Right Bundle Branch Block and Impaired Left Ventricular Function as Evidence of a Left Ventricular Conduction Delay

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Background The indications and efficacy of cardiac resynchronization therapy (CRT) have not been sufficiently clarified in patients with right bundle branch block (RBBB).

Methods and Results This study included 55 patients with normal QRS morphology and duration (Control-Gr) and 49 patients with complete RBBB (CRBBB-Gr). Using tissue Doppler imaging, the time difference (TD) between the electromechanical delay of the septal wall, left ventricular (LV) lateral wall, and right ventricular free wall were measured. Using tissue tracking imaging, the coefficient of the time variation from the beginning of the QRS to the peak displacement time of 6 regions of the LV (CV-PMD_{LV}) was calculated. The TD between the septal wall and that of the LV lateral wall (TD_{SEPT-LAT}) did not differ between the Control-Gr and RBBB-Gr. However, a significant difference was found in the TD_{SEPT-LAT} between the CRBBB patients with LV systolic dysfunction (ejection fraction (EF) ≤50%) and those with normal LV function (EF >50%; $p<0.001$). The CV-PMD_{LV} was greater in the CRBBB patients with LV systolic dysfunction than in those with a normal LV function ($p<0.05$). The RBBB-Gr patients with LV dysfunction and a great TD_{SEPT-LAT}, improved clinically after the CRT.

Conclusions The presence of RBBB and LV dysfunction may indicate LV dyssynchrony and a heterogeneous mechanical dysfunction. (Circ J 2008; 72: 120–126)

Key Words: Heart failure; Right bundle branch block; Tissue Doppler imaging; Tissue tracking imaging; Ventricular dyssynchrony

Cardiac resynchronization therapy (CRT) in patients with advanced heart failure (HF) and a ventricular conduction delay improves symptoms and functional capacity, induces reverse remodeling, and reduces morbidity, and probably mortality.^{1–4} It is well known that although the vast majority of CRT recipients present with left bundle branch block (LBBB), up to 10% of the minority have nonspecific conduction delays or right bundle branch block (RBBB).^{2,5} Recently, RBBB was proven to be an important predictor of mortality in patients with congestive HF, just as has LBBB.⁶ However, patients with RBBB and congestive HF have not been adequately evaluated because of an under-representation in the referenced studies. Therefore, which type of patients with RBBB has ventricular conduction delays, and thus are good candidates for CRT, has not been fully clarified.

Methods

Study Population

This study included 104 patients (34 women, 70 men; mean age, 59±15 years) who underwent conventional echocardiography, including tissue Doppler imaging (TDI) and tissue tracking imaging (TTI) between January 2002 and December 2004. Of these, 49 consecutive patients had complete RBBB (CRBBB) with a duration of the QRS complex of ≥120 ms (RBBB-Gr). The remaining 55 had normal morphology of the QRS complex and narrow QRS (<120 ms; Control-Gr), and were randomly selected so that the data collected would be statistically matched to the RBBB-Gr patients in terms of age and gender (Table 1). In total, 33 patients had hypertension, 5 dilated cardiomyopathy, 5 mitral valvular diseases, 2 hypertrophic cardiomyopathy, 1 congenital heart disease, 3 ischemic heart disease, and the remaining 93 (89%) had no structural heart disease. Echocardiography demonstrated that the mean left ventricular ejection fraction (LVEF) was 64.2±11.8% (range, 12–83), and left ventricular end-diastolic diameter (LVEDD) 46.5±8.5 mm (range, 34–75). All patients were in sinus rhythm, and the mean QRS duration was 113.8±33.3 ms (range, 70–200). All patients in the Control-Gr had LVEF >50% (range, 51–83).

Echocardiographic Examination Including TDI and TTI

The left ventricular (LV) dimensions were measured by M-mode echocardiography in the parasternal long-axis view. The LVEDD was measured at the onset of the QRS

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complex, and the LV end-systolic dimension (LVESD) was defined as the smallest LV cavity.^{7,8} Pulsed-wave TDI and TTI were performed with a Vivid 7 echocardiography system (GE Vingmed Ultrasound, Horten, Norway) after completing conventional echocardiography.^{7,8} The images were obtained using a 1.5–4.0 MHz phased array transducer (M3S transducer; GE Vingmed Ultrasound) at a depth of 10–16 cm in the LV apical 4-chamber view. The image sector width was set as narrow as possible to allow a frame rate acquisition greater than 100 frames/s. At least 2 cine loops of raw TDI data, each containing 3 cardiac cycles, were stored online as digital scan-line data using the Vivid 7 scanner and were retrieved later and analyzed using the built-in analysis tools.

