Strain Dyssynchrony Index Correlates With Improvement in Left Ventricular Volume After Cardiac Resynchronization Therapy Better Than Tissue Velocity Dyssynchrony Indexes

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Background—Various dyssynchrony indexes derived from tissue velocity and strain imaging have been proposed to predict the effectiveness of cardiac resynchronization therapy (CRT). We sought to compare the effect of CRT on dyssynchrony indexes derived by tissue velocity and strain and to determine which baseline intraventricular dyssynchrony parameters correlate with improvement in left ventricular volume after CRT.

Methods and Results—Echocardiography with tissue Doppler imaging was performed in 45 patients with systolic heart failure at baseline, 1 day after CRT, and a median of 6 months after CRT. We calculated septal–lateral delay and anteroseptal–posterior delay and standard deviation of time to peak systolic velocity in the 12 basal and mid-left ventricular segments (Tv-SD). The standard deviation for time to peak strain in the 12 basal and mid-left ventricular segments (Te-SD) was calculated as a strain-derived dyssynchrony index. None of the tissue velocity–derived dyssynchrony indexes improved after CRT (septal–lateral delay, \(P = 0.39\); anteroseptal–posterior delay, \(P = 0.46\); Tv-SD, \(P = 0.30\)), whereas Te-SD decreased significantly after CRT (\(P < 0.001\)). Improvement in Te-SD 1 day after CRT correlated with the reduction in end-systolic volume at follow-up (\(r = 0.66; P < 0.001\)). Baseline Te-SD demonstrated significant correlation with the reduction of end-systolic volume at follow-up (\(r = 0.57; P < 0.001\)); however, baseline tissue velocity–derived dyssynchrony indexes failed to predict the effect of CRT.

Conclusions—The strain-derived dyssynchrony index is a better measurement than the tissue velocity dyssynchrony index for monitoring changes in mechanical dyssynchrony after CRT and for predicting reduction in left ventricular volume after CRT. (Circ Cardiovasc Imaging. 2008;1:14-22.)

Key Words: resynchronization, cardiac ■ cardiomyopathy ■ dyssynchrony ■ echocardiography ■ pacing

Cardiac resynchronization therapy (CRT) improves the functional status and mortality rate of patients with advanced systolic heart failure and prolonged QRS.1–4 However, >30% of patients who meet these criteria do not demonstrate improvement after CRT.2 Several studies have reported that mechanical dyssynchrony evaluated by timing of the peak systolic tissue velocity with tissue Doppler imaging (TDI) predicts the response to CRT.5–10 A potential limitation of using tissue velocity for timing mechanical events is that it can be affected by a passive translational motion or a tethering effect, especially in patients with ischemic cardiomyopathy or reduced longitudinal motion. Strain imaging is less affected by these factors. Therefore, it can be a more precise measure for regional contraction timing and for prediction of the effect of CRT.11–13

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Because correction of intraventricular dyssynchrony appears to be a main therapeutic mechanism of CRT, reliable parameters of intraventricular dyssynchrony should also improve after CRT. However, it has been our clinical observation that a substantial subset of patients without improvement in mechanical dyssynchrony by TDI achieve improvement in left ventricular (LV) volume. The purpose of this study, therefore, was (1) to describe the effect of CRT on intraventricular dyssynchrony indexes derived by tissue velocity and strain and (2) to determine which baseline intraventricular dyssynchrony parameter correlates best with improvement in LV volume at follow-up.
Methods

