Clinical Relevance of Abnormal Early Septal Shortening As An Etiological Component Of Left Ventricular Dyssynchrony

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Objectives: We investigated the significance of interventricular septal motion as a component of left ventricular (LV) mechanical dyssynchrony. Background: The presence of ventricular dyssynchrony is a determining factor of response to cardiac resynchronization therapy (CRT), and its evaluation is important for successful CRT. We hypothesized that interventricular septal motion is a key etiological component of ventricular dyssynchrony. Methods: Thirty-nine heart failure patients who were assigned to CRT and 15 healthy subjects were studied. Time interval from the onset of the QRS configuration to peak regional strain was measured by speckle tracking imaging. LV dyssynchrony was defined as a difference in time to peak strain between the septum and lateral wall in circumferential (D-CS) and longitudinal (D-LS) motion. Results: In both circumferential and longitudinal motion, time to peak septal strain was significantly shorter in the patient group than in the normal control group, indicating early shortening. Time to peak lateral wall strain was significantly longer. Consequently, D-CS and D-LS were significantly longer in the patient than normal control groups. Twenty-nine patients responded to CRT at 6 months. D-CS and D-LS were significantly longer in responders than in nonresponders. In the septum, times to peak circumferential and longitudinal strains were significantly shorter in responders than in nonresponders, although no statistically significant difference in the lateral wall between the two groups. After CRT, time to peak septal strain was significantly prolonged, especially in responders. Conclusions: Abnormal early septal shortening is an important etiological component of LV dyssynchrony, and its evaluation is valuable in patient selection for CRT.

Key words: heart failure, left ventricular dyssynchrony, echocardiography, resynchronization therapy

Introduction

Several clinical studies\(^1\)\(^-\)\(^4\) have demonstrated the efficacy of cardiac resynchronization therapy (CRT) with biventricular pacing for patients with advanced heart failure (HF) involving intraventricular conduction delay. CRT should be conducted for patients in whom CRT can exert its expected clinical effects. However, it has also become clear that approximately 30% of patients do not respond to CRT when selected based on QRS duration\(^1\). Recently, better therapeutic efficacy was reported for CRT along with greater severity of ventricular mechanical dyssynchrony\(^3\)\(^0\). Consequently, various methods of assessment for left ventricular (LV) mechanical dyssynchrony have been proposed to be useful in selecting candidate patients for CRT\(^3\)\(^-\)\(^12\). Especially, speckle tracking imaging was developed as a new echocardiographic technique to precisely capture the motion of the regional myocardium\(^13\)\(^-\)\(^15\).

In the 1960s through the 1980s, the deterioration of hemodynamics induced by dyssynchronous LV motion associated with left bundle branch block (LBBB) was examined by mechanocardiography\(^13\), cardiac catheter examination\(^16\)\(^-\)\(^17\), radionuclide scintigraphy\(^18\)\(^-\)\(^19\) and echocardiography\(^20\)\(^-\)\(^21\). Some of the studies have described abnormal motion of the interventricular septum and its early motion at the onset of contraction\(^22\)\(^-\)\(^23\). However, any clinical test-
ing capable of precisely evaluating the ventricular regional motion was not available at the time. Therefore, such analyses may not be considered sufficient.

In the present study, we conducted speckle tracking imaging to examine the pathophysiological and clinical relevance of early septal shortening as an etiological component of LV dyssynchrony.

**Patients and Methods**

This study enrolled 15 healthy subjects and 39 consecutive patients (24 males; 15 females; mean age: 59 ± 10 years) who met the following inclusion criteria: symptoms of heart failure persisting at least for 6 months despite optimal medical therapy; QRS duration ≥ 120 ms, involving LBBB or intraventricular conduction delay; left ventricular ejection fraction (LVEF) ≤ 35%. The etiologies of HF were nonischemic in 31 patients and ischemic in 8 patients. A biventricular pacemaker was implanted in all patients. The LV pacing lead was placed intravenously via the coronary sinus. In 3 patients in whom the placement involved difficulty, it was placed surgically on the epicardium. In all the patients, the LV lead was placed in the middle to the basal segments of the lateral or posterolateral wall. Atroventricular delay was adjusted according to the mitral inflow method previously reported.

Within 1 week prior to the implantation, the following parameters were evaluated: New York Heart Association (NYHA) functional class, QRS duration, left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), LVEF and severity of LV dyssynchrony as assessed by echocardiography. The same evaluations were repeated again at 6 months after implantation. LV volume and LVEF were calculated from the apical 4- and 2-chamber views according to the biplane Simpson’s method.

