Left ventricular systolic and diastolic dyssynchrony in coronary artery disease with preserved ejection fraction

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ABSTRACT

The present study aims to evaluate LV (left ventricular) mechanical dyssynchrony in CAD (coronary artery disease) with preserved and depressed EF (ejection fraction). Echocardiography with TDI (tissue Doppler imaging) was performed in 311 consecutive CAD patients (94 had preserved EF ≥ 50% and 217 had depressed EF < 50%) and 117 healthy subjects to determine LV systolic and diastolic dyssynchrony by measuring Ts-SD (S.D. of time to peak myocardial systolic velocity during the ejection period) and Te-SD (S.D. of time to peak myocardial early diastolic velocity during the filling period) respectively, using a six-basal/six-mid-segmental model. In CAD patients with preserved EF, both Ts-SD (32.2 ± 17.3 compared with 17.7 ± 8.6 ms; \( P < 0.05 \)) and Te-SD (26.2 ± 13.6 compared with 20.3 ± 8.1 ms; \( P < 0.05 \)) were significantly prolonged when compared with controls, although they were less prolonged than CAD patients with depressed EF (Ts-SD, 37.8 ± 16.5 ms; and Te-SD, 36.0 ± 23.9 ms; both \( P < 0.005 \)). Patients with preserved EF who had no prior MI (myocardial infarction) had Ts-SD (32.9 ± 17.5 ms) and Te-SD (28.6 ± 14.8 ms) prolonged to a similar extent (\( P = \) not significant) to those with prior MI (Ts-SD, 28.4 ± 16.8 ms; and Te-SD, 25.5 ± 15.0 ms). Patients with class III/IV angina or multi-vessel disease were associated with more severe mechanical dyssynchrony (\( P < 0.05 \)). Furthermore, the majority of patients with mechanical dyssynchrony had narrow QRS complexes in those with preserved EF. This is in contrast with patients with depressed EF in whom systolic and diastolic dyssynchrony were more commonly associated with wide QRS complexes. In conclusion, LV mechanical dyssynchrony is evident in CAD patients with preserved EF, although it was less prevalent than those with depressed EF. In addition, mechanical dyssynchrony occurred in CAD patients without prior MI and narrow QRS complexes.

INTRODUCTION

In heart failure with systolic dysfunction, LV (left ventricular) dyssynchrony is a common phenomenon [1]. It is believed that the presence of LV dyssynchrony can impair the function of a failing ventricle further, resulting in worsening of symptoms and clinical outcomes [2,3]. Electrical dyssynchrony refers to the asynchronous electrical activation of the left ventricle in association with intraventricular conduction disturbance (e.g., bundle branch block), whereas the term mechanical dyssynchrony is becoming increasingly used to describe the mechanical

Key words: coronary artery disease (CAD), dyssynchrony, ejection fraction, tissue Doppler imaging.

Abbreviations: CAD, coronary artery disease; EF, ejection fraction; LV, left ventricular; MI, myocardial infarction; TDI, tissue Doppler imaging; Te, time to peak myocardial early diastolic velocity during the filling period; Te-diff, maximal difference in Te; Te-SD, S.D. of Te; Ts, time to peak myocardial systolic velocity during the ejection period; Ts-diff, maximal difference in Ts; Ts-SD, S.D. of Ts.

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efforts of asynchronous ventricular contraction and relaxation, which may or may not be associated with electrical conduction delay. Although LV dyssynchrony was initially recognized as a phenomenon related only to electrical conduction delay in systolic heart failure with widened QRS complexes, previous studies have reported that it also exists in approx. 30–40% of patients with a normal QRS duration [1] and in a significant number of patients with heart failure and preserved EF (ejection fraction) [4,5]. CAD (coronary artery disease) is one of the commonest causes of heart failure with preserved EF; however, there are limited results about mechanical dyssynchrony in CAD patients with preserved EF. Several previous studies in animals and patients have suggested that myocardial ischaemia results in a delay in relaxation [6–10] and contraction [10–13] without producing a regional wall motion abnormality on conventional echocardiography. TDI (tissue Doppler imaging) has been validated for assessment of the timing of regional myocardial motions from which indices of systolic and diastolic dyssynchrony can be derived [1,14,15]. In the present study, we examined the presence and characteristics of mechanical dyssynchrony (both systolic and diastolic) in CAD patients with preserved EF in comparison with CAD patients with depressed EF and healthy controls.