The electromechanical conduction delays were measured by TDI.^{7,8} The region of interest was positioned at the basal portion of the septum and lateral wall of the LV and the free wall of the right ventricle (RV), and the time from the onset of the QRS to the peak myocardial sustained systolic velocity was measured (Figs 1A,B). The peak myocardial sustained systolic velocity was defined as the maximum velocity during systole, excluding the isovolumic contraction.^{7,8} The time difference between the electromechanical delay of the septal wall and that of the LV lateral wall (TD_{SEPT-LAT}) was defined as the intraventricular conduction delay in the LV. We also measured the time difference between the electromechanical delay of the LV lateral wall and that of the RV free wall (TDRV-LAT) and that between the RV free wall and septum (TDRV-SEPT).

TTI enabled visualization of the longitudinal motion in each myocardial segment.^{9,10} It displayed the integral of the tissue velocity during systole, which equaled the distance motion along the Doppler axis during systole (Figs 1C,D). To assess LV synchrony, a quantitative tissue displacement–time curve was used for the analysis. Using this curve, the interval from the QRS onset to the peak myocardial displacement (PMD) in each region could be obtained (Fig 1D). We determined the coefficient of the variation in the time of the PMD in 6 regions (base, mid-point between the basal and apical portions, and mid-point between the 2 points on the

Table 1 Clinical Characteristics and Measurement Variables in the Control-Gr and RBBB-Gr Patients

	Control-Gr (n=55)	RBBB-Gr (n=49)	p value
Gender (M/F)	34/21	36/13	0.21
Age (years)	56.4±14.7	62.1±14.6	0.0502
Structural heart disease (%)	8 (14.5)	8 (16.3)	0.98
LVEDD (mm)	45.9±4.8	47.9±9.6	0.18
LVESD (mm)	28.5±4.5	33.1±10.8	<0.01
LVEF (%)	68.3±6.7	59.5±14.3	<0.0001
QRS duration (ms)	82.7±6.8	145.3±19.8	<0.0001
Medication (%)			
ARBs/ACEI	4 (7)	8 (16)	0.26
Ca antagonists	12 (21)	6 (12)	0.30
-blockers	6 (11)	6 (12)	0.92
Digitalis	0 (0)	4 (8)	0.10
Diuretics	1 (2)	6 (12)	0.08
Antiarrhythmic drugs	4 (7)	7 (14)	0.40
TD			
TD _{SEPT-LAT} (ms)	15.3±17.5	19.5±25.0	0.33
TDRV-LAT (ms)	32.2±26.6	55.2±32.1	<0.0005
TDRV-SEPT (ms)	23.1±20.7	51.3±33.9	<0.0001
CV-PMD _{LV} (%)	3.7±2.9	7.2±5.9	<0.0005

Values are the mean ± standard deviation.

The left ventricular (LV) lateral wall (TD_{SEPT-LAT}) is the intraventricular conduction delay in the LV. The time difference (TD) between the electromechanical conduction delay (EMD) of the septum and that of the left ventricular (LV) lateral wall (TD_{SEPT-LAT}), EMD of the LV lateral wall and that of the right ventricular (RV) free wall (TDRV-LAT), and EMD of the RV free wall and that of the septum (TDRV-SEPT) were determined by tissue Doppler imaging. The coefficient of variation in the time of the peak myocardial displacement in 6 regions of the LV (CV-PMD_{LV}) was determined by tissue tracking imaging. Control-Gr, normal QRS morphology and duration; RBBB-Gr, right bundle branch block with a duration of the QRS complex of ≥120 ms; ED(S)D, end-diastolic (systolic) dimension; EF, ejection fraction; ARB, angiotensin II receptor blocker; ACEI, angiotensin-converting enzyme inhibitor; Ca, calcium.

septal and lateral walls) of the LV (CV-PMD_{LV}; Fig 1D).⁸

We confirmed that the regional myocardial velocity curves obtained by TDI and the PMD curve by TTI were identical or nearly identical, and 3 beats were measured and averaged for each measurement. Two experienced observers