Written informed consent was obtained from all individuals who were enrolled in this study, and the study was conducted according to the principles of the Declaration of Helsinki.

1. **Measurement of time to peak regional strain**

   LV time-strain curve was examined by speckle tracking imaging. The measuring device used was Vivid 7 (GE Medical Systems, Milwaukee, WI). The image sector width was set as narrow as possible to achieve the highest possible acquisition frame rates (≥ 75 frames/sec), and 5 consecutive images were stored. A customized software running on a personal computer workstation (EchoPAC platform [2D-software package, version 4.0.0]), GE Medical Systems) was used for the subsequent off-line analysis of speckle tracking imaging results. As illustrated in Fig. 1, regional strains in circumferential and longitudinal motion in the septum and lateral wall were analyzed at the same basal level of the LV. The parasternal short axis view and the apical 4-chamber views were used to analyze circumferential and longitudinal strains in each region, respectively. Time interval from the onset of QRS configuration to peak negative strain in each region was measured. Those intervals were used as time to peak regional strain. Differences in time to peak strain between the septum and the lateral wall, i.e., the difference in time to peak circumferential strain (D-CS) and the difference in time to peak longitudinal strain (D-LS), were measured as indices for LV dyssynchrony.

   Furthermore, 15 healthy subjects (mean age: 31 ± 6 years; mean QRS duration: 82 ± 7 ms) were analyzed similarly; the normal control group was then compared with the patient group.

2. **Assessment of response to CRT**

   The chronic effects of CRT were evaluated based on changes in NYHA class and LVESV from baseline to 6 months after CRT. Patients who showed an improvement in NYHA class of ≥ 1 category and a decrease in LVESV of ≥ 10% after CRT were defined as responders to CRT. Patients who failed to meet these conditions, who died of HF within 6 months after CRT, or in whom a LV assist device was implanted, were defined as nonresponders.

   The responder group and the nonresponder group were compared with respect to baseline characteristics and severity of LV dyssynchrony, and times to peak regional strain before and after CRT.
Fig. 1

Fig. 2

A Time to peak septal strain in circumferential motion
B Time to peak septal strain in longitudinal motion
C Time to peak lateral strain in circumferential motion
D Time to peak lateral strain in longitudinal motion
3. Analysis of LV pressure-regional strain relationship

LV pressure and regional myocardial strain were measured simultaneously to evaluate their relationship in 3 patients.

A micromanometer catheter (Millar Instruments, Houston, TX) was inserted via the right femoral artery to measure LV pressure while simultaneously conducting speckle tracking imaging. To synchronize LV pressure and regional strain, electrocardiographic data were entered into an amplifier for pressure recording and the ECG unit of an echocardiograph via a branch cable. Data on pressure were digitized at 1,000 Hz and analyzed off-line. The LV pressure-regional strain loop was drawn based on the time-pressure curve and time-regional strain curve.

Fig. 1 Measurement of time to peak regional strain using speckle tracking imaging in a normal control subject
A: Scheme showing measurement sites and measurement directions.
B: In the interventricular septum and the left ventricular lateral wall at the basal level, time from onset of QRS configuration to peak negative strain (white arrow) was measured in circumferential. The blue line denotes the circumferential time-strain curve of the interventricular septum, and the green line that of the lateral wall.
C: Time to peak negative strain (white arrow) was also measured in longitudinal motion. The yellow line denotes the longitudinal time-strain curve of the interventricular septum, and the blue line that of the lateral wall.

Fig. 2 Circumferential and longitudinal time-strain curves in a typical patient
A: Circumferential time-strain curve in the interventricular septum.
B: Longitudinal time-strain curve in the interventricular septum.
C: Circumferential time-strain curve in the lateral wall.
D: Longitudinal time-strain curve in the lateral wall.

4. Statistical Analysis

Values are expressed as mean ± SD. Unpaired Student's t-test or χ²-test was conducted to compare the normal control group with the patient group and the CRT responder group with the CRT nonresponder group. For the comparison of parametric variables before and after CRT, paired Student's t-test was used. A value of p < 0.05 was considered statistically significant.

Results

1. Time to peak regional strain

Times to peak circumferential and longitudinal strains in the lateral wall were significantly (p < 0.0005) longer in the patient group than in the normal control group (Table 1). In the septum, in contrast, times to peak circumferential and longitudinal strains were significantly shorter by 69 ± 75 ms (p < 0.05) and 53 ± 82 ms (p < 0.05), respectively, in the patient group than in the normal control group. The patient group showed early septal shortening, along with a delay in lateral wall shortening, compared with the normal control group.

