Heart Failure

Severe Left Ventricular Dyssynchrony Is Associated With Poor Prognosis in Patients With Moderate Systolic Heart Failure Undergoing Coronary Artery Bypass Grafting

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Objectives
The objective of the present study was to assess the relationship between the presence of left ventricular (LV) dyssynchrony and clinical outcome in patients with moderate systolic heart failure undergoing coronary artery bypass graft (CABG) surgery.

Background
The presence of LV dyssynchrony is associated with poor prognosis in patients with LV dysfunction.

Methods
The study consisted of 215 consecutive patients with ischemic cardiomyopathy and dyspnea (age 65 ± 9 years, 81% male) undergoing CABG. Dyssynchrony was calculated by tissue Doppler imaging from regional time intervals in basal LV segments before and 1 month after CABG. Myocardial viability was assessed using single-photon emission computed tomography (SPECT) before CABG.

Results
Twenty-five patients (11.6%) died within 30 days (in-hospital mortality) of CABG. The presence of pre-CABG dyssynchrony ≥119 ms had the highest predictive accuracy for in-hospital mortality, with a sensitivity of 84% and a specificity of 71%. During the median follow-up period of 359 days (interquartile range 219 to 561), an additional 19 patients (10.3%) died and 34 patients (18.5%) were hospitalized for worsening heart failure. At Cox regression analysis, post-CABG dyssynchrony ≥72 ms and ≥5 viable segments were identified as independent predictors of clinical events, with a hazard ratio (HR) of 5.02, 95% confidence interval (CI) 2.57 to 10.02 (p < 0.001), and an HR of 0.63, 95% CI 0.55 to 0.75 (p < 0.001), respectively. Patients without post-CABG dyssynchrony and with viable myocardium had excellent prognosis compared with patients with severe post-CABG dyssynchrony and nonviable myocardium (event rate 3% vs. 64%; p < 0.001).

Conclusions
The presence of severe LV dyssynchrony is associated with poor clinical outcomes despite revascularization. These results advocate a routine assessment of both LV dyssynchrony and viability to predict outcome in systolic heart failure patients undergoing CABG surgery. (J Am Coll Cardiol 2007;50:1315–23) © 2007 by the American College of Cardiology Foundation

Coronary artery bypass graft (CABG) surgery improves prognosis in patients with ischemic cardiomyopathy and angina (1–2). In contrast, in patients with left ventricular (LV) dysfunction who have dyspnea as the main symptom, the role of surgical revascularization is poorly defined (3). These patients are scarce in clinical trials, and it is controversial whether they benefit from revascularization. Observational studies (4) have emphasized the need for detection of myocardial viability to identify patients with reversible dysfunction, in whom survival may improve after CABG surgery. However, a significant number of patients with a large area of viable myocardium do not experience functional and prognostic improvement despite CABG surgery (5). In contrast, several studies have reported a survival benefit of postsurgical revascularization, irrespective of the degree of viability (6), or recovery of left ventricular ejection fraction (LVEF) (7). This suggests that, in patients with
heart failure, the assessment of myocardial viability alone is not accurate enough to predict outcome after revascularization and that other variables may be important. Cardiac dyssynchrony is associated with functional deterioration and poor prognosis in advanced systolic heart failure (8–11). Correction of dyssynchrony with biventricular pacing improves symptoms, LVEF, and survival (12). The objective of the present study was to assess the prevalence of pre- and post-CABG LV dyssynchrony and its relationship to clinical outcome in patients with stable ischemic cardiomyopathy and dyspnea as the predominant symptom.

Methods

Patients. This was a prospective multicenter study. Between June 2002 and December 2005, 215 consecutive patients (age 65 ± 9 years, 81% male) with ischemic cardiomyopathy, undergoing CABG surgery, who fulfilled the inclusion/exclusion criteria were recruited to the study. Inclusion criteria included: 1) effort dyspnea (New York Heart Association [NYHA] functional class I, II, or III) as the main cardiovascular symptom for at least 3 months; and 2) stable LV dysfunction with LVEF <40% for at least 3 months. Patients with NYHA functional class IV symptoms during the 30-day period before CABG surgery, acute coronary syndrome in the previous 3 months, any valvular heart disease requiring surgery, malignancy, sustained ventricular tachycardia, or survivors of cardiac arrest were excluded from the study. All patients recruited to the study had CABG surgery as the sole procedure. No patients underwent concomitant LV remodeling, aneurysmectomy, mitral valve repair, or Maze procedure. The study was approved by the ethical committee of each institution. All patients gave written informed consent before recruitment.