Patients
From October 2004 to July 2006, 97 patients scheduled for CRT at Mayo Clinic, Rochester, Minn, were approached for this study. Ten patients declined to participate, 15 were excluded because of atrial fibrillation or frequent ectopic beats, and 4 did not meet the current criteria for CRT (ejection fraction [EF] < 35%, New York Heart Association class III or IV heart failure symptoms, QRS > 120 ms). Among 68 patients eligible for the study, 19 did not return for a follow-up, and 1 underwent heart transplantation before a follow-up study. The remaining 48 patients were examined. Tissue velocity timing analysis was feasible in all 48 patients; however, strain analysis was not possible in 3 patients (6%) because of unreliable signals. These 3 patients were excluded, leaving 45 patients (mean ± SD age, 70 ± 9 years) for this study. All 45 were classified as New York Heart Association functional class III (39 patients) or IV (6 patients), with a mean EF of 25 ± 7%. All were in sinus rhythm or regular ventricular paced rhythm. All had prolonged QRS complex > 120 ms (mean, 171 ± 31 ms) in one of the following forms: left bundle-branch block (18 patients), ventricular pacemaker (17 patients), right bundle-branch block (5 patients), or unclassified intraventricular conduction delay (5 patients). Causes of heart failure were nonischemic dilated cardiomyopathy (22 patients; 49%) and ischemic heart disease (23 patients; 51%).

All 45 patients received a biventricular pacemaker with a defibrillator. The tip of the LV lead was located at the anterior (7 patients), anterolateral (28 patients), or the inferolateral wall (10 patients). The tip of the right ventricular lead was located at the right ventricular apex in all patients. The sensed and paced atrioventricular interval was empirically set at 100 and 130 ms, respectively.

Echocardiography
Standard echocardiography with TDI was performed with a cardiovascular ultrasound system (Vivid 7, GE Medical Systems, Milwaukeee, Wis) at baseline, 1 day after biventricular pacemaker implantation, and > 3 months after biventricular pacemaker implantation. LV end-systolic volume (ESV), end-diastolic volume, and EF were measured by the biplane Simpson method. Improvement in LV volume was predefined as a > 10% reduction in ESV at follow-up. Interventricular dyssynchrony was defined as the absolute timing difference between aortic prejection time and pulmonary prejection time measured from the pulsed-wave Doppler of aortic flow and pulmonary flow.

Color-coded TDI was performed in 3 standard apical views. Images of individual LV walls were acquired with the narrowest sector size possible for strain measurements. Longitudinal tissue velocity timing was measured in 3 apical views, and strain timing was measured from the images of individual LV walls, with the onset of the QRS used as a reference point. The region of interest was defined as a 6 × 6-mm area with a circular shape for tissue velocity measurements and as a 5 × 10-mm area with an oval shape for strain measurement. The region of interest was tracked semiautomatically by anchoring to the myocardium at end diastole and end systole and after early filling in strain analysis. The timing of aortic valve opening and closure was determined from pulsed-wave Doppler of the LV outflow tract and was superimposed on the tissue velocity and strain waveforms. All measurements were performed in 3 representative cardiac cycles and averaged. The time to peak mechanical events was defined as the following:

1. Time to peak systolic tissue velocity during the ejection period: the interval from onset of Q waves to the maximum positive velocity during the ejection period. If no positive velocity was observed during the ejection period, the corresponding segment was excluded from dyssynchrony index calculation. If there was beat-to-beat variability in the dominance of multiple peaks during the ejection period, the first peak was chosen.

2. Time to peak systolic velocity, including the postejection period: the interval from onset of the Q wave to the maximum positive velocity, including the period after aortic valve closure.

3. Time to peak strain: the interval from onset of the Q wave to peak negative strain throughout cardiac cycle. If negative strain was not identified, the segment was excluded. If there were multiple distinct peaks, the largest peak during systole was taken as the peak systolic velocity. If multiple peaks had similar amplitude or showed beat-to-beat variability, the earliest one was taken.

The following dyssynchrony indexes were calculated on the basis of previous reports:

1. Septal–lateral (S-L) delay: absolute difference in time to peak systolic velocity during the ejection period between the basal interseptal and basal anterolateral segments.

2. Anteroseptal–posterolateral (AS-P) delay: absolute difference in time to peak systolic velocity, including the postejection period, between basal anteroseptal and basal inferolateral segments.