The time-regional strain curves of a typical patient are shown in Figure 2. Time to peak septal strain was extremely short in both circumferential and longitudinal motion, and the peak of septal strain appeared within QRS duration. In the late-systole, the interventricular septum showed positive strain value, i.e., its stretching. The lateral wall showed a positive strain value in the early systole, indicating its contraction once extended. Peak lateral wall strain appeared later than aortic valve closing, i.e., myocardial contraction continued also after entering the diastole.

Compared with baseline, times to peak septal strain after CRT prolonged significantly to 417 ± 76 ms (p < 0.0001) and 457 ± 68 ms (p < 0.0001) in circumferential and longitudinal motion, respectively. Times to peak lateral wall strain shortened significantly to 388 ± 70 ms (p < 0.0001) and 447 ± 68 ms (p < 0.0001) in respective motion.

2. LV dyssynchrony

In both the normal control and patient groups, time to peak strain was significantly longer in the lateral wall than in the interventricular septum (Ta-
Table 1 Time to peak systolic regional strain and parameters for LV dyssynchrony in patients and normal controls

<table>
<thead>
<tr>
<th></th>
<th>Septum (ms)</th>
<th>Lateral wall (ms)</th>
<th>Difference in time to peak strain</th>
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<tbody>
<tr>
<td><strong>Circumferential motion</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient</td>
<td>303 ± 106 *</td>
<td>482 ± 71.5 †</td>
<td>D-CS: 179 ± 101 †</td>
</tr>
<tr>
<td>Control</td>
<td>372 ± 31.0 *</td>
<td>400 ± 34.4</td>
<td>D-CS: 27.1 ± 19.7</td>
</tr>
<tr>
<td><strong>Longitudinal motion</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Patient</td>
<td>309 ± 103 *</td>
<td>536 ± 65.5 †</td>
<td>D-LS: 184 ± 107 †</td>
</tr>
<tr>
<td>Control</td>
<td>362 ± 21.8 *</td>
<td>388 ± 29.0</td>
<td>D-LS: 25.9 ± 18.1</td>
</tr>
</tbody>
</table>

* p < 0.0001 vs lateral wall, † p < 0.0001 vs control group, ‡ p < 0.005 vs control group.
D-CS = difference in time to peak circumferential strain between the septum and lateral wall.
D-LS = difference in time to peak longitudinal strain between the septum and lateral wall.

Fig. 3 Correlations between QRS duration and time to peak regional strain
QRS duration and time to peak septal strain showed no significant correlation in both circumferential (A) and longitudinal (B) motion. However, QRS duration and time to peak lateral wall strain showed a significant correlation in both circumferential (C) and longitudinal (D) motion.

In the normal control group, however, both D-CS and D-LS were less than 30 ms. By contrast, both D-CS and D-LS in the patient group were significantly longer (approximately 180 ms) than in the normal control group, thus exhibiting LV dyssynchrony.

3. Relation of time to peak regional strain with QRS duration
The mean QRS duration before implantation was 178 ± 28 ms (range, 120-230 ms). Relations of QRS duration with respective parameters for speckle tracking imaging are shown in Figure 3. In the in-
terventricular septum, times to peak strain in both circumferential and longitudinal motion showed no significant correlation with QRS duration. In the lateral wall, on the other hand, times to peak strain in both circumferential and longitudinal motion were longer with greater QRS duration, and there was a significant correlation between them (% r = 0.46; p = 0.003).

4. Relationship of LV pressure and regional strain

A typical example of LV pressure-regional circumferential strain loops in septal and lateral segments is shown in Figure 4. Septal strain peaked before LV pressure rose, thus indicating shortening at a very early systolic phase. The peak strain value was small, and subsequent changes in strain value in the course of LV pressure increase were slight; hence, shortening did not progress further. The pressure-strain loop in the lateral wall rotated counterclockwise. The strain values at the early systolic phase were positive, suggesting wall extension. The strain values in the lateral wall increased continuously in the course of LV pressure increase, and timing for peak shortening appeared later than timing for maximum LV pressure. The area of pressure-regional strain loop was obviously larger in the lateral wall than in the septum. The relationship between longitudinal strain and LV pressure was similar. The findings were similar for other two patients.

5. Comparison of baseline clinical and echocardiographic parameters between responders and nonresponders to CRT

The mean values at baseline were as follows: LVEDV, 246 ± 105 ml; LVESV, 197 ± 93 ml and LVEF were 21.5 ± 7.5%.

