for five stimulation configurations: RV apex, LV apex, LV base, LV apex + LV base, and LV base + RV apex. Data are reported for six dogs at four AV delays before and after LBB ablation. Before ablation, all stimulation decreased both %LV dP/dt max (Fig. 4A) and aortic %PP (Fig. 4B) at all AV delays and all configurations compared with local baseline. RV apex stimulation decreased both parameters significantly more than LV or BV stimulation (P < 0.001). After ablation (Fig. 4), LV and BV stimulation improved hemodynamics at all AV delays and maximally increased LV dP/dt by 15.7 ± 7.6% and PP by 7.0 ± 7.7%.
at an AV delay of ~70 ms ($P < 0.001$ for both vs. local baseline). LV single-site stimulation, LV dual-site stimulation, and BV stimulation all increased the LV dP/dt and PP significantly and to a similar extent at each AV delay. RV apex stimulation alone, however, had little effect on LV dP/dt ($<3\%$ at all AV delays) and worsened PP at all AV delays compared with local baseline ($P < 0.001$).

**Electrophysiology.** During baseline (Fig. 5A), the interventricular septum moved towards the LV posterior wall during ventricular contraction whereas the posterior wall of the LV moved anteriorly towards the septum. The simultaneous inward movement of both the septum and free wall efficiently ejected the blood from the LV into the aorta. In contrast, LBBB delayed LV activation (Table 1) and caused the septum to move downward after the onset of electrical depolarization (vertical line in Fig. 5B), followed by movement away from the LV posterior wall at the time when the LV started to contract. This motion produced a downward beaking (arrow in Fig. 5B) of the left septum shortly after depolarization and a parallel movement of the interventricular septum and the LV posterior wall during ventricular ejection. Such a paradoxical septal motion caused inefficient propelling of blood from LV chamber into the aorta. However, in the LBBB model, LV stimulation with a shortened AV delay produced M-mode traces (Fig. 5C) similar to those seen in normal sinus rhythm.

**DISCUSSION**

In this study, we have developed an animal model of LBBB using focal endocardial RF ablation. LBBB resulted in delayed LV activation and a corresponding delayed LV systole. This phenomenon was associated with an asynchronous contraction of the septum and the LV free wall. This asynchronous activation and contraction resulted in decreased LV global function as evaluated by PP and LV dP/dt$max$, respectively. Because PP and stroke volume changes have been proven to be correlated with each other during both steady-state pacing in a CHF patient population (16) and burst pacing using FlexStim protocol in an animal study (19), and provided that LBB ablation did not affect HR (Table 1), the increase in LVEDP and decrease in PP was most likely caused by a less efficient LV pump and associated with a lower cardiac output. Although ventricular stimulation in normal dogs worsened global ventricular function, LV or BV stimulation significantly improved LV systolic and diastolic function in canines with LBBB, probably by correcting the interventricular contraction asynchrony. These findings support the hypothesis that LV and BV stimulation provide hemodynamic benefit by improving electromechanical coordination of LV contraction in the presence of LBBB.

This hypothesis is further supported by the fact that the improvement in global LV function as evaluated by dP/dt$_{max}$ obtained with LV and BV stimulation occurred at every AV delay from 10 to 100 ms (Fig. 4A). PP, in contrast to LV dP/dt$_{max}$, showed a marked decrease when the AV delay was shortened <70 ms (Fig. 4B) which indicated that preload was an important factor for PP. The independence of the paced AV delay and the magnitude of the LV dP/dt$_{max}$ improvement may indicate that neither left-sided AV synchrony nor preload is the primary mechanism mediating LV dP/dt$_{max}$ improvements provided by LV and BV stimulation in this model.