3. The standard deviation of time to peak systolic velocity in the 12 basal and mid-level LV segments during the ejection period (Tv-SD).10

The standard deviation of time to peak strain in the 12 basal and mid-level LV segments (Te-SD) was also calculated in addition to the previously reported dyssynchrony indexes, and it served as a strain-derived dyssynchrony index.13

Statistical Analysis
Data are expressed as mean ± SD for continuous variables and as absolute frequencies or relative percentages for categorical variables. We determined the sample size to test the following: (1) improvement of Kv-SD after CRT and (2) correlation between the baseline Kv-SD and the reduction in ESV after CRT. According to previously published data, the sample size required to detect 8.4 ms of improvement in Kv-SD under power of 0.8 and α = 0.016 (with consideration of the Bonferroni correction for multiple comparisons among 3 different time points) was 21. To detect this difference even in the responder subgroup, we determined that 35 patients would be required for our study, assuming that the nonresponder rate is 40%. For analysis of the correlation between the baseline Kv-SD and of the reduction in ESV after CRT, the minimal sample required to detect the r = 0.45 in α of 0.05 was 36. Thus, we sought to examine at least 36 patients in our study.

Repeated-measures ANOVA was performed followed by the paired t test with Bonferroni correction to examine change in the parameters over time. Because AS-P delay and interventricular dyssynchrony were not normally distributed, we performed repeated-measures ANOVA using a log transformation of these 2 parameters. Because the conclusions were the same (data not shown), results for the untransformed variables were presented for simplicity and ease of interpretation. The relationship between 2 continuous variables was evaluated with linear regression analysis with Pearson’s correlation coefficient. One-way ANOVA was used for the comparison of different LV lead locations. Interobserver and intraobserver variabili- ty was assessed as the difference between measurements expressed as a percentage of the mean ± SD for tissue velocity, strain, and ESV in 16 randomly selected studies. The aforementioned statistical computations were performed with JMP statistical software, version 6 (SAS Institute, Inc, Cary, NC).

The authors had full access to and take responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results
Effects of CRT on LV Volume and EF
A significant reduction in LV volume was achieved during the follow-up (median, 6 months; range, 3 to 12 months)
The end-diastolic volume decreased from 221 ± 79 mL to 189 ± 77 mL (P < 0.001) and ESV from 168 ± 70 mL to 134 ± 73 mL (P < 0.001), resulting in an increase in EF from 25 ± 7% to 32 ± 11% (P < 0.001). A reduction of >10% in ESV was observed in 27 patients (60%) at follow-up; these patients were defined as the responder group. The reduction of ESV at follow-up did not differ among different LV lead locations (anterior, anterolateral, or inferolateral) (P = 0.33). Cardiac output also improved acutely after CRT without further improvement at follow-up.

Effect of CRT on Intraventricular Mechanical Dyssynchrony Indexes
Table 1 and Figure 1 show the change in dyssynchrony indexes after CRT. The tissue velocity–derived dyssynchrony indexes did not change over time (S-L delay, P = 0.39; AS-P delay, P = 0.46; Tvs-SD, P = 0.30 for overall test). Even in the responder subgroup of 27 patients, there was no significant change in tissue velocity–derived dyssynchrony indexes after CRT (S-L delay, P = 0.23; AS-P delay, P = 0.71; Tvs-SD, P = 0.07 for overall comparison).

Figure 1. Changes in dyssynchrony indexes after CRT. Bars in graphs indicate standard deviations. Responders and nonresponders were analyzed separately. *P < 0.01, †P < 0.05.
In contrast, Te-SD changed significantly after CRT ($P<0.001$ for overall test). Te-SD decreased significantly at 1 day after CRT (114±46 versus 80±32 ms; $P<0.001$) and remained similar during the follow-up period (87±32 ms; $P=0.02$ versus baseline, $P=0.26$ versus 1 day after CRT). A significant reduction in Te-SD was observed in responders (132±41 ms at baseline versus 79±30 ms at 1 day, $P<0.001$; 83±34 ms at follow-up, $P<0.001$ versus baseline) but not in nonresponders (89±42 ms at baseline versus 81±35 ms at 1 day, $P=0.43$; 94±27 ms at follow-up, $P=0.20$ versus baseline).