After CRT, NYHA class improved by 1 category in 24 patients (62%) and by ≥ 2 categories in 9 patients (23%). LVESV significantly decreased by 27% on average (p < 0.0001), and 31 patients (79%) had a ≥ 10% decrease. Among the 39 patients, there were 29 (74%) responders and 10 (26%) nonresponders. No significant difference was found between the two groups with respect to age, gender, underlying
Table 2 Comparisons of Baseline Clinical Characteristics and Echocardiographic Parameters between Responders and Nonresponders to CRT

<table>
<thead>
<tr>
<th></th>
<th>Responder (n = 29)</th>
<th>Nonresponder (n = 10)</th>
<th>p Value</th>
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<tbody>
<tr>
<td>Age (yrs)</td>
<td>57 ± 14</td>
<td>58 ± 16</td>
<td>NS</td>
</tr>
<tr>
<td>Men/Women</td>
<td>16/13</td>
<td>8/2</td>
<td></td>
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<tr>
<td>Etiology</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Nonsurgical/Ischemic (%)</td>
<td>24/5 (83/17)</td>
<td>7/3 (70/30)</td>
<td></td>
</tr>
<tr>
<td>NYHA class IV (%)</td>
<td>8 (26)</td>
<td>4 (40)</td>
<td></td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>185 ± 27</td>
<td>162 ± 25</td>
<td>0.02</td>
</tr>
<tr>
<td>LVEDV (ml)</td>
<td>233 ± 94</td>
<td>278 ± 133</td>
<td>NS</td>
</tr>
<tr>
<td>LVESV (ml)</td>
<td>188 ± 84</td>
<td>223 ± 115</td>
<td>NS</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>21.5 ± 7.8</td>
<td>21.6 ± 7.0</td>
<td>NS</td>
</tr>
<tr>
<td>Index of LV dyssynchrony</td>
<td></td>
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</tr>
<tr>
<td>D-CS (ms)</td>
<td>212 ± 91.8</td>
<td>847 ± 44.2</td>
<td>0.0002</td>
</tr>
<tr>
<td>D-LS (ms)</td>
<td>229 ± 136</td>
<td>114 ± 77.2</td>
<td>0.02</td>
</tr>
</tbody>
</table>


heart disease, NYHA class at baseline, LVEDV, LVESV and LVEF (Table 2). QRS duration was significantly wider in the responder group than in the nonresponder group (p = 0.02). Two indices for LV dyssynchrony, D-CS and D-LS, were significantly greater in the responder group than in the nonresponder group (p = 0.0002, p = 0.02, respectively).

Times to peak strain in each segment and motion were compared among the responder group, the nonresponder group, and the normal control group (Fig. 5). In the septum, time to peak circumferential strain was significantly shorter in the responder group (283 ± 114 ms) than in the nonresponder (363 ± 33.7 ms, p < 0.05) and normal control group (372 ± 31 ms, p < 0.01). Also in the longitudinal motion, time to peak septal strain was significantly shorter in the responder group (304 ± 102 ms) than in the nonresponder (370 ± 45.2 ms, p < 0.05) and normal control groups (362 ± 21.8 ms, p < 0.05). In the lateral wall, time to peak circumferential strain was longer in the responder and nonresponder groups than in the normal control group. However, no significant difference was found between the responder and nonresponder groups. The results were similar also in the longitudinal motion.

After CRT, time to peak septal strain prolonged significantly in both the responder and nonresponder groups (Fig. 6) as compared with baseline in circumferential and longitudinal motion (p < 0.0001, p < 0.0001, respectively). A comparison of the two groups revealed a significant improvement in the responder group. The extent of the prolongation was significantly greater in the responder group than in the nonresponder group in circumferential (138 ± 120 ms vs. 58.1 ± 64.6 ms, p < 0.05) and longitudinal (143 ± 113 ms vs. 48.2 ± 55.2 ms, p = 0.02) motion. On the other hand, time to peak lateral wall strain shortened significantly in the two groups, with no significant difference in the extent of improvement between the two groups.