Study protocol. In the week leading up to CABG surgery, each patient underwent echocardiography and tissue Doppler imaging (TDI) to assess LV volumes, LVEF, and pre-CABG LV dyssynchrony. In addition, myocardial perfusion and glucose uptake were assessed by single-photon emission computed tomography (SPECT) using technetium-99m tetrofosmin and F18-fluorodeoxyglucose, respectively. The TDI was performed 1 month after surgery to record post-CABG dyssynchrony. At 6-month follow-up, echocardiography was repeated to assess LV volumes and LVEF.

Echocardiography and TDI. All studies were performed with a commercially available system equipped with TDI (Vivid 7, Vingmed-General Electric, Horten, Norway). The LV volumes and LVEF were assessed in apical 4- and 2-chamber views using the biplane Simpson method. The TDI was performed in pulsed wave mode. In 3 apical views (4-, 3-, and 2-chamber), longitudinal myocardial velocities were recorded in 6 basal segments of the LV. Moreover, peak mitral annular velocities during systole (Sm) and early diastole (Em) were assessed as the mean from 4 corners of the mitral annulus (septal, lateral, anterior, and inferior). Sample volume was placed in the middle of each basal segment. Gain and filters were adjusted to obtain an optimal tissue signal. Myocardial velocities were recorded at end-expiration at a sweep speed of 100 mm/s. All studies were stored both in digital (raw data) format and on S-VHS videotape for off-line analysis. The mean from 3 consecutive beats was taken for each measurement. Echocardiographers were blinded to clinical follow-up data.

Assessment of LV dyssynchrony by TDI. To assess the pre- and post-CABG LV dyssynchrony, time delay between the onset of QRS complex on the surface electrocardiogram and the onset of the systolic velocity wave on the TDI recording was assessed in each basal LV segment. Dyssynchrony was calculated as the difference between the shortest and the longest time delay in the 6 basal segments. Thus, LV dyssynchrony represents a delay in the onset of contraction between the segment with the earliest and the segments with the latest systolic wall motion (13). Intra- and interobserver variability for the assessment of LV dyssynchrony were 7.1% and 8.2%, respectively.

Assessment of viability by SPECT. In brief (5), technetium-99m tetrofosmin (600 MBq) was injected intravenously to evaluate resting perfusion. After a light meal and administration of acipimox, F18-fluorodeoxyglucose (185 MBq) was injected intravenously to assess myocardial glucose uptake. Dual-isotope simultaneous image acquisition was performed 45 min after F18-fluorodeoxyglucose injection using high-energy 511-keV collimators. A symmetrical 15% energy window was preset on each side of the 140-keV photon peak of technetium-99m tetrofosmin and 511-keV photon peak of F18-fluorodeoxyglucose. Data were acquired over 360° and stored in a 64 × 64 computer matrix. The images were displayed as polar maps, which were normalized to maximum activity (set at 100%). To assess myocardial viability, polar maps were divided into 16 segments. Segments showing normal perfusion of technetium-99m tetrofosmin and segments with perfusion defect but preserved or increased F18-fluorodeoxyglucose (perfusion-metabolism mismatch) were considered to be viable. Segments with a match (concordantly reduced perfusion and metabolism) were considered to be nonviable.

