Comparison of Echocardiographic Dyssynchrony Assessment by Tissue Velocity and Strain Imaging in Subjects With or Without Systolic Dysfunction and With or Without Left Bundle-Branch Block

Chinami Miyazaki, MD; Brian D. Powell, MD; Charles J. Bruce, MD; Raul E. Espinosa, MD; Margaret M. Redfield, MD; Fletcher A. Miller, MD; David L. Hayes, MD; Yong-Mei Cha, MD; Jae K. Oh, MD

Background—Several echocardiographic dyssynchrony indexes have been proposed to identify responders to cardiac resynchronization therapy using tissue velocity and strain. The present study aimed to compare tissue velocity–derived and strain-derived dyssynchrony indexes in patients with or without systolic dysfunction and left bundle-branch block.

Methods and Results—Tissue Doppler imaging was performed in 120 subjects divided into 4 groups: group 1 (n=40), normal subjects; group 2 (n=20), normal left ventricular ejection fraction and left bundle-branch block; group 3 (n=20), left ventricular ejection fraction <35% and normal conduction; and group 4 (n=40), left ventricular ejection fraction <35% and left bundle-branch block. Dyssynchrony indexes based on time to peak tissue velocity (septal-lateral delay, anteroseptal-posterior delay, and SD in time to peak systolic velocity in the 12 left ventricular segments) and strain (SD of time to peak strain in 12 segments) were measured. The SD in time to peak systolic velocity in the 12 left ventricular segments was greater in group 4 (54 ms; 25th and 75th percentiles, 46 to 64 ms) than group 1 (44 ms; 25th and 75th percentiles, 28 to 53 ms; P=0.006), but there was a considerable overlap of all tissue velocity–derived indexes among 4 groups, with 40% to 68% of group 1 having values proposed for predicting the responders of cardiac resynchronization therapy. The SD of time to peak strain in 12 segments distinguished these groups with much less overlap (P<0.01 for all pairwise comparisons).

Conclusions—A substantial proportion of normal subjects have tissue velocity–derived dyssynchrony indexes higher than the cutoff value proposed for predicting beneficial effect of cardiac resynchronization therapy. Strain-derived timing index appears to be more specific for dyssynchrony in patients with systolic dysfunction and left bundle-branch block. Identifying an optimal tissue velocity– or strain-derived dyssynchrony index requires a large prospective clinical trial.

Key Words: bundle-branch block ■ conduction ■ dyssynchrony ■ echocardiography ■ heart failure

Cardiac resynchronization therapy (CRT) improves symptoms and survival of patients with advanced systolic heart failure1–3; however, up to 30% of patients do not improve after CRT when selected by current recommended criteria.2,3 To improve patient selection, many studies have quantified the magnitude of intraventricular mechanical dysynchrony.4–9 Systolic velocity timing derived from tissue Doppler imaging has been used most frequently, and several indexes have been reported to predict positive responses to CRT.10–17 Others showed that dyssynchrony indexes based on time to peak strain predict a favorable effect of CRT.18–20 However, no study has compared tissue velocity dyssynchrony indexes with strain indexes in various populations. Moreover, whether the various cutoff values of echocardiographic dyssynchrony indexes associated with left ventricular (LV) reverse remodeling after CRT are specific for the patients with decreased LV systolic function and/or conduction delay is not well known.

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Therefore, the aims of our study were to determine the prevalence of intraventricular dyssynchrony based on previously reported cutoff values measured with peak systolic tissue velocity and peak negative longitudinal strain timing in healthy control subjects and to compare the intraventricular dyssynchrony measured by strain parameters with that measured by tissue velocity in subjects divided into 4 groups.
Figure 1. Definition of time of peaks in tissue velocity and strain imaging. Left, Tissue velocity waveform. The maximal positive peak located between the timing of aortic valve opening (AVO) and closure (AVC) was defined as the peak systolic velocity.1 Right, Strain waveform. The maximal peak throughout the cardiac cycle was defined as the peak strain.2 Time from onset of the Q wave on the ECG to each peak was measured as time to peak tissue velocity and strain.