Discussion

The major findings in the present study can be summarized as follows: 1) peak septal shortening occurs earlier in patients with chronic HF involving intraventricular conduction delay than in normal controls; 2) this abnormal early septal shortening is an etiological component for LV dyssynchrony; and 3) the earliness of ventricular septal shortening is closely associated with the chronic effects of CRT. Since the mid-term response to CRT is closely related to long-term prognosis, the prediction of the former is crucial in selecting patients for CRT. Many studies have reported the difficulty of predicting the response to CRT based on QRS duration. In contrast, the severity of ventricular dyssynchrony is strongly correlated with the mid-term effects of CRT. Many investigators have described the usefulness of echocardiography and have used the time difference in reaching peak systole between the interventricular septum/antero-
Fig. 5 Comparisons of time to peak regional strain among responders, nonresponders, and normal controls.

A: Time to peak septal strain in circumferential motion
B: Time to peak septal strain in longitudinal motion
C: Time to peak lateral wall strain in circumferential motion
D: Time to peak lateral wall strain in longitudinal motion

Time to peak septal strain in circumferential (A) and longitudinal (B) motions were significantly shorter in the responder group than in the nonresponder and normal control groups. Lateral wall circumferential (C) and longitudinal (D) strains were significantly prolonged in the responder and nonresponder groups compared with the normal control group. However, no significant difference was found between the responder and nonresponder groups.

*p<0.05, †p<0.01, ‡p<0.001.

Although previous studies have emphasized contraction delay of LV lateral wall/posterior wall as a causative factor of LV dyssynchrony, no studies have focused on the relevance of early septal shortening. In the present study, time to peak lateral wall strain was significantly longer in patients with heart failure involving intraventricular conduction delay than in normal controls. At the same time, time to peak strain in the interventricular septum was significantly shorter in patients than in normal controls. After CRT, abnormal early shortening of the septum was definitely corrected. These results were constant regardless of circumferential and longitudinal motion. Therefore, we can interpret that dyssynchrony between the septum and the lateral wall is attributable to both delayed shortening of the lateral wall and early shortening of the septum.

Our study showed that time to peak lateral wall strain tended to be later in the responder group than in the nonresponder group. However, no statistically significant difference was found between the two groups. In contrast, time to peak septal strain in circumferential and longitudinal motion was significantly shorter in the responder group. After CRT, time to peak septal strain prolonged significantly in the responder group than in the nonresponder group. Therefore, the improvement of dyssynchrony was evidenced to involve not only shortening of time to lateral wall strain but also prolongation of time to peak septal strain. These findings suggest that early septal shortening is closely related to the response to CRT. Previous studies have reported that QRS duration at baseline fails to
predict response to CRT and that QRS duration is not correlated with the severity of LV dyssynchrony. Interestingly, our study indicated that QRS duration was correlated with time to peak circumferential and longitudinal strains in the lateral wall, but not with that in the interventricular septum. We can speculate that one of the causes for the lack of correlation between QRS duration and LV dyssynchrony is early septal shortening.

Abnormal septal motion in patients with LBBB has been described in several papers. In 1971, Haft et al. described for the first time left ventricular wall motion abnormality in association with LBBB. In 1973, McDonald et al. used echocardiograms to report the early and abrupt contraction of the interventricular septum in patients with LBBB. Later, this early movement of the interventricular septum was explained by a right-to-left ventricular pressure difference. Grines et al. examined the relationship between right and left ventricular pressures and interventricular septal motion that had been documented by M-mode echocardiography in patients with LBBB. They concluded in patients with LBBB, in whom the right ventricles contract prior to the LV, that the interventricular septum is displaced passively into the LV because right ventricular pressure which increased during isovolumetric contraction time exceeded LV pressure still in the diastole. Timing for abrupt displacement that they showed is close to timing for early septal short-
The mechanism by which septal abnormal shortening occurs is not clear. However, the mechanism is possibly explained by load on the regional myocardium and by the tension generated in association with it. At the onset of contraction of the interventricular septum—earliest activation site, other segments of the LV are still relaxed. Therefore, the interventricular septum contracts nearly isotonically toward a very low load. Subsequently, the septum undergoes increased loading of a pressure elevation due to contraction of other segments at the moment when its shortening progresses to a certain extent. However, tension generated from a short muscle length is small. If the tension is smaller than the stretching force provoked by an increase in inner pressure, further shortening will not occur. In addition, stretching of the contracting myocardium breaks cross-bridge and reduces the contractile force of the interventricular septum. Gillebert et al. have examined the generation of LV tension when cramping the aorta during LV contraction and have shown that tension decreased with later timing for afterload induced by the cramping." This finding leads us to believe that the contractile force of the interventricular septum increases less because LV pressure increases slower due to a greater delay in contraction of the segments other than the septum during septal contraction and shortening of the septum terminates earlier. Specifically, we believe that septal shortening peaks earlier if contraction of the LV free wall is greatly delayed, with a synergistically greater increase in LV dyssynchrony.