**MATERIALS AND METHODS**

**Subjects**

A total of 311 consecutive patients referred to a cardiac speciality clinic in a tertiary university hospital with the diagnosis of CAD and 117 age- and gender-matched healthy subjects were prospectively recruited into the present study. The diagnosis of CAD was established by the presence of chest pain plus one or more of the following objective evidence of CAD: (i) documented prior MI (myocardial infarction) based on the presence of pathological Q waves in two or more contiguous leads on an ECG; (ii) coronary stenosis on invasive coronary angiography of at least 70%; and (iii) in very few patients by the presence of myocardial ischaemia on functional stress testing. Of the 311 patients in the present study, 115 had documented coronary stenosis on coronary angiography, of which 57 had an EF ≥50% and 58 had an EF <50%. Prior MI was evident in 208 patients. In 22 patients, CAD was diagnosed based on typical angina symptoms and positive stress testing. These patients either refused or were considered not to be clinical candidates for coronary revascularization due to co-morbid conditions. Patients who had atrial fibrillation, previous pacemaker implantation, restrictive cardiomyopathy, aortic or mitral stenosis, prosthetic valves, severe mitral annular calcification or revascularization within the past 6 months were excluded.

Study patients were considered to have preserved EF if EF was ≥50% (preserved EF group; n = 94) as measured by echocardiography using biplane Simpson’s method. Those who had an EF <50% were considered as having depressed EF (depressed EF group; n = 217). Control subjects had no history of cardiovascular or systemic disease, and had normal physical examination, ECG and echocardiographic results.

The present study was approved by the ethics committee for clinical research at our institution, and written informed consent was obtained from all subjects.

**Echocardiography**

Transthoracic echocardiography was performed for all subjects (Vivid 7; Vingmed-General Electric). LV volumes and EFs were assessed by biplane Simpson’s method using the apical four- and two-chamber views. At least three consecutive beats in sinus rhythm were measured, and the average values were taken. Regional wall motion abnormalities were evaluated by visual assessment of systolic wall thickening on two-dimensional echocardiography [16]. TDI was performed at apical views (apical four-chamber, two-chamber and long-axis views) for assessing the longitudinal motions of the left ventricle, as described previously [1,14,15]. Two-dimensional echocardiography with colour TDI optimized for pulse repetition frequency, colour saturation, sector size and depth were obtained to maximize the frame rate to 105 Hz or higher. At least three consecutive beats were stored, and the images were analysed offline using customized software (EchoPac-PC, version 6.1.0; Vingmed-General Electric). Myocardial velocity curves were reconstructed offline using the six-basal/six-mid-segmental model, which consisted of septal, lateral, anteroseptal, posterior, anterior and inferior segments at both basal and mid-levels of the left ventricle [1,14,15]. The investigators who performed the TDI analysis were blinded from the clinical information of the subjects. Two-dimensional image and TDI analyses were performed separately such that the investigator who analysed the TDI curves was blinded from the two-dimensional information, such as EF and regional wall motion abnormalities. The basal segments were sampled just above the mitral annulus level, and the mid-segments were sampled at the mid-level of the left ventricle. Ts (time to peak myocardial systolic velocity during the ejection period) and Te (time to peak myocardial early diastolic velocity during the filling period) were measured with reference to the onset of the QRS complex. The timings of the beginning and the end of LV ejection (aortic valve opening and closure) and filling (mitral valve opening and closure) periods were determined by continuous-wave Doppler of the aortic forward flow and pulse-wave Doppler of the mitral inflow. Markers with valve opening and closing events
Table 1  Clinical and demographic characteristics of the CAD patients