Methods

Subject Population

A total of 126 patients were examined. Tissue velocity timing analysis was feasible in all patients. However, strain timing analysis was not possible in 6 patients (5%) because of an unreliable signal. Thus, 120 patients were included in the final analysis consisting of 4 groups: group 1, 40 asymptomatic subjects with normal LVEF (>50%) and QRS <120 ms; group 2, 20 subjects with normal LVEF and LBBB; group 3, 20 subjects with decreased LVEF (<35%) and normal conduction; and group 4, 40 patients with decreased LVEF and LBBB. Patients with right bundle-branch block, nonspecific intraventricular conduction delay, ventricular pacing, or atrial fibrillation were excluded. The study was approved by the Institutional Review Board of the Mayo Foundation, and all patients provided verbal consent.

Echocardiography Examination

A comprehensive 2-dimensional and Doppler echocardiography was performed with commercially available ultrasound equipment. LV volumes and LVEF were calculated by the biplane Simpson method. Color-coded tissue Doppler imaging was acquired from 3 apical views with Vivid 7 (GE Medical Systems, Milwaukee, Wis). Images of individual walls with narrow sectors and the shallowest depth were obtained to minimize the angle between the ultrasound beam and the longitudinal axis of the cardiac wall with the highest possible frame rate (226±36 frames per second). Gain settings and pulse repetition frequency were adjusted to optimize color saturation and to avoid aliasing.

Intraventricular Dyssynchrony by Tissue Velocity and Strain Imaging

Longitudinal tissue velocity and strain timing analyses were performed from individual LV walls with QRS onset as the reference point of timing analysis. The region of interest was determined as a 6×6-mm circle for tissue velocity and a 6×12-mm oval for strain. The region of interest was tracked according to myocardial motion at end diastole, end systole, and early diastole in the strain measurement. It was not tracked for tissue velocity timing analysis because our pilot study in 13 subjects showed a good correlation between the fixed region of interest method and the tracked method (r=0.94, P<0.001) in time to peak systolic velocity measurements. Variability between 2 repeated measurements in the fixed region of interest method was 4.7±6.9% and in the tracked region of interest method was 6.7±10.8%.

Regional myocardial strain rate was estimated over a computation distance of 12 mm. Strain was calculated as a time integral of strain rate in which the integration starting point was adjusted to the onset of the QRS. The timing of aortic valve opening and closing was superimposed on the waveform analysis based on the time of aortic flow from the pulsed-wave Doppler signal in the apical long-axis view. All analyses were performed offline with Echopac PC (version 6.0.0, GE Medical Systems).

The following 3 timing intervals reported in the literature were measured (Figure 1). The first was time to peak systolic velocity, the interval from the onset of QRS to the maximum positive velocity during the ejection period. The velocities in the isovolumic contraction and relaxation periods were not used. If a positive velocity was not observed, the segment was excluded from the calculation. If there were multiple peaks in ejection period with the same velocity, the earliest peak was chosen.22 The second, time to peak systolic velocity, including the post-ejection period, was the interval from onset of the QRS to the maximum positive velocity, including the period after aortic valve closure.14 The third was time to peak strain, the interval from the onset of QRS to peak negative strain throughout the cardiac cycle, including post-systolic shortening. If negative strain was not identified, the segment was excluded from the calculation.

The following dyssynchrony indexes were calculated as previously reported (the cutoff value to predict positive response to CRT is shown in parentheses): (1) septal/inferior-lateral/anterior delay (S-L delay) (65 ms), the longer time difference of peak systolic tissue velocities either between basal septal and lateral segments from the apical 4-chamber view or between the basal anterior and inferior segments from the apical 2-chamber view13; (2) anteroseptal-posterior delay (AS-P delay) (65 ms), the absolute difference in time to peak systolic velocity, including the post-ejection period, between the basal inferolateral and basal anteroseptal segments14; (3) the SD in time to peak systolic velocity in the 12 basal and mid segments (Tv-SD) (34.4 ms)14; and (4) the SD in time to peak strain (Te-SD) (60 ms) among 12 basal and mid segments as a strain-derived dyssynchrony index.20