The correlations between improvement of dyssynchrony indexes acutely after CRT and the percent reduction in ESV at follow-up are shown in Table 2 and Figure 2. There was a significant linear correlation between the improvement in Te-SD at 1 day after CRT and the reduction in ESV at follow-up ($r=0.66$, $P<0.001$), whereas no correlation was found between the changes in tissue velocity–derived dyssynchrony indexes and the reduction in ESV. The correlation between ΔTe-SD and the reduction in ESV remained significant when linear regression was restricted to the ischemic

### Table 2. Correlation Between Improvement in Intraventricular Dyssynchrony Parameters and Reduction in ESV

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Overall (n=45)</th>
<th>Nonischemic Group (n=22)</th>
<th>Ischemic Group (n=23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ΔS-L delay, ms</td>
<td>$r=-0.27$</td>
<td>$r=0.15$</td>
<td>$r=-0.27$</td>
</tr>
<tr>
<td>ΔAS-P delay, ms</td>
<td>$P=0.07$</td>
<td>$P=0.51$</td>
<td>$P=0.22$</td>
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<tr>
<td>ΔTv-SD, ms</td>
<td>$r=-0.07$</td>
<td>$r=-0.12$</td>
<td>$r=-0.38$</td>
</tr>
<tr>
<td>ΔTe-SD, ms</td>
<td>$P&lt;0.001$</td>
<td>$P=0.60$</td>
<td>$P=0.07$</td>
</tr>
</tbody>
</table>

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![Figure 2](http://circimaging.ahajournals.org/Downloaded from http://circimaging.ahajournals.org)
subgroup (n=23; r=0.76, P<0.001). An example of tissue velocity and strain waveform is shown in Figure 3.

Correlation Between Baseline Dyssynchrony and Effect of CRT

Tissue velocity–derived dyssynchrony indexes at baseline did not correlate significantly with the percent reduction in ESV at follow-up (S-L delay, r=0.11 [P=0.48]; AS-P delay, r=0.14 [P=0.36]; Tv-SD, r=0.03 [P=0.84]), whereas Te-SD showed significant correlation at follow-up (r=0.57; P<0.001) (Figure 4).

Interobserver and Intraobserver Variability

Interobserver variability for time to peak systolic velocity and time to peak strain was 11±14% and 13±15%, respectively, in an analysis of 192 segments from 16 randomly selected studies. Intraobserver variability for each measurement was 8±13% and 8±11%, respectively. Interobserver variability for calculated Tv-SD and Te-SD was 8±7% and 11±7%, respectively, and intraobserver variability for Tv-SD and Te-SD was 7±6% and 11±7%, respectively. Interobserver and intraobserver variability for ESV was 5±4% and 4±4%, respectively.

Discussion

Our findings demonstrated a significant improvement in dyssynchrony index measured by time to peak strain but not in dyssynchrony indexes measured by tissue velocity within 24 hours after CRT. Moreover, improvement in ESV correlated significantly with both the baseline dyssynchrony index and the improvement in the dyssynchrony index measured by strain but not with tissue velocity–derived dyssynchrony indexes.

Evaluation of Dyssynchrony by Tissue Velocity

Bax et al. showed that the S-L delay of time to peak systolic velocity improved 3 months after CRT and that baseline S-L delay predicted the reverse remodeling after CRT. Yu et al demonstrated that the Tv-SD improved acutely and at 3 months after CRT and that this index is also predictive of reverse remodeling after CRT. In these studies, dyssynchrony indexes were calculated by using the time to peak systolic velocity only within the ejection period. Gorcsan and associates reported that the AS-P delay, including the postejction period, predicted the acute effect of CRT. Contrary to these studies, conflicting results have been reported recently by other investigators, suggesting that the S-L delay based on pulsed-wave tissue velocity has limited predictive value for the effect of CRT. Most recently, the Predictors of Response to Cardiac Resynchronization Therapy (PROSPECT) trial, a prospective multicenter trial, demonstrated that dyssynchrony indexes measured by tissue velocity were not reliable in predicting clinical or LV volume response to CRT.

Our results also failed to demonstrate either an improvement of tissue velocity–derived dyssynchrony indexes or a correlation with reverse remodeling after CRT, in agreement with studies showing the limited predictive value of S-L delay.