Fig 1. (A) Position of the region of interest (ROI) for the measurement of the time difference between the electromechanical delay of the septal wall (SEPT), left ventricular lateral wall (LAT), and that of the right ventricular free wall (RV). (B) Measurement of the time difference between the electromechanical delay of the septal wall and that of the left ventricle (LV) lateral wall (TD_{SEPT-LAT}). The ROI was positioned at the basal portion of the septum and lateral wall, and the time from the onset of the QRS to the peak myocardial sustained systolic velocity was measured. The time difference between these was defined as the TD_{SEPT-LAT}. (C) Position of the ROIs for the measurement of the coefficient of variation in the time of the peak myocardial displacement (PMD) in 6 regions of the LV. (D) Quantitative tissue displacement–time curves obtained from the 3 regions of the LV lateral wall in a healthy control subject. The coefficient of variation in the time at which the PMD was recorded in 6 regions of the LV was calculated. L(R)A, left (right) atrium.

Table 2 Clinical Characteristics and Measurement Variables of the RBBB Patients With LV Systolic Dysfunction (LVEF $\leq$ 50%) and Those With Normal LV Function (LVEF $>$ 50%)

	Normal LV function (n=41)	Reduced LV function (n=8)	p value
Gender (M/F)	30/11	6/2	0.74
Age (years)	60.9 $\pm$ 15.3	68.3 $\pm$ 8.3	0.20
Structural heart disease (%)	1 (2)	7 (88)	<0.0001
LVEDD (mm)	44.7 $\pm$ 4.3	64.4 $\pm$ 12.5	<0.0001
LVESD (mm)	29.0 $\pm$ 4.0	53.4 $\pm$ 12.9	<0.0001
LVEF (%)	64.7 $\pm$ 7.0	32.8 $\pm$ 12.3	<0.0001
QRS duration (ms)	140.0 $\pm$ 15.3	172.5 $\pm$ 18.3	<0.01
Medication (%)			
ARBs/ACEI	3 (7)	5 (63)	<0.001
Ca antagonists	5 (12)	1 (13)	0.57
-blockers	1 (2)	5 (63)	<0.0001
Digitalis	1 (2)	3 (38)	<0.01
Diuretics	0 (0)	6 (75)	<0.0001
Antiarrhythmic drugs	1 (2)	6 (75)	<0.0001
TD			
TDSEPT-LAT (ms)	14.0 $\pm$ 12.1	47.4 $\pm$ 48.9	<0.0005
TDRV-LAT (ms)	53.1 $\pm$ 31.4	65.9 $\pm$ 35.7	0.31
TDRV-SEPT (ms)	48.8 $\pm$ 31.0	64.0 $\pm$ 46.8	0.25
CV-PMD _{LV} (%)	6.4 $\pm$ 5.1	11.7 $\pm$ 7.7	<0.05

Values are the mean $\pm$ standard deviation.

RBBB, right bundle branch block. Other abbreviations as in Table 1.

who were familiar with TDI and TTI and unaware of the clinical status of the patients analyzed the data. In the RBBB-Gr, each parameter was also compared between patients with an impaired LV systolic dysfunction (LVEF $\leq$ 50%) and those with normal LV function (LVEF $>$ 50%)^{11,12}

Statistical Analysis

All variables are expressed as the mean $\pm$ SD, and were compared using Student's paired t-test or non-parametric test as appropriate. The categorical variables were compared using a chi-squared analysis, using the Yate's correction if necessary. An overall Chi-square test for a 2 $\times$ n table was constructed when comparisons involved $>$ 2 groups. Correlations between variables were assessed by Pearson's linear correlation and tested using Fisher's z transformation.

A p-value $<$ 0.05 was considered statistically significant.

Results

Clinical Characteristics, Measurement Variables, Electro-mechanical Conduction Delay, and CV-PMD_{LV} in the Control-Gr and RBBB-Gr Patients

No significant difference was found between the 2 groups for gender, LVEDD, or frequency of using medications (Table 1). However, the patients in the RBBB-Gr had a greater LVESD ($p<0.01$) and lower LVEF ($p<0.0001$) than those in the Control-Gr (Table 1). No significant difference was found between the 2 groups for TDSEPT-LAT ($p=0.33$; Table 1), however, the TDRV-SEPT ($p<0.0001$), TDRV-LAT ($p<0.0005$), and CV-PMD_{LV} ($p<0.0005$) were greater in the RBBB-Gr patients than in the Control-Gr patients (Table 1). In the RBBB-Gr patients with normal LV function ($n=41$; LVEF $>$ 50%; Table 2), the TDSEPT-LAT was comparable with that in the Control-Gr patients ($p=0.68$). However, the TDRV-SEPT ($p<0.0001$), TDRV-LAT ($p<0.001$), and CV-PMD_{LV} ($p<0.005$) were greater in the RBBB-Gr patients with normal LV function than in the Control-Gr patients.