Of the total 1440 segments in 120 patients, 14 segments did not show a positive peak during the ejection period in tissue velocity analysis; thus, these segments were excluded from calculations of S-L delay and Tv-SD. Positive peaks, including the post-ejection period in the antero-septal and inferolateral walls, were measured in all 120 patients. Of the 1440 segments, 11 did not show negative strain, and these segments were excluded from calculations of Te-SD.
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Table 1. Comparison of Characteristics of Patients in the 4 Study Groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (Normal)</th>
<th>Group 2 (LBBB, Normal LVEF)</th>
<th>Group 3 (LVEF &lt;35%, Normal QRS)</th>
<th>Group 4 (LVEF &lt;35%, LBBB)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, n</td>
<td>40</td>
<td>20</td>
<td>20</td>
<td>40</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>47±15</td>
<td>69±8†</td>
<td>66±11†</td>
<td>64±12†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Male sex, n (%)</td>
<td>24 (60)</td>
<td>11 (55)</td>
<td>16 (80)</td>
<td>23 (58)</td>
<td>0.28</td>
</tr>
<tr>
<td>NYHA class I/II/III/IV, %</td>
<td>6/4/7/3 (30/20/35/15)</td>
<td>3/11/22/4 (7.5/27.5/55/10)</td>
<td>0.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medications, n (%)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>β-Blocker</td>
<td>· · ·</td>
<td>· · ·</td>
<td>18 (90)</td>
<td>36 (90)</td>
<td>1.0</td>
</tr>
<tr>
<td>ACE-I or ARB</td>
<td>· · ·</td>
<td>· · ·</td>
<td>19 (95)</td>
<td>32 (80)</td>
<td>0.10</td>
</tr>
<tr>
<td>Frosemide</td>
<td>· · ·</td>
<td>· · ·</td>
<td>13 (65)</td>
<td>23 (58)</td>
<td>0.57</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>· · ·</td>
<td>· · ·</td>
<td>5 (25)</td>
<td>13 (33)</td>
<td>0.55</td>
</tr>
<tr>
<td>Digoxin</td>
<td>· · ·</td>
<td>· · ·</td>
<td>8 (40)</td>
<td>24 (60)</td>
<td>0.14</td>
</tr>
<tr>
<td>LVEDV, mean±SD, mL</td>
<td>97±25</td>
<td>113±39</td>
<td>178±53†</td>
<td>226±83†‡§</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF, mean±SD, mL</td>
<td>38±11</td>
<td>50±18</td>
<td>135±42†</td>
<td>177±79†‡§</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LVEF, mean±SD, %</td>
<td>61±4</td>
<td>56±4†</td>
<td>24±6†</td>
<td>24±9†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>E/e’, mean±SD</td>
<td>8.7±1.7</td>
<td>12.1±4.1</td>
<td>20.5±11†</td>
<td>20.2±9.3†</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>PAP, mm Hg, mean±SD</td>
<td>21±13</td>
<td>31±9†</td>
<td>44±18†</td>
<td>35±13†§</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

NYHA indicates New York Heart Association; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; LVEDV, LV end-diastolic volume; LVEF, LV end-systolic volume; and PAP, estimated pulmonary artery pressure.

*ANOVA.
†P<0.05 vs group 1; ‡P<0.05 vs group 2; §P<0.05 vs group 3.

Table 2. Comparison of Dyssynchrony Indexes Among the 4 Groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (Normal)</th>
<th>Group 2 (LBBB, Normal LVEF)</th>
<th>Group 3 (LVEF &lt;35%, Normal QRS)</th>
<th>Group 4 (LVEF &lt;35%, LBBB)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>S-L delay, ms</td>
<td>66 (21, 88)</td>
<td>87 (52, 98)</td>
<td>87 (46, 125)</td>
<td>86 (63, 110)</td>
<td>0.046</td>
</tr>
<tr>
<td>Tv-SD, ms</td>
<td>44 (28, 53)</td>
<td>48 (43, 62)</td>
<td>57 (32, 66)</td>
<td>54 (46, 64)</td>
<td>0.008</td>
</tr>
<tr>
<td>AS-P delay, ms</td>
<td>24 (8, 91)</td>
<td>82 (33, 153)</td>
<td>115 (37, 213)</td>
<td>132 (58, 293)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Te-S delay, ms</td>
<td>46 (41, 51)</td>
<td>68 (59, 75)</td>
<td>90 (72, 106)</td>
<td>107 (94, 142)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Data are expressed as the median (first, third quartiles).
†Parameters were log transformed, and results were adjusted for age and gender with multiple regression.
‡Bonferroni-corrected P<0.05 for the following between-group comparisons, A, groups 1 and 2; B, groups 1 and 3; C, groups 1 and 4; D, groups 2 and 3; E, groups 2 and 4; and F, groups 3 and 4.
AS-P delay was significantly larger in groups 2 (82 ms; 25th and 75th percentiles, 33 and 153 ms), 3 (115 ms; 25th and 75th percentiles, 37 to 213 ms), and 4 (132 ms; 25th and 75th percentiles, 58 to 293) compared with group 1 (24 ms; 25th and 75th percentiles, 8 and 91 ms; \( P < 0.03 \) versus group 2, \( P < 0.02 \) versus group 3, \( P < 0.001 \) versus group 4). There were no significant differences among other pairs of groups in AS-P delay. Tv-SD and S-L delay demonstrated a considerable overlap among groups. AS-P delay provided a better separation of groups, yet no significant difference was found between groups 3 and 4 (\( P = 0.60 \)). Even after adjustment of age and sex, none of tissue velocity–derived parameters provided clear separation among groups.