<table>
<thead>
<tr>
<th></th>
<th>Preserved EF (n = 94)</th>
<th>Depressed EF (n = 217)</th>
<th>$\chi^2$</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>63.5 ± 10.1</td>
<td>64.8 ± 11.6</td>
<td>—</td>
<td>NS</td>
</tr>
<tr>
<td>Male gender (%)</td>
<td>76</td>
<td>74</td>
<td>1.35</td>
<td>NS</td>
</tr>
<tr>
<td>CCS angina class (%)</td>
<td>57</td>
<td>9</td>
<td>67.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>I</td>
<td>35</td>
<td>42</td>
<td>9.05</td>
<td>NS</td>
</tr>
<tr>
<td>II</td>
<td>8</td>
<td>44</td>
<td>0.52</td>
<td>NS</td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>5</td>
<td>3.25</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>53</td>
<td>55</td>
<td>0.07</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus (%)</td>
<td>39</td>
<td>44</td>
<td>0.07</td>
<td>NS</td>
</tr>
<tr>
<td>MI (%)</td>
<td>60</td>
<td>70</td>
<td>3.25</td>
<td>NS</td>
</tr>
<tr>
<td>QRS duration (ms)</td>
<td>84.0 ± 15.7</td>
<td>120.2 ± 32.6</td>
<td>—</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>QRS &gt; 120 ms (%)</td>
<td>6</td>
<td>30</td>
<td>20.68</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>EF (%)</td>
<td>61.7 ± 8.3</td>
<td>34.8 ± 9.7</td>
<td>—</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Coronary angiography (%)</td>
<td>SVD/DVD</td>
<td>86</td>
<td>46</td>
<td>18.5</td>
</tr>
<tr>
<td></td>
<td>TVD/LMD</td>
<td>14</td>
<td>54</td>
<td></td>
</tr>
<tr>
<td>Medication (%)</td>
<td>Antiplatelet agent</td>
<td>93</td>
<td>73</td>
<td>15.29</td>
</tr>
<tr>
<td></td>
<td>ACEI or ARB</td>
<td>54</td>
<td>71</td>
<td>8.15</td>
</tr>
<tr>
<td></td>
<td>$\beta$-Blocker</td>
<td>73</td>
<td>40</td>
<td>29.11</td>
</tr>
<tr>
<td></td>
<td>Loop diuretic</td>
<td>24</td>
<td>65</td>
<td>48.18</td>
</tr>
</tbody>
</table>

would appear on the ECG recordings during offline TDI analysis to ensure only the peak myocardial systolic and early diastolic velocities with their corresponding Ts and Te were measured accurately. For the assessment of LV intra-ventricular dyssynchrony, Ts-SD (S.D. of Ts) and Te-SD (S.D. of Te) as well as Ts-diff (maximal difference in Ts) and Te-diff (maximal difference in Te) of all at the 12 left ventricle segments were calculated [1,14]. The inter- and intra-observer variability for measuring dyssynchrony were compared in 60 consecutive measurements and were 4.7 and 3.2 % respectively.

RESULTS

Among the preserved EF, depressed EF and control groups, there were no significant differences in age (63.5 ± 10.1, 64.8 ± 11.6 and 64.2 ± 9.4 years respectively) and gender distribution (76, 74, and 71 % male respectively). The clinical and demographic characteristics of the two groups of CAD patients are shown in Table 1. The prevalence of hypertension, diabetes and MI were similar. However, more patients with depressed EF were prescribed loop diuretics, ACEIs (angiotensin-converting enzyme inhibitors) and/or ARBs (angiotensin receptor blockers).

Statistics

Data were analysed using SPSS (version 13.0). For the comparison of mechanical dyssynchrony and other parametric data among the various groups, independent Student’s t tests and one-way ANOVAs with post-hoc Scheffe’s test for inter-group differences were employed, where appropriate. Linear regression was employed to investigate the correlation between two parametric variables. Comparison of categorical data was performed using a Pearson $\chi^2$ test. Continuous data are expressed as means ± S.D. A P value < 0.05 was considered statistically significant.