Effect of LV Systolic Dysfunction on QRS Duration, Echo-cardiographic Measurements, and Electromechanical Conduction Delay in the RBBB-Gr Patients

Among the RBBB-Gr patients, the total duration of the QRS complex was greater in patients with LV systolic dysfunction ($n=8$) than in those with normal LV function (Table 2; $p<0.01$). The LVEDD and LVESD were greater in the patients with LV systolic dysfunction than in those with normal LV function (both $p<0.0001$; Table 2). There was no difference in the TDRV-SEPT ($p=0.31$) or TDRV-LAT ($p=0.25$) of the CRBBB patients with LV systolic dysfunction and that of the patients with a normal LV function (Table 2). However, there was a significant difference between the 2 groups for TDSEPT-LAT ($p<0.001$; Table 2). The CV-PMD_{LV} was also greater in the CRBBB patients with LV systolic dysfunction than in those with normal LV function ($p<0.05$; Table 2).

Fig 2. Correlation between the LVEF and the TDSEPT-LAT, TDRV-LAT, TDRV-SEPT, and CV-PMD_{LV}. See text for abbreviations.

Fig 3. Correlation between the total QRS duration and the TDSEPT-LAT, TDRV-LAT, TDRV-SEPT, and CV-PMDLV. See text for abbreviations.

Relationship of LVEF to the Conduction Delay and CV-PMDLV in the CRBBB Patients

No significant correlation was found between the LVEF and TDRV-SEPT ($p=0.13$, $r=0.22$) or TDRV-LAT ($p=0.70$, $r=0.06$) in the CRBBB patients (Fig 2). However, a weak but significant correlation was found between the LVEF and TDSEPT-LAT ($p=0.02$, $r=0.33$) and between the LVEF and CV-PMDLV ($p=0.003$, $r=0.42$; Fig 2).

Relationship of Total QRS Duration to the Conduction Delay and CV-PMDLV in the CRBBB Patients

There was no significant correlation between the duration of the QRS complex and TDRV-SEPT ($p=0.12$, $r=0.22$), TDRV-LAT ($p=0.21$, $r=0.15$), or CV-PMDLV ($p=0.44$, $r=0.11$) in any of the 49 CRBBB patients (Fig 3). However, a weak but significant positive correlation was found between the QRS duration and TDSEPT-LAT ($p=0.0002$, $r=0.51$; Fig 3).

Changes in the Measurement Variables Before and After CRT in the CRBBB Patients

Five RBBB-Gr patients with LV dysfunction and advanced HF underwent CRT-P (Figs 4,5). The QRS duration was >140 ms and the LVEF determined by echocardiography was $<35\%$ in all of them. An atrial lead was implanted in the right atrial appendage, and the RV lead was placed at the RV apex. The LV pacing lead was inserted via the transvenous approach through the coronary sinus, and placed in the left marginal or posterolateral cardiac vein.⁷ One month after receiving CRT-P, 3 patients were classified as being responders who had clinically improved to New York Heart Association functional class of I or II and in whom the LV end-systolic volume decreased by 10%. However, neither of the remaining 2 patients improved clinically nor did their LV end-systolic volume decrease by 10% (non-responders). There was no significant difference between the responders and non-responders for mean age ($p=0.51$), gender, QRS duration ($p=0.27$), LVEDD ($p=0.70$), LVESD ($p=0.73$) or LVEF ($p=0.98$) before receiving CRT-P.

No significant difference was found between the 2 groups for the TDSEPT-LAT ($p=0.18$), TDRV-LAT ($p=0.52$), or TDRV-SEPT ($p=0.47$) before receiving CRT-P. However, in the 3 responders, the TDSEPT-LAT before receiving CRT-P was greater than that in the 2 non-responders, and it decreased

Fig 4. TDSEPT-LAT (A), TDRV-LAT (B), TDRV-SEPT (C), and CV-PMDLV (D) before and after CRT in responders (closed circles) and non-responders (open circles) among selected CRBBB patients with LV systolic dysfunction. See text for abbreviations.

after CRT-P (Fig 4). The TDRV-LAT and TDRV-SEPT before and after receiving CRT-P exhibited the same tendency (Fig 4). In the 2 non-responders, the TDSEPT-LAT before receiving CRT-P was quite small, comparable with that in the Control-Gr. One non-responder also had a small TDRV-LAT and TDRV-SEPT before receiving CRT-P. The other patient had a greater TDRV-LAT and TDRV-SEPT before receiving CRT-P, but neither decreased after CRT-P. The CV-PMDLV before receiving CRT-P varied in both groups ($p=0.96$), and did not improve in 4 of 5 patients after receiving CRT-P (Fig 4).