In contrast to the considerable overlap in tissue velocity–derived indexes, a stepwise increase in Te-SD was found with the presence of systolic dysfunction and LBBB (group 1, 46 ms [25th and 75th percentiles, 41 to 51 ms]; group 2, 68 ms [25th and 75th percentiles, 59 to 75 ms]; group 3, 90 ms [25th and 75th percentiles, 72 and 106 ms]; group 4, 107 ms [25th and 75th percentiles, 94 and 142 ms]; \( P = 0.003 \) for group 3 versus 4, \( P < 0.001 \) for other pairwise comparisons). The statistically significant differences for Te-SD remained unchanged even after adjustment for age and gender. Examples of tissue velocity and strain waveforms of subjects from groups 1 and 4 are shown in Figures 3 and 4.

There was a significant correlation between all dyssynchrony indexes and the LV end-diastolic volume index (S-L delay, \( P = 0.004 \); Tv-SD, \( P = 0.002 \); AS-P delay and Te-SD, \( P < 0.001 \)). The correlation coefficient was the largest in Te-SD (S-L delay, \( r = 0.26 \); AS-P delay, \( r = 0.47 \); Tv-SD, \( r = 0.28 \); Te-SD, \( r = 0.81 \)).

Prevalence of Dyssynchrony Based on Previously Reported Cutoff Value

The median S-L delay (66 ms) and Tv-SD (44 ms) in normal subjects exceeded the reported cutoff values of positive CRT response (>65 ms for S-L delay and >34.4 ms for Tv-SD). The prevalence of intraventricular dyssynchrony based on the reported values is shown in Figure 5. Notably, 68% of group 1 had dyssynchrony on the basis of Tv-SD. For S-L delay and AS-P delay, the prevalence in group 1 was 50% and 40%, respectively. On the other hand, when the cutoff value of Te-SD >60 ms was used, only 5% of group 1 showed positive findings. Prevalence of dyssynchrony based on S-L delay and Te-SD did not differ among groups (\( \chi^2 = 4.9, P = 0.18 \) for S-L delay; \( \chi^2 = 4.3, P = 0.23 \) for Tv-SD), whereas significant difference was found in AS-P delay and Te-SD (\( \chi^2 = 10.1, P = 0.02 \) for AS-P delay; \( \chi^2 = 85, P < 0.001 \) for Te-SD).
Interobserver and Intraobserver Variabilities

Interobserver variability for time to peak systolic velocity and time to peak strain was 6\% and 6\%, respectively, in an analysis of 240 segments from 20 randomly selected patients. Intraobserver variability was 5\% and 6\%, respectively. The interobserver variability for Tv-SD and T\v-dep-SD was 10\% and 11\%, respectively, and the intraobserver variability was 10\% and 8\%.

Discussion

Our results suggest that tissue velocity–derived indexes may not represent actual intraventricular dyssynchrony because values of these indexes reported to predict a positive response to CRT in patients with heart failure also are present frequently in asymptomatic normal subjects. These indexes may falsely indicate intraventricular dyssynchrony in patients who in fact have normal synchronous contraction. In contrast, the strain-derived dyssynchrony index distinguished patients with LBBB or decreased LVEF (or both) from those with normal systolic function and normal QRS duration with minimal overlap and appears to identify patients with intraventricular dyssynchrony more reliably.