Systolic mechanical dyssynchrony in CAD patients with preserved and depressed EF

Compared with normal controls, systolic mechanical dyssynchrony was highly prevalent in CAD patients with preserved EF. Both Ts-SD and Ts-diff were significantly prolonged in the preserved EF group, but were more so in the depressed EF group (Table 2). Using a cut-off value of Ts-SD > 33 ms derived from the upper 2 S.D. of the mean from normal subjects [8], 41 % (39 out of 94) of patients in the preserved EF group and 57 % (123 out of 217) in the depressed EF group had
Table 2  Comparison of systolic and diastolic dyssynchrony in CAD patients who had preserved or depressed EF, and normal controls

P values are Scheffe-corrected.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Controls</th>
<th>CAD patients with Preserved EF</th>
<th>CAD patients with Depressed EF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Controls compared with preserved EF</td>
</tr>
<tr>
<td>Ts-SD (ms)</td>
<td>17.7 ± 8.6</td>
<td>32.3 ± 17.3</td>
<td>37.8 ± 16.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Ts-diff (ms)</td>
<td>53 ± 24</td>
<td>95 ± 49</td>
<td>109 ± 45</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Te-SD (ms)</td>
<td>20.3 ± 8.1</td>
<td>26.2 ± 13.6</td>
<td>36.0 ± 23.9</td>
<td>0.026</td>
</tr>
<tr>
<td>Te-diff (ms)</td>
<td>66 ± 28</td>
<td>85 ± 47</td>
<td>111 ± 69</td>
<td>0.016</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>70.7 ± 8.0</td>
<td>61.7 ± 8.3</td>
<td>34.8 ± 9.7</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Figure 1  Scatter plots of Ts-SD (A) and Te-SD (B)
The dotted lines denote the cut-off values for systolic and diastolic dyssynchrony derived from normal controls [8]. Mechanical dyssynchrony in CAD was most severe in patients with depressed EF, but was also evident in patients with preserved EF.

Systolic dyssynchrony ($\chi^2 = 6.07, P = 0.01$) (Figure 1A). Using Ts-diff > 100 ms as the cut-off value derived from the upper 2 S.D. of the mean from normal subjects [8], a similar prevalence was found in the preserved and depressed EF group [39% (37 out of 94) compared with 58% (126 out of 217); $\chi^2 = 9.20, P = 0.002$].

Diastolic dyssynchrony in CAD patients with preserved and depressed EF
Likewise, diastolic dyssynchrony was evident in the preserved EF group. Both Te-SD and Te-diff were significantly prolonged in the preserved EF group, but were more severe in the depressed EF group (Table 2). Using a cut-off value of Te-SD > 34 ms, as derived from the upper 2 S.D. of the mean from normal subjects [8], diastolic dyssynchrony was present in 20% (19 out of 94) of patients in the preserved EF group and 38% (83 out of 217) in the depressed EF group ($\chi^2 = 9.68, P = 0.002$) (Figure 1B). Similarly, when Te-diff > 113 ms was used as the cut-off value [8], the prevalence of diastolic dyssynchrony was significantly lower in the preserved EF group than the depressed EF groups [19% (18 out of 94) compared with 37% (81 out of 217); $\chi^2 = 9.99, P = 0.002$].

Clinical symptoms and coronary angiographic findings in relation to mechanical dyssynchrony
Angina was more severe in patients with depressed EF (Table 1) using the Canadian Cardiovascular Society classification. Patients with class III/IV angina had significantly higher Ts-SD (36.4 ± 16.3 compared with 31.4 ± 17.6 ms; $P = 0.01$) and Te-SD (37.3 ± 24.3 compared with 29.4 ± 17.1 ms; $P < 0.001$) than those with class I/II angina.

Coronary angiography was performed in 57 patients in the preserved EF group and 58 in the depressed EF group. More patients with depressed EF had coronary angiograms showing triple-vessel or left main CAD (Table 1). Patients with triple-vessel/left main diseases had significantly higher Ts-SD (48.9 ± 12.9 compared with 21.6 ± 9.7 ms; $P < 0.001$) and Te-SD (36.2 ± 19.1 compared with 21.7 ± 6.2 ms; $P < 0.001$) than those with single/double-vessel diseases.