One possible reason for different results is a frequent difficulty in identifying the peak systolic velocity. Two distinct positive peaks are common during the ejection period, especially along the LV free wall, even in normal subjects. In addition, beat-to-beat variability in amplitude of the peak, which impedes identification of the dominant peak, occurs frequently even under careful breath-hold, although there is no marked difference in onset or termination of systolic velocity. As in previously published reports, we defined the largest positive peak during the ejection period as the peak systolic velocity for the S-L delay and the Tv-SD, and we defined the largest positive peak, including the postejction period, for the AS-P delay. However, a slight difference in the magnitude of peak velocity can change the identification of peak systolic velocity, which may have contributed to the different results we found, although we carefully followed the method previously described in the literature. This observation also indicates a major limitation of the application of tissue velocity to quantify the mechanical dyssynchrony in clinical practice.

Advantage of Strain Imaging for Evaluation of Dyssynchrony

We have demonstrated that the time to peak strain is more robust than tissue velocity timing analysis for evaluation of improvement in LV dyssynchrony with CRT and for prediction of the beneficial effects of CRT. This finding requires confirmation by other investigators but appears to be compelling for 2 reasons. First, strain imaging is free from the passive effect of tethering and translational motion. To prove this, we should have compared the dyssynchrony index derived from the timing of peak systolic strain rate with the ones from tissue velocity during ejection period. We found that the timing measurement of systolic strain rate was difficult because it was more sensitive to noise than strain, as described before, and it was not as stable as the timing of peak strain. In addition, strain rate during the ejection period is often very low or positive, especially in the septal wall, in patients with left bundle-branch block. In such cases, more prominent presystolic or postsystolic shortening waves were observed, which led us to use the timing of strain instead of strain rate, including presystolic and postsystolic phases.

Second, peak negative strain during the entire cardiac cycle was used in our study. The time to peak tissue velocity only during the ejection period may not represent the actual severity of dyssynchrony because it fails to recognize contraction during isovolumic periods or during diastole. Intraventricular dyssynchrony in left bundle-branch block is characterized by early septal contraction, which often peaks during the isovolumic contraction period with associated lateral prestretching. This finding has been demonstrated with the use of M-mode echocardiography, cineangiography, magnetic resonance imaging tagging, and tissue Doppler–derived strain imaging in left bundle-branch block and right ventricular apical pacing. Forces generated by the early activated fibers are dissipated in generating sufficient energy to open the aortic valve and in prestretching the not-yet-activated fibers, which leads to a waste of energy.
Figure 3. A, An example of tissue velocity waveforms in 12 mid-level and basal segments in a responder with left bundle-branch block. Peak velocity during the ejection period is identified by a cluster of 4 arrowheads for baseline (3 top panels) and for 1 day after CRT (3 bottom panels). In this patient, the baseline tissue velocity measurement showed S-L delay of 64 ms, AS-P delay of 51 ms, and Tv-SD of 45 ms. The day after CRT, each dyssynchrony index increased (S-L delay, 110 ms; AS-P delay, 99 ms; Tv-SD, 71 ms) despite a reduction in ESV. AVO indicates aortic valve opening; AVC, aortic valve closure. B, Strain waveforms of the same patient in the 12 mid-level and basal segments at baseline. Basal inferoseptal (top left) and mid anteroseptal (top middle) segments show substantial early shortening (*) before the aortic valve opens, whereas anterolateral (bottom left) and inferolateral (bottom middle) segments demonstrate delayed shortening accompanied by prestretching (arrow) in the early systolic phase. Peak strain in each segment is indicated by arrowhead. C, Strain waveforms of the same patient 1 day after CRT. The peak strain (*) of the basal inferoseptal wall (top left) and the mid anteroseptal wall (top middle) is delayed compared with baseline. Note that the amount of prestretching (arrow) in the anterolateral wall (bottom left) and in the inferolateral wall (bottom middle) decreased compared with baseline. Tr-SD diminished from 125 ms at baseline to 67 ms at 1 day after CRT.
Positive velocity during the ejection period does not necessarily reflect the early activation of the septal wall if an early activation induces premature contraction of the septum during the isovolumic contraction period. Exclusion of the isovolumic contraction period may result in incorrectly viewing the second peak during the ejection period as the first activating peak.