Statistical analysis. Data are presented as mean ± SD or median and interquartile range (IQR). Two-sided paired and unpaired Student t test or Pearson correlation coefficient were used as appropriate. The Fisher exact test was used to compare categoric variables in 2 × 2 contingency table. In cases where the contingency table had more than 2 rows or 2 columns, the chi-square test was used. Receiver-
operating characteristic (ROC) curves were constructed to assess optimal cutoff values for LV dyssynchrony and the number of dysfunctional viable segments required to predict clinical events. Independent predictors of death from any cause and hospitalization for worsening heart failure were identified using the Cox proportional hazard model and expressed as a hazard ratio (HR) and 95% confidence interval (CI). Cumulative survival curves for composite of death from any cause and hospitalization for worsening heart failure were derived according to the Kaplan-Meier method, and differences between curves were analyzed by log-rank statistics. For all tests, \( p < 0.05 \) was considered to be significant. All analyses were conducted using SPSS software (version 13, SPSS Inc., Chicago, Illinois).

**Results**

**Thirty-day outcome.** A total of 25 patients (11.6%) died within 30 days after CABG surgery. The causes of deaths were refractory heart failure in 23 patients and sepsis in 2 patients. Table 1 shows baseline characteristics in the 30-day survivors and nonsurvivors. Patients had on average 2.5 ± 0.8 significantly stenosed coronary arteries. Complete revascularization of all stenosed lesions was obtained in 189 (88%) of patients.

Patients who died within 30 days after surgery had significantly greater pre-CABG LV dyssynchrony and a smaller area of viable myocardium than survivors (\( p < 0.001 \)) (Fig. 1). Other baseline clinical and echocardiographic variables, including the EuroSCORE, were similar in both groups. At ROC analysis, the presence of pre-CABG surgery dyssynchrony of ≥119 ms (sensitivity 84%, specificity 71%) and ≤ 5 dysfunctional viable segments (sensitivity 72%, specificity 61%), showed the highest predictive accuracy for in-hospital mortality (Fig. 2). The prevalence of severe (≥119 ms) pre-CABG dyssynchrony in the entire cohort was 36%. Twenty-one (27%) of 77 patients with severe pre-CABG dyssynchrony died compared with only 4 (3%) of 138 patients without significant dyssynchrony (\( p < 0.001, \) Fisher exact test).

**Outcome from day 31 to the end of the follow-up period.** Six patients (3.2%) who had an acute coronary syndrome during this follow-up period were excluded. Thus, a total of 184 patients (age 66 ± 9 years, 84% male) who survived the first 30 days after CABG surgery entered the analysis. During median follow-up of 359 days (IQR 219 to 561 days), an additional 19 patients (10.3%) died and 34 patients (18.5%) were hospitalized for worsening heart failure. All of the observed deaths were cardiovascular (9

<table>
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<td>Nonsurvivors (n = 25)</td>
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<tr>
<td>Age, yrs</td>
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<tr>
<td>Male gender, n (%)</td>
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<tr>
<td>Diabetes mellitus, n (%)</td>
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<tr>
<td>Stenosed coronary arteries, n</td>
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<tr>
<td>ACE inhibitors, n (%)</td>
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<tr>
<td>Beta-blockers, n (%)</td>
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<tr>
<td>Loop diuretics, n (%)</td>
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<td>Amiodarone, n (%)</td>
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<td>Angina pectoris, n (%)</td>
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<tr>
<td>NYHA functional class</td>
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<tr>
<td>Duration of heart failure, yrs</td>
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<tr>
<td>Logistic EuroSCORE (%)</td>
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<td>Atrial fibrillation, n (%)</td>
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<td>QRS duration, ms</td>
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<tr>
<td>Left bundle branch block, n (%)</td>
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<tr>
<td>LV end-diastolic volume, ml</td>
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<tr>
<td>LV end-systolic volume, ml</td>
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<tr>
<td>LVEF, %</td>
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<tr>
<td>Pre-CABG mitral regurgitation, n (%)</td>
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<tr>
<td>Moderate (grades 2/4 and 2+/4)</td>
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<td>Sm, cm/s</td>
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<tr>
<td>Em, cm/s</td>
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<tr>
<td>Pre-CABG LV dyssynchrony, ms</td>
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<tr>
<td>Severe (≥119 ms) pre-CABG dyssynchrony, n (%)</td>
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<td>Dysfunctional viable segments, n (%)</td>
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</table>