Prior MI and mechanical dyssynchrony
A total of 208 patients had had a prior MI. These patients had ECG evidence together with akinesia of the corresponding left ventricle segments on echocardiography. Among patients with preserved EF, mechanical synchrony was abnormal when compared with healthy
Table 3 Comparison of mechanical dyssynchrony in CAD patients with respect to the absence or presence of a prior MI

<table>
<thead>
<tr>
<th>Variable</th>
<th>CAD patients with preserved EF</th>
<th>CAD patients with depressed EF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Prior MI (n = 56)</td>
<td>No prior MI (n = 38)</td>
</tr>
<tr>
<td>Ts-SD (ms)</td>
<td>28.4 ± 16.8</td>
<td>32.9 ± 17.5</td>
</tr>
<tr>
<td>Ts-diff (ms)</td>
<td>83.0 ± 47.0</td>
<td>93.2 ± 44.9</td>
</tr>
<tr>
<td>Te-SD (ms)</td>
<td>25.3 ± 15.0</td>
<td>28.6 ± 14.8</td>
</tr>
<tr>
<td>Te-diff (ms)</td>
<td>81.3 ± 48.9</td>
<td>92.8 ± 54.8</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>62.4 ± 8.7</td>
<td>65.1 ± 8.6</td>
</tr>
</tbody>
</table>

controls (Table 3 and Figure 2), and was increased to a similar extent irrespective of whether or not there had been a prior MI (examples of mechanical dyssynchrony in patients with CAD and preserved EF without and with a prior MI are shown in Figure 3). On the contrary, in patients with depressed EF, mechanical dyssynchrony was significantly higher in those with a prior MI (Table 3).

Among all CAD patients, those with an anterior MI (n = 118) had slightly lower EF values (45.6 ± 14.2 compared with 52.1 ± 15.8 %; P < 0.05) than others who had inferior MI (n = 90). However, there was no significant difference in Ts-SD (28.7 ± 15.7 compared with 31.8 ± 18.5 ms) and Te-SD (26.6 ± 14.1 compared with 28.5 ± 20.5 ms) between patients with an anterior or inferior MI.

QRS duration and mechanical dyssynchrony

Only 6% (6 out of 94) of patients in the preserved EF group had wide QRS complexes of > 120 ms, as opposed to 30% (65 out of 217) in the depressed EF group ($\chi^2 = 19.2$, P < 0.001) (Figure 4). Among patients with preserved EF, the vast majority of systolic (37 out of 39) and diastolic (16 out of 19) dyssynchrony were in the narrow QRS complexes subgroup. In contrast, among patients with depressed EF, the prevalence of systolic dyssynchrony in patients with wide QRS complexes (52 out of 65) was significantly higher ($\chi^2 = 19.2$, P < 0.001) than those with narrow QRS complexes (71 out of 152). Likewise, diastolic dyssynchrony was more common in patients with wide QRS (50 out of 65) than narrow (33 out of 152) QRS complexes ($\chi^2 = 56.4$, P < 0.001).

DISCUSSION

To our knowledge, this is the largest study to date that has examined the prevalence and patterns of systolic and diastolic dyssynchrony in CAD patients with a wide range of LVEF. The study demonstrated that systolic and diastolic dyssynchrony were highly prevalent even in patients with preserved EF, irrespective of the status of prior MI and QRS duration.

Relationship between myocardial ischaemia and mechanical dyssynchrony in CAD

In our present study, mechanical dyssynchrony was associated with higher angina class and multi-vessel disease, suggesting an important role of myocardial ischaemia in the pathogenesis of mechanical dyssynchrony in CAD. As changes in timing of regional mechanical events may precede local motion abnormalities during myocardial...
ischaemia, mechanical dyssynchrony could be present when LVEF is relatively preserved. This phenomenon has been observed in an animal model by Wang et al. [6]. When resting myocardium was subjected to progressive coronary stenosis, a delayed onset of subendocardial thinning was demonstrated in the early stage of hypoperfusion before the development of regional wall motion abnormalities. In another clinical study, the time to peak systolic velocity measured by TDI was found to be associated with coronary stenosis in patients with chest pain who had no apparent ventricular wall motion abnormalities on echocardiography [13]. In addition, Fan et al. [17] found that mechanical dyssynchrony was affected by regionally stunned myocardium in porcine hearts.