**Canine LBBB model.** LBB ablation produced interventricular asynchrony and deteriorated hemodynamics in the normal canine heart. During sinus rhythm, the onset of the LV electrogram and the upstroke of the LV pressure occurred almost simultaneously with the RV, and the LV pressure exceeded the RV pressure throughout the cardiac cycle. The echocardiogram showed that the interventricular septum moved posteriorly as it began to contract (Fig. 5A). This coordinated contraction pattern was immediately lost with LBBB (Table 2). In the LBBB heart, the LV free wall was in its relaxation phase while the RV started to contract.

### Table 1. ECG parameters before and after LBB ablation

<table>
<thead>
<tr>
<th></th>
<th>HR, beats/min</th>
<th>PR interval, ms</th>
<th>QRS duration, ms</th>
<th>QRS axis, degrees</th>
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<tr>
<td>Baseline</td>
<td>90.3 ± 19.0</td>
<td>126.6 ± 15.7</td>
<td>71.0 ± 8.4</td>
<td>82.7 ± 4.7</td>
</tr>
<tr>
<td>LBBB</td>
<td>92.0 ± 23.1</td>
<td>129.6 ± 21.5</td>
<td>139.2 ± 12.1</td>
<td>89.3 ± 2.0</td>
</tr>
</tbody>
</table>

Values are means ± SD. HR, heart rate; LBBB, left bundle branch block; ECG, electrocardiogram. *$P < 0.05$ and $P < 0.001$ vs. baseline.

### Table 2. Interventricular delay before and after LBB ablation and during LV or BV stimulation in the presence of LBBB

<table>
<thead>
<tr>
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<th>Activation Timing From RV to LV, ms</th>
<th>Onset of Systole From RVP to LVP, ms</th>
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<tbody>
<tr>
<td>Baseline</td>
<td>1.2 ± 5.4</td>
<td>0.9 ± 7.1</td>
</tr>
<tr>
<td>LBBB</td>
<td>35.4 ± 26.0b</td>
<td>26.5 ± 7.0a</td>
</tr>
<tr>
<td>BV or LV</td>
<td>3.3 ± 5.7c</td>
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Values are means ± SD. BV, biventricular; LV, left ventricle; RV, right ventricle; RVP, right ventricular pressure; LVP, LV pressure.

Interventricular delay, calculated by the difference of activation timing between the deflection of RV apex and the deflection of LV apex on their intracardiac EGMs and contraction timing from the upstroke of RV systole to the upstroke of LV systole on their pressure curves at the baseline, after left bundle branch ablation, and during LV or stimulation in LBBB. *$P < 0.05$ and $P < 0.001$ vs. baseline; **$P < 0.001$ vs. LBBB. AJP-Heart Circ Physiol • VOL 282 • JUNE 2002 • www.ajpheart.org
causing an abrupt posterior motion of the interventricular septum. The delayed onset of LV contraction, occurring as the septum begins to relax, resulted in a paradoxical septal movement whereby the septum moved away from the LV posterior wall during LV systole (Fig. 5B: Beak) and a decreased septal contribution to stroke volume.

At the myocyte level, delayed and uncoordinated contraction may cause myofilament cross-bridge detachment (e.g., in septal myocardium). Ter Keurs et al. (26) observed that the fraction of tension redeveloped declines following transient myocyte lengthening at times after the peak twitch tension has occurred. This is likely due to decreased cytosolic calcium levels following the peak tension, which is insufficient to allow all the myofilament cross bridges to reattach. At the whole heart level, this phenomenon may further reduce LV global systolic function (i.e., decrease of LV dP/dt max and aortic PP; Table 3). Interestingly, late activation and contraction of the LV also resulted in diastolic abnormalities demonstrated by altered LV −dP/dt (Table 3).

Both BV and LV stimulation resynchronized interventricular contraction in LBBB. Simultaneous activation of both ventricles via BV or LV stimulation with an optimal AV delay (Table 2) allowed ejection to occur in both ventricles before relaxation of the septum and corrected the paradoxical septal-LV free wall motion as assessed by echo (Fig. 5C). During CRT, LV dP/dt max and aortic PP increased by 15.7 ± 7.6% and 7.0 ± 7.7%, respectively. Because RV stimulation created a conduction pattern similar to LBBB (18), it is not surprising that it worsened LV systolic function the most in the normal heart. Moreover, RV stimulation provided the least benefit in the presence of LBBB.