In addition to diminished premature septal contraction, a reduction in postsystolic shortening may play an important role in the observed improvement after CRT. However, the AS-P delay, which included the postejiction wave, failed to predict the effect of CRT. Sogaard et al reported that the extent of postsystolic displacement by tissue tracking was useful for predicting CRT effect. They used strain imaging to identify the true postsystolic deformation from passive motion. If the postsystolic motion is detected solely by tissue velocity, it is likely that the velocity identifies the peak of the postsystolic passive motion without contraction rather than true postsystolic myocardial deformation.

**Prediction of the Effect of CRT by Assessment of Strain-Derived Dyssynchrony**

Which parameter, tissue velocity or strain, is better clinically to identify the patients who benefit most from CRT? Three studies concluded that the dyssynchrony index derived by strain did not predict CRT effect and that tissue velocity–derived dyssynchrony indexes were superior to strain-derived dyssynchrony for prediction of reverse remodeling. The technical aspects of imaging may partly contribute to these discordant results. We imaged each cardiac wall individually, with the narrowest possible sector size, to minimize the Doppler angle and to increase lateral resolution with the highest obtainable frame rate. In patients with a dilated heart, it is often difficult to obtain an optimal angle between the ultrasound beam and the longitudinal orientation of individual LV segments when the heart is scanned with a wide sector for apical views. Therefore, if the wider sector angles were used in the previous study, it would have affected the strain measurements more than the tissue velocity measurements.

The favorable correlation between the baseline strain-derived dyssynchrony and the benefit from CRT in our study is concordant with several other studies. Mele et al demonstrated that the strain-derived dyssynchrony index from 12 segments (as in our study) was a better predictor of positive response to CRT than septal–posterior motion delay by M-mode echocardiography. Other strain-derived dyssynchrony indexes, such as the AS-P timing delay in peak radial strain and the maximum timing difference in time to peak

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**Figure 4.** Correlation between baseline intraventricular dyssynchrony indexes and the percent reduction of ESV at follow-up.
radial strain in the mid 6 segments by 2-dimensional speckle tracking, also have been reported to predict the effect of CRT.\textsuperscript{11,12} Together, these results suggest that strain timing analysis is a promising means to evaluate baseline dyssynchrony, predict the response, and assess improvement in dyssynchrony after CRT. The recently completed PROSPECT trial did not include strain analysis, and prospective clinical trials will be necessary to test the clinical utility of the strain dyssynchrony index. Although we demonstrated that strain-derived dyssynchrony correlates better with LV volume changes after CRT than does tissue velocity–derived dyssynchrony, the predictive value of strain-derived dyssynchrony needs to be validated in a larger prospective study. Three-dimensional echocardiography is another potential modality for quantification of dyssynchrony and prediction of the effect of CRT.\textsuperscript{40,41} Several different mechanisms may be involved in CRT, and the best patient selection method may require several combined and/or stepwise approaches other than the single intraventricular dyssynchrony index.

Limitations

Because we measured timing intervals in individual walls separately, the variation of the R-R interval might have caused variability in the measured strain time intervals. Although care was taken to measure the strain in the region in which Doppler angle was acceptable, some included segments may not have had a perfect angle for the correct estimation of longitudinal strain. Other clinical parameters, such as exercise capacity, were not assessed in our study, and thus the relationships between clinical improvement and the dyssynchrony indexes were not addressed. Our sample size was small; therefore, further study will be needed to evaluate the predictive value of dyssynchrony indexes for the effect of CRT.

Conclusions

Significant improvement in a mechanical dyssynchrony index measured by peak longitudinal strain timing was found in parallel with the reduction in LV volume after CRT. Mechanical dyssynchrony measured by time to peak systolic velocity did not correlate with changes in LV volume after CRT. The reduction in LV volume after CRT correlated better with the baseline dyssynchrony index measured by time to peak strain than with the tissue velocity–derived dyssynchrony indexes.

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Disclosures

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