Fig 5. Representative case of CRBBB and LV systolic dysfunction before and after CRT (53-year-old female; in dilated phase of the non-obstructive hypertrophic cardiomyopathy; New York Heart Association (NYHA) functional class III). (A) 12-lead electrocardiograms. (B) TDI before and after CRT. Before CRT, there is a significant time difference between the electromechanical delay of the LV lateral wall (LAT) and septum (SEPT), the LAT and right ventricular free wall (RV), and the RV-SEPT, indicating that there is significant intra- and inter-ventricular dyssynchrony (Upper panel). However, after CRT, the LV dyssynchrony disappears (Lower panel). The severity of the mitral regurgitation became mild (grade II to grade I), and the LV ejection fraction improved from 43% to 65%. The clinical status also improved to NYHA class I. See text for other abbreviations.

Discussion

Major Findings

From the measurements of ventricular dyssynchrony using TDI and TTI in patients with a normal QRS complex and CRBBB, the results of this study demonstrated the following findings.

(1) Although the TDRV-SEPT, TDRV-LAT, and CV-PMDLV were greater in the CRBBB-Gr patients than in the Control-Gr patients, irrespective of the LVEF in the patients with CRBBB, the TDSEPT-LAT was comparable between the CRBBB patients and control subjects.

(2) In the CRBBB patients, no significant difference in the TDRV-SEPT or TDRV-LAT was found between the patients with a normal LV function and those with LV dysfunction. However, the TDSEPT-LAT, as well as the CV-PMDLV, was significantly greater in the patients with LV dysfunction, and there was a significant correlation between the LVEF and TDSEPT-LAT and between the LVEF and CV-PMDLV.

(3) The CRBBB patients with LV dysfunction and a high TDSEPT-LAT, improved clinically shortly after receiving CRT-P.

These results indicate that the presence of LV dysfunction (LVEF <50%) may indicate a great intra-LV dyssynchrony in CRBBB patients.

QRS Width, CRBBB, LV Dyssynchrony, and CRT

Several studies have demonstrated clinical improvement in CRT recipients with advanced HF and a wide QRS complex.¹⁻⁴ A widened QRS complex on the surface ECG is the most commonly used surrogate marker for interventricular dyssynchrony;¹³ however, it is becoming clear that the QRS duration may not always predict the clinical response, and that the prevalence of LV systolic asynchrony in patients with congestive HF and a normal QRS duration is not rare.¹⁴⁻¹⁶ Therefore, the relationship between QRS duration and ventricular dyssynchrony is still controversial. A recent study indicated that the presence of an intra-LV (but not inter-V) asynchrony was an independent predictor of severe cardiac events, independent of LVEF and the QRS width.¹⁷ However, that study also demonstrated that the surface

ECG could identify patients with and without loco-regional contraction abnormalities only when the QRS duration was >140 ms, and that a QRS duration >140 ms was found to be independently related to rehospitalization because of worsening of the HF.¹⁷ In the present study, the CRBBB patients with LV systolic dysfunction had a longer QRS complex than those with normal LV function, and a significant positive correlation was found between QRS duration and TDSEPT-LAT. Therefore, QRS duration >140 ms may be a marker for the presence of TDSEPT-LAT in CRBBB patients.

As with LBBB, CRBBB has been recently identified as an important and independent predictor of mortality in patients with congestive HF.⁶ However, the majority of CRT recipients reported in previous studies have had LBBB QRS morphology,¹⁻³ and therefore the electrophysiological and mechanical rationale for using CRT in such patients has been well characterized.¹⁸⁻²⁰ In contrast there is little data for patients who present with nonspecific conduction delays or RBBB.²¹ Thus, the relationship of CRBBB to ventricular dyssynchrony and the predictors of CRT effectiveness in CRBBB patients have not been fully clarified.