Comparison of canine LBBB model to human LBBB. The asynchronous behavior and the hemodynamic changes caused by LBB ablation in our model are similar to the abnormalities found in LBBB patients. Abnormal interventricular septal motion in patients with LBBB has been described since the 1970s (1, 9, 20). Abbasi et al. (1) reported that 14 of 17 patients with complete LBBB had abnormal interventricular septal motion analogous to that observed in the present study. Furthermore, in 2 of 14 cases with intermittent LBBB, abnormal septal motion was present only during LBBB. This abnormality was explained by asynchronous LV contraction with early activation and contraction of the septum but delayed activation and contraction of the LV free wall (20).

Table 3. Hemodynamic data at the baseline, after LBB ablation, and during ventricular pacing

<table>
<thead>
<tr>
<th></th>
<th>LV dP/dt, mmHg/s</th>
<th>LV −dP/dt, mmHg/s</th>
<th>LVEDP, mmHg</th>
<th>AoSP, mmHg</th>
<th>AoDP, mmHg</th>
<th>PP, mmHg</th>
</tr>
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<tbody>
<tr>
<td>Baseline</td>
<td>1,162.1 ± 106.1</td>
<td>−1,310.9 ± 183.3</td>
<td>5.1 ± 2.1</td>
<td>94.5 ± 4.7</td>
<td>74.1 ± 5.0</td>
<td>20.5 ± 1.7</td>
</tr>
<tr>
<td>LBBB</td>
<td>830.6 ± 135.8</td>
<td>−910.1 ± 258.6</td>
<td>7.9 ± 3.7</td>
<td>87.0 ± 18.2</td>
<td>69.1 ± 17.2</td>
<td>18.0 ± 2.9</td>
</tr>
<tr>
<td>BV</td>
<td>899.1 ± 195.8</td>
<td>−943.1 ± 266.3</td>
<td>7.6 ± 2.9</td>
<td>90.5 ± 18.4</td>
<td>71.4 ± 16.8</td>
<td>19.1 ± 3.5</td>
</tr>
<tr>
<td>LV apex</td>
<td>923.7 ± 131.2</td>
<td>−989.7 ± 269.0</td>
<td>7.2 ± 2.6</td>
<td>89.8 ± 17.4</td>
<td>69.9 ± 15.8</td>
<td>19.7 ± 2.7</td>
</tr>
<tr>
<td>LV base</td>
<td>901.2 ± 205.4</td>
<td>−898.6 ± 236.1</td>
<td>7.3 ± 2.4</td>
<td>89.1 ± 18.4</td>
<td>69.7 ± 16.5</td>
<td>19.4 ± 3.6</td>
</tr>
<tr>
<td>LV a + b</td>
<td>934.8 ± 159.3</td>
<td>−967.0 ± 235.1</td>
<td>7.7 ± 3.2</td>
<td>91.0 ± 18.8</td>
<td>71.3 ± 16.8</td>
<td>19.7 ± 3.3</td>
</tr>
<tr>
<td>RV</td>
<td>854.7 ± 203.3</td>
<td>−1,015.6 ± 336.8</td>
<td>7.7 ± 3.0</td>
<td>92.7 ± 17.7</td>
<td>74.8 ± 15.4</td>
<td>17.9 ± 3.7</td>
</tr>
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</table>

Values are means ± SD. LVEDP, LV end-diastolic pressure; AoSP, aortic systolic pressure; AoDP, aortic diastolic pressure; BV, RV apex and LV base stimulation; LV apex, LV apical stimulation; LV base, LV basal stimulation; LV a + b = LV apex + LV base stimulation; RV, RV apical stimulation. Values of LV maximum pressure derivative over time (dP/dt max) and pulse pressure (PP) (B) before and after LBBB.