Mechanical Dyssynchrony by Tissue Velocity Timing Measurement: Previous Findings

Previously, several indexes derived from timing of peak systolic tissue velocities were proposed as predictors of CRT response. This concept has been extended to the quantification of dyssynchrony in the heart failure population with normal LVEF\(^23\) or with right ventricular pacing after atrioventricular node ablation.\(^24\) These indexes also have been reported to predict adverse events among systolic heart failure patients with normal QRS.\(^25\) Several cutoff values of differences in timing of peak systolic velocities from various segments have been derived from patients with heart failure, but these timing differences in normal subjects have not been clearly established yet. Yu et al\(^4\) reported that Tv-SD was 17.0±7.8 ms in 88 healthy volunteers. Lafitte\(^26\) reported a similar value for normal control subject (16±9 ms). Poerner et al,\(^27\) however, reported that the SD in time to peak
systolic velocity among 12 basal, mid, and apical segments was 40±27 ms in 47 normal subjects.

Recently, several reports raised a concern about dyssynchrony evaluation using time to peak systolic velocity. Soliman et al.28 showed that the basal septal-lateral delay was not able to predict the clinical improvement and reverse remodeling after CRT. Duncan et al.29 did not find significant correlation between septal-lateral delay and the improvement in cardiac output after CRT. Our experience also failed to demonstrate the improvement in tissue velocity–derived dys-synchrony indexes after CRT.30

High Prevalence of Dyssynchrony in Normal Control Subject by Tissue Velocity Timing

Our median value of Tv-SD (44 ms) in normal subjects was higher than the mean value in a previous study (17 ms) and exceeded the cutoff value proposed for the prediction of a favorable effect of CRT (34.4 ms).11 Our normal value was closer to the 40 ms reported by Poerner et al.27 Dyssynchrony measured with time to peak velocities would have classified 40% to 68% of normal subjects as having mechanical dyssynchrony if the previously proposed cutoff values are used.

A reason for dyssynchronous timing of peak velocity in normal subjects could be due to frequent double peaks in tissue velocity measurement (Figure 3), especially in the free walls.31,32 Depending on which wave is selected as the peak systolic velocity, the dyssynchrony indexes can vary considerably. Double peaks often show beat-to-beat variability in velocities. Besides, in patients with conduction delay and impaired systolic function, sometimes there may not be a distinct peak during the ejection period. According to the previously published definition,22 we defined peak systolic velocity as the maximum peak during the ejection period for S-L delay and Tv-SD consistently11,15,22,33 and included the post-ejection period only for

Figure 4. Example of tissue velocity (left) and strain (right) waveforms in a patient with systolic dysfunction and LBBB. Positive pre-ejection and post-ejection waves were prominent, and a small positive wave occurred during ejection period. According to the definition, the positive peak during ejection was measured as a systolic peak velocity in the inferoseptal and anterolateral walls. The tissue velocity dyssynchrony indexes were less than the predetermined cutoff value (S-L delay, 32 ms; Tv-SD, 29 ms). Because of the prominent post-ejection wave in the anteroseptal wall (waveform not shown), which is similar in timing as the inferoseptal segment (waveform not shown), AS-P delay was 290 ms. In the strain curve, the mid inferoseptal segment showed a significantly earlier peak (asterisk in top right) at the end of the isovolumic contraction period, and the anterolateral wall showed postsystolic shortening. Te-SD was 151 ms.

AVO indicates aortic valve opening; AVC, aortic valve closure.
measuring AS-P delay. In such definitions of peaks, a slight difference in peak velocity can change the identification of peaks and time to peak systolic velocity markedly. Therefore, considerable variations in measurements may arise, depending on which peak is selected. However, our intraobserver and interobserver variabilities of the dyssynchrony indexes were acceptably low, indicating that the peaks were identified in a reproducible manner according to the predetermined definitions. Identification of a dominant systolic peak was reproducible even with double peaks, especially in the normal group, as long as we followed the definition of systolic peak as described in the method. Most recently, the Resynchronization Therapy in Normal QRS (RethinQ) study failed to demonstrate the benefit of CRT in patients with narrow QRS and positive dyssynchrony of S-L delay by tissue velocity imaging. This study suggests that tissue velocity–derived dyssynchrony may not represent clinically important mechanical dyssynchrony.

**Dyssynchrony Measurement by Strain**

The strain-derived dyssynchrony index (T-SD) was significantly different among the groups with and without reduced LVEF or LBBB with less overlap compared with tissue velocity–derived indexes. This finding suggests that the strain-derived timing interval could be better for detecting intraventricular dyssynchrony than tissue velocity–derived indexes.