In the present study, the TDRV-SEPT and TDRV-LAT were significantly greater in the CRBBB patients than in the control subjects. In the patients with RBBB, a single RV breakthrough site appeared in the septum, and its activation slowly spread toward the anterior region and continued so that the latest activated regions were the right lateral wall and the outflow tract.²¹ The mechanism of the RV activation sequence in RBBB patients is considered to be most likely a result of cell-to-cell conduction, from the septum to the anterior wall, to the lateral wall, and finally to the outflow tract.²¹ That mechanism may be responsible for the differences in the TDRV-SEPT and TDRV-LAT between the CRBBB patients and control subjects in the present study.

Among the CRBBB patients in the present study, a significant intraventricular conduction delay in the LV (TDRV-SEPT) was observed in patients with LV systolic dysfunction, which was clearly different from the patients with normal LV systolic function. In the CRBBB patients, the CV-PMDLV, which is considered to reflect LV synchrony, was also significantly greater in the patients with LV sys-

tolic dysfunction than in those with normal LV function. Schuster et al showed that even with a narrow QRS or RBBB, HF patients had a significant left-sided longitudinal asynchrony, as detected by color TDI.¹⁷ Bader et al also reported that, in the patients with HF, the degree of intra-ventricular asynchrony, detected by TDI, was a better predictor of outcome than QRS duration or the EF, and that no difference was found in the prevalence of such asynchrony in patients with RBBB or LBBB.¹⁴ Furthermore, a recent study demonstrated that LV activation spreads slowly, from the septal or anterior breakthrough site, toward the apical and lateral regions, to the posterolateral basal region and around the mitral valve, which is the latest activated zone in HF patients with either RBBB or LBBB.²¹ Thus, in CRBBB patients with advanced HF, LV activation is delayed as much as in the patients with isolated LBBB. In the present study, CRT was effective for the CRBBB patients with LV dyssynchrony. The previous studies and our results indicate that LV conduction delay may be present in CRBBB patients with LV systolic dysfunction, and that CRT may be effective in those patients because more coordinated ventricular contractions are responsible for the beneficial effects of CRT. We believe that patients with RBBB and LBBB have a similar response to CRT when they have significant LV dyssynchrony.

Role of TDI and TTI in Evaluating Ventricular Dyssynchrony

Among the noninvasive examination techniques, echocardiography, including TDI, is simple, easy and ideal for evaluating regional wall motion.^{7,8,14,22,23} TDI also enables accurate assessment of the regional timing of mechanical events relative to the phase of the cardiac cycle, and an intra- or inter-ventricular conduction delay can be easily measured by placing 2 sample volumes at the basal portion of the ventricle.^{7,8} In the present study, using TTI, we also tested the utility of the CV-PMD_{LV} as an index for evaluating LV dyssynchrony. Strain and strain rate imaging are useful for evaluating ventricular synchrony and cardiac performance.^{7,22} However, these modalities are noise sensitive, especially in the setting of global LV dysfunction. TTI is not noise-sensitive, and has sufficient reproducibility and accuracy, with good spatial and temporal resolution and low inter- and intraobserver variabilities.^{9,10} In the present study, we could evaluate ventricular dyssynchrony using TDI and TTI, as well as the relationships between CRBBB and ventricular dyssynchrony and LV cardiac function. Our results were almost identical to those obtained from a study using an electro-anatomical mapping system.²¹ Therefore, we think that measurement using TDI and TTI is a useful and reliable tool for evaluating the ventricular synchrony.

Study Limitations

First, this study had a relatively small sample size. In particular, the effect of CRT was examined in only 5 patients with CRBBB and LV dysfunction. The results obtained in those patients for TDI and TTI might indicate that a greater TDRV-LAT and TDRV-SEPT predict the effectiveness of CRT. Inversely, CV-PMD_{LV} might be useless as a predictor for responders after receiving CRT. However, our sample size was too small to reach those conclusions. Therefore, future studies with a larger number of CRBBB patients receiving CRT are needed to clarify these points. Second, patients with both ischemic and non-ischemic CHF were included and therefore some patients with ischemic CHF would have

had regional wall motion abnormalities that might affect mechanical dyssynchrony.

Conclusions

These results show that the presence of RBBB and LV dysfunction suggests LV dyssynchrony and heterogeneous mechanical dysfunction. A high intraventricular conduction delay in the LV may predict clinical improvement in patients with a CRBBB QRS morphology and LV dysfunction undergoing CRT.

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