ACE = angiotensin-converting enzyme; CABG = coronary artery bypass grafting; Em = peak early diastolic mitral annular velocity; IQR = interquartile range; LV = left ventricular; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; Sm = peak early systolic mitral annular velocity.
sudden cardiac and 10 pump failure deaths). No patient was lost to follow-up, and survival status was established in all individuals. Table 2 shows baseline and 1-month post-CABG surgery clinical and echocardiographic characteristics in patients with and without clinical events. Patients with a clinical event were older than patients without an event. In addition, patients with clinical events had smaller post-CABG Sm and Em ($p < 0.05$), greater pre- and post-CABG dyssynchrony ($p < 0.001$), and less viable myocardium ($p < 0.001$) than patients without clinical events. Other clinical variables and degree of LV remodeling were similar between groups.

**Independent predictors of death from any cause and hospitalization for worsening heart failure between day 31 to the end of the follow-up period.** The ROC curves were constructed to analyze the accuracy of the 3 baseline variables with the greatest difference between the groups to predict clinical events (Fig. 3). Post-CABG dyssynchrony, with a cut-off value of $\geq 72$ ms ($n = 77$ patients), showed the highest accuracy to predict clinical events. The accuracy of the number of dysfunctional but viable segments (cut-off value $\geq 5$ segments [$n = 88$ patients]) and pre-CABG dyssynchrony (cut-off value $\geq 72$ ms [$n = 94$ patients]) were lower than post-CABG dyssynchrony. In Cox regression analysis (Table 3), post-CABG LV dyssynchrony $\geq 72$ ms and $\geq 5$ dysfunctional but viable segments were identified as the independent predictors of all-cause death and heart failure hospitalization. Table 4 and Figure 4 show clinical outcome in 4 groups of patients divided according to the degree of post-CABG dyssynchrony ($\geq 72$ vs. $< 72$ ms) and extent of viable myocardium ($\geq 5$ viable segments vs. $< 5$ viable segments). Patients with significant post-CABG dyssynchrony and a small extent of viable myocardium had the highest event rate (64%) and significantly shorter event-free survival ($p < 0.001$) compared with the 3 other groups. In contrast, patients without post-CABG dyssynchrony and with a large extent of viable myocardium had excellent outcome, with an event rate of 3%, and significantly longer event-free survival ($p < 0.001$) than the other groups. Intermediate event rates were observed in patients without post-CABG dyssynchrony but with small extent of viable myocardium and in patients with significant post-CABG dyssynchrony but large extent of viable myocardium.
Impact of surgery on LV dyssynchrony. The prevalence of significant (≥72 ms) pre- and post-CABG dyssynchrony was 51% and 42%, respectively. The pre-CABG dyssynchrony correlated significantly with the post-CABG dyssynchrony (r = 0.73; r² = 0.53; p < 0.001). Twenty-seven (29%) of 94 patients with significant pre-CABG surgery dyssynchrony (≥72 ms) showed minor dyssynchrony (<72 ms) after CABG, and none of these patients experienced any clinical events during the follow-up period. Ten (11%) of 90 patients without pre-CABG dyssynchrony developed significant dyssynchrony after CABG. Of note, all of these patients had nonviable myocardium. Relationship of post-CABG LV dyssynchrony with functional and clinical outcome. Table 5 shows baseline and 6-month follow-up characteristics of patients with (n = 77) and without (n = 107) significant (≥72 ms) post-CABG dyssynchrony. The baseline clinical variables and degree of LV remodeling were similar in both groups. Patients with significant post-CABG dyssynchrony had a smaller extent of viable myocardium and greater pre-CABG dyssynchrony than patients without post-CABG dyssynchrony. At the 6-month follow-up, patients without post-CABG dyssynchrony showed superior functional and clinical outcome compared with patients with significant post-CABG dyssynchrony. Both groups showed a significant reduction in angina and NYHA functional class. However, greater improvement (p < 0.01) was observed in patients without post-CABG dyssynchrony. The LVEF also increased in both groups, but significantly more (p < 0.001) in patients without post-CABG dyssynchrony. The LV volumes decreased only in patients without post-CABG dyssynchrony. Post-CABG dyssynchrony showed a significant correlation with the change in both LV end-systolic volume (r = 0.30; p < 0.001) and LVEF (r = −0.51; p < 0.001) between baseline and 6-month follow-up. Furthermore, a higher percentage of patients without post-CABG dyssynchrony felt improved by surgery compared with patients with residual post-CABG dyssynchrony (p < 0.01, Fisher exact
test). Finally, 42 clinical events (55%; 16 deaths) occurred in 77 patients with significant post-CABG dyssynchrony compared with only 11 events (10%) in 107 patients without post-CABG dyssynchrony ($p < 0.001$, Fisher exact test). Median event-free survival was significantly longer in patients without post-CABG dyssynchrony ($p < 0.01$).