Peak strain and systolic tissue velocity represent different mechanical events: Strain peak indicates the end of shortening or the crossover point of myocardial shortening to lengthening, and tissue velocity peak indicates the timing of maximal speed of myocardial motion. There are 2 possible explanations for the superiority of strain to tissue velocity in our study.

First, strain represents regional contraction more reliably because deformation measurements are not affected by tethering and translational motion. Timing of myocardial motion and displacement may underestimate the degree of timing difference in regional contraction compared with deformation, especially if there is more regional heterogeneity in the timing of contraction.

Second, measuring mechanical timing only in the ejection period may underestimate the severity of mechanical dyssynchrony in LBBB patients because the typical mechanical abnormality of LBBB can be observed during the isovolumic periods. Mechanical dyssynchrony in LBBB or the paced heart is characterized by early septal contraction and prestretching in the lateral wall, which often occurs during the isovolumic contraction period, and postsystolic contraction. Therefore, assess-
Tissue Velocity Versus Strain for the Quantification of Mechanical Dyssynchrony

Without a large prospective clinical trial, it is difficult to determine which parameter best assesses mechanical dyssynchrony or predicts the effect of CRT. The usefulness of strain imaging in evaluating the CRT population has been reported in several studies, but others were not able to demonstrate the usefulness of the strain-derived dyssynchrony index. In addition, in our study, almost 100% of patients with systolic dysfunction showed dyssynchrony if we use the proposed strain-derived cutoff value in the Mele et al study. The reason for this discrepant result is not clear, but we speculate that a cutoff value that can predict the effect of CRT among heart failure population is higher than the one that can separate a normal from an abnormal population.

Nonetheless, the present study supports the superiority of the strain-derived index over systolic tissue velocity timing measurements for evaluating dyssynchrony. Our findings raise a caution for the clinical use of the time to peak systolic tissue velocity for the selection of patients for CRT and/or for atrioventricular or V-V optimization of biventricular pacemaker.

Study Limitations

A major limitation of our study is that we did not compare echocardiography dyssynchrony indexes with other modalities such as cardiac MRI or nuclear study. There is a possibility that a dyssynchronous finding in normal subjects by tissue velocity is a true subclinical abnormality. However, they were definitely not a candidate for CRT. Our study was not designed to assess the predictive value of various dyssynchrony indexes for the response to CRT. Large prospective studies are needed to determine which parameter is best for selecting patients for CRT. Because we measured the timing in individual walls separately, the variation in the RR interval may have caused variability in measurements of time intervals, although considerable care was taken to discard waveforms with obvious noise in strain imaging and obvious differences in the length of the cardiac cycle.

Conclusions

According to previously described tissue velocity timing indexes, a substantial proportion of asymptomatic subjects without LV systolic dysfunction or intraventricular conduction delay exhibit intraventricular mechanical dyssynchrony. Dyssynchrony indexes based on strain timing measurements differentiated normal from abnormal populations more distinctly. The optimal method for measuring LV mechanical dyssynchrony and predicting response to CRT requires further investigation.

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Disclosures

None.

References

Dysynchrony by Tissue Velocity and Strain

Because a substantial number of patients who meet current clinical and ECG criteria for cardiac resynchronization therapy (CRT) do not improve after biventricular pacing, the importance of using a mechanical dysynchrony parameter as an additional selection criterion has been emphasized. Intraventricular dysynchrony measured by tissue velocity imaging differs from peak systolic tissue velocity was found to be a major predictor of the effect of CRT. Currently, echocardiographic dysynchrony indexes are regarded as a standard method for measuring dyssynchrony and are used as a tool to select patients for CRT, to monitor the effect of CRT, and to optimize biventricular pacing. These indexes also have been shown to be frequently positive in heart failure patients with normal QRS duration and/or normal ejection fraction, indicating that CRT may improve their symptoms. Most of these studies, however, are from a single center and are not randomized in a relatively small number of patients. Moreover, how often this echocardiographically derived dysynchrony is present in normal subjects and in patients with heart failure but without conduction delay is not well known. Our study demonstrated that tissue velocity--derived dysynchrony is common in normal healthy control subjects and that there is a large overlap of these values among groups who may or may not be a candidate for CRT. The strain-derived dysynchrony index was found to be better than tissue velocity--derived dysynchrony in distinguishing groups of different ejection fractions and QRS durations. Further clinical investigations are necessary to identify the best echocardiographic dysynchrony parameters and to determine the role of echocardiography in CRT.
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