Fig. 4. Effect of pacing site and atrioventricular (AV) delay on LV maximum pressure derivative over time (dP/dt) (A) and pulse pressure (PP) (B) before and after LBBB.

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This LV activation and contraction delay was also described by Grines et al. (13) in LBBB patients, where LV activation was delayed 85 ± 31 ms compared with normal subjects (62 ± 5%, \( P < 0.05 \)) (13). Xiao et al. (28, 29) noted that in patients with dilated cardiomyopathy, LBBB was associated with prolonged systolic activity by increasing pre-ejection and relaxation times and consequent loss of LV filling time that would likely limit stroke volume.

Clinical relevance. As early as the 1970s, researchers (11) proposed that LBBB could be a predisposing factor for subsequent congestive cardiomyopathy; however, it was believed that congestive cardiomyopathy would develop only with additional specific influences, such as arterial hypertension, viral infection, alcohol, or pregnancy. Later, Kuhn et al. (17) observed that latent cardiomyopathy might be an advanced stage for patients with lone LBBB. Furthermore, in the Framingham Heart study, people with acquired bundle branch block, particularly LBBB, were more likely to develop advanced cardiovascular disease (23). Several studies (24, 25) have also found increased mortality in patients with LBBB, and the effect of LBBB on mortality is most pronounced in those with severe LV dysfunction.

Our data demonstrate that CRT with BV or LV stimulation can improve the hemodynamic and mechanical deterioration resulting from LBBB. Recent studies (3–5, 7, 8, 10, 12, 16, 22) have shown that LV or BV preexcitation with atrial-synchronous stimulation while sensing in the right atrium (VDD mode) improves systolic function in patients with dilated cardiomyopathy and LBBB. In contrast to positive inotropes such as dobutamine that concomitantly elevate myocardial oxygen demand, VDD stimulation enhances systolic function while at the same time decreasing the energy requirements of the failing heart (21).

Limitations. The LBBB model created in the present study was discrete and may not mimic diffuse changes of the conduction system due to progressive chronic pathological changes in HF patients with dilated cardiomyopathy. This LBBB model was developed in normal hearts that exhibit no other pathophysiological alterations such as myocardial fibrosis, necrosis, or apoptosis. Results from experimental models or humans with more complex substrates may differ from those of the present study. However, our model was intended to isolate the conduction delay component observed in many HF patients without the confounding effects of other pathophysiological substrate changes. The model is intended to serve as a building block for the inclusion of more complex myocardial insults to more closely match the clinical condition. Regardless, the hemodynamic improvement observed during CRT in this study was quite similar to that observed in HF patients with various etiologies and extent of conduction delay.

In conclusion, we have shown that LBBB induced in an otherwise normal heart caused an asynchronous LV activation and contraction. These changes were associated with a paradoxical septal-LV free wall motion. Cardiac resynchronization therapy with LV and BV stimulation significantly improved LV function in this model by improving the pattern of ventricular excita-

Fig. 5. M-mode echocardiograms of the left ventricle at baseline (A), after LBBB (B), and during LV stimulation in the presence of LBBB (C). RV, right ventricular chamber; IVS, interventricular septum; LV, left ventricular chamber; LVPW, posterior wall of the left ventricle. Vertical line marks the beginning of QRS complex.
tion and the corresponding pattern of interventricular contraction. Our data support the hypothesis that LV and BV stimulation improve acute hemodynamics by improving electromechanical coordination of LV contraction in the presence of LBBB. Because this model mimics some of the functional abnormalities found in LBBB patients, it could be a useful experimental building block for further studying the mechanism of acute and chronic CRT in the treatment of patients with left bundle conduction abnormalities accompanied by HF and other myocardial diseases.

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REFERENCES