**Relationship between prior MI and mechanical dyssynchrony in CAD**

Another major finding of our present study was that mechanical dyssynchrony was highly prevalent even in the absence of a prior MI. This observation supports
that factors other than the presence of a prior MI, such as ischaemia and stunning, may contribute to mechanical dyssynchrony in CAD patients with preserved EF. In addition, it highlights that mechanical dyssynchrony can be present without a regional wall motion abnormality on resting echocardiography. The lack of accuracy of the visual assessment of regional wall motion dyssynchrony has been reported previously by Kvitting et al. [18]. In contrast, TDI allows quantitative evaluation of mechanical dyssynchrony with high temporal resolution and is more objective.

The relationship between prior MI and mechanical dyssynchrony also appeared to be different in patients with preserved and depressed EF. In the present study, the presence of a prior MI worsened mechanical dyssynchrony in patients with depressed EF, a finding consistent with previous reports by Zhang et al. [19] and Fahmy Elnoamany et al. [20]. However, among patients with preserved EF, the status of a prior MI apparently did not alter the degree of mechanical dyssynchrony. The present study was not intended to explore the mechanisms contributing to the difference, although this might be related to the larger infarct size or scar tissues as well as more severe peri-infarct ischaemia in patients with depressed EF, resulting in worsening of regional dyssynchrony, or alternatively the reduced EF is a marker of more severe myocardial ischaemia, which has a contributory role to mechanical dyssynchrony.

**Comparison with previous studies**

In 138 heart failure patients (60 patients with preserved EF), De Sutter et al. [21] found a relatively low
prevalence (17%) of systolic dyssynchrony in patients with preserved EF, but the prevalence was higher in patients with a QRS duration > 120 ms (50%). In that particular study, 20% of the patients had non-ischaemic cardiomyopathy. Moreover, the use of pulse-wave TDI in only four basal left ventricle segments in that study [12] is likely to have underestimated the prevalence of mechanical dyssynchrony.

It has been reported previously that systolic and diastolic dyssynchrony occurred in 36 and 39% respectively, of patients with heart failure and normal EF [4]. In the present study, however, the prevalence of diastolic dyssynchrony in patients with CAD and preserved EF appeared to be lower (20%), although the prevalence of systolic dyssynchrony was similar (42%). This discrepancy may be explained by the differences in the clinical characteristics of the patients. In the previous study, all patients had a prior history of heart failure and preserved EF, and among them 62% had a non-ischaemic aetiology. It is possible that, in patients with diastolic heart failure, the more impaired LV filling may have induced diastolic dyssynergy, resulting in a higher prevalence of diastolic dyssynchrony.

Limitations
The present study has several limitations. Only longitudinal myocardial motion and dyssynchrony were examined in the present study, whereas radial and circumferential motions were not evaluated. However, as subendocardial muscle fibres are oriented longitudinally [22] and are more vulnerable to ischaemia than the circumferentially arranged mid-wall fibres, evaluation of longitudinal motions would be a sensible measure to characterize mechanical dyssynchrony in CAD. The reproducibility and usefulness of TDI in the evaluation of mechanical dyssynchrony has been questioned recently [23]. However, we believe that the technique is highly dependent on the adequate training of operators and its reproducibility remains high in experienced laboratories [24]. Although all patients in the present study had chest pain and most had angiographic or ECG evidence of CAD, myocardial ischaemia was documented by functional stress testing in only few patients.

Conclusions
Mechanical dyssynchrony has been shown to be associated with impaired LV diastolic filling in CAD with preserved systolic function [25] and to be an independent predictor of morbidity and mortality in patients with depressed EF [26]. The present study has demonstrated a high prevalence of both systolic and diastolic dyssynchrony in patients with CAD and preserved EF. Unlike those with depressed EF, mechanical dyssynchrony in patients with preserved EF mainly occurs in those with narrow QRS complexes and was independent of the presence of a prior MI. Nonetheless, both systolic and diastolic dyssynchrony were more prevalent in CAD patients with depressed EF.

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