External work of the septum is very small because it contracts isometrically during ejection time. As indicated in the pressure-regional strain relationship in the present study, the interventricular septum presents early peak strain. However, its value is small and decreases gradually in such a manner to be overcome by an increase in LV pressure. The pressure-regional strain area in the interventricular septum is very small compared with that in the lateral wall, indicating that the work of the septum is very small.

This study has some limitations. We used time-lag in time to peak strain between the interventricular septum and the LV lateral wall as a parameter for LV dyssynchrony. However, whether or not the lag is the best parameter to depict LV dyssynchrony remains unknown at present. Therefore, further study will be required in other unexamined segments.

The results from the speckle tracking imaging analysis led us to conclude that early septal motion is active contraction. Nevertheless, we do not rule out the effects of right ventricular pressure elevation. The mechanism by which the interventricular septum poses complicated movements is possibly attributable to its displacement caused by compression from the right ventricular, apart from its active contraction and changes in load induced by an increase in LV pressure. To better comprehend these factors, we need to simultaneously measure LV pressure, right ventricular pressure, septal strain, and septal displacement in an attempt to analyze them in detail.

**Conclusion**

Abnormal early septal shortening in patients with LV conduction delay is an important etiological component of LV dyssynchrony. Since this early septal shortening is closely related to the chronic effects of CRT, the evaluation of not only free wall contraction delay but also interventricular septal motion is important in selecting candidate patients for CRT.

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左室同期不全における中隔早期収縮の臨床的意義

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〔背景〕心不全患者の予後はβ遮断薬の導入により向上したが、今なお薬物治療抵抗性的重症心不全患者が多数存在する。心不全症例の約半数に心室内伝導障害を認め、これは広範な心筋ダメージを反映しきそゆえ予後と関連すると解釈されてきた。しかし近年、伝導障害によりもたらされる心室収縮運動の非同期性が血行動態に大きな悪影響を及ぼすことが明らかとなった。これを是正する治療として心房再同期療法（CRT）が行われるようになり、約40％の生命予後改善効果が認められるようになった。一方、現在の心電図QRS幅による適応基準では30％のCRT非適応群が存在することが指摘されている。

心室同期不全の有無はCRTを成功する決定要因であり、その直接評価が重要である。我々は心室中隔の異常運動が心室同期不全の重要な構成要素であり、CRTの適応決定に重要であると仮説した。

〔対象および方法〕CRTを行うことになる39人の左室伝導障害を伴う重症心不全患者と15人の健康者を登録した。Speckle tracking imagingを用いて円周方向（circumferential motion）と長軸方向（longitudinal motion）の左室基部における心室中隔と側壁の各領域における局所最大収縮時間time to peak regional strainを求める。心室中隔と側壁間の収縮時間の差を心室同期不全の指標とした：Difference of time to peak strain between septum and lateral wall in circumferential direction（D-CS）とthat in longitudinal direction（D-LS）。

〔結果〕円周方向、長軸方向のいずれも健康コントロール群と比較し症例群では側壁における最大収縮時間は有意に長かった。その一方で心室中隔における最大収縮時間は症例群で有意に短く（327±31.0ms vs.303±106ms，362±21.8ms vs.309±103ms，それぞれp＞0.05）。かつ収縮早期時相に局所心筋の最大短縮を示していた。その結果、症例群のD-CS，D-LSは健康コントロール群に比べ著しく長かった。CRT6ヶ月後，29例（74％）にCRTによる効果を認めた。CRT非適応群のD-CS（212±91.8ms vs.84.7±44.2ms，p=0.0002）とD-LS（229±136ms vs.114±77.2ms，p=0.02）は、非適応群のそれよりも有意に長かった。側壁における最大収縮時間は、CRT非適応群において非適応群のそれよりも有意に長かったが、円周方向および長軸方向ともCRT非適応群在非適応群間で統計学的有意差を認めなかった。これに対し、心室中隔における最大収縮時間は2つの方向ともCRT非適応群では非適応群よりも有意に短かった。

〔結論〕左室伝導障害を有する症例では、心室中隔は異常早期収縮運動abnormal early shorteningを呈しており、それが心室同期不全の大きな構成要素となっていた。この中隔におけるabnormal early shorteningがCRTの慢性効果と密接に関わっていることから、CRTの患者選択においては側壁における収縮遅延のみならず、心室中隔の異常運動の評価が重要である。