**Discussion**

The present study investigated the prevalence of significant pre- and post-CABG LV dyssynchrony and its relationship to clinical outcome in 215 patients with stable ischemic heart failure undergoing CABG surgery. Main findings can be summarized as follows: 1) severe pre-CABG dyssynchrony was present in approximately one-third of the patients, and its presence was associated with increased in-hospital mortality; 2) CABG surgery alone was insufficient to resynchronize the LV contraction pattern in the majority of patients, although patients who experienced a major reduction in dyssynchrony after CABG surgery had superb long-term prognosis; and 3) post-CABG dyssynchrony and myocardial viability emerged as independent predictors of long-term survival and hospitalization for heart failure, with dyssynchrony being the best predictor. Patients without LV dyssynchrony, both before and after CABG surgery, and with a large segment of viable myocardium had excellent 30-day and long-term prognosis. In contrast, patients with severe dyssynchrony had a poor outcome irrespective of degree of viability. These results advocate routine assessment for LV dyssynchrony, in addition to myocardial viability, in patients with systolic heart failure undergoing CABG.

**Revascularization in systolic heart failure.** Ischemic heart failure is a leading cause of hospitalization in developed countries. Because the outcome of medical therapy remains unsatisfactory (4,14), other therapeutic modalities such as revascularization or resynchronization have to be considered. Myocardial revascularization improves survival in patients with chronic coronary artery disease and potentially reversible LV dysfunction (3). Recent meta-analyses have demonstrated a strong relationship between the presence of myocardial viability and improved survival after CABG surgery (4,14). Absence of viability was associated with a

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<th>HR by Cox Regression Analysis for a Composite of Death From Any Cause or Hospitalizations for Worsening Heart Failure From Day 31 After CABG to the End of Follow-Up Period</th>
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<tr>
<td></td>
<td>Univariable Analysis</td>
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<tr>
<td></td>
<td>HR</td>
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<tr>
<td>Age</td>
<td>1.16</td>
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<tr>
<td>Male gender</td>
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<tr>
<td>Diabetes mellitus</td>
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<td>1.10</td>
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<tr>
<td>Pre-CABG QRS duration</td>
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<tr>
<td>Pre-CABG LV end-diastolic volume</td>
<td>0.99</td>
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<tr>
<td>Pre-CABG LV end-systolic volume</td>
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<tr>
<td>Pre-CABG LVEF</td>
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<tr>
<td>Post-CABG Sm</td>
<td>0.86</td>
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<tr>
<td>Post CABG Em</td>
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<tr>
<td>Post-CABG LV dyssynchrony $\geq$ 72 ms</td>
<td>6.58</td>
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<tr>
<td>$\geq$ 5 dysfunctional viable segments</td>
<td>0.50</td>
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Post-CABG indicates 1 month after CABG. CI = confidence interval; HR = hazard ratio; other abbreviations as in Table 1.

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<tr>
<th>Table 4</th>
<th>Clinical Outcome From Day 31 After CABG to End of Follow-Up Period in 4 Groups of Patients According to Degree of Post-CABG Dyssynchrony and Myocardial Viability</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Death From Any Cause (n = 19)</td>
</tr>
<tr>
<td>Post-CABG dyssynchrony $&lt;$ 72 ms and $\geq$ 5 viable segments (n = 63)</td>
<td>0 (3%)</td>
</tr>
<tr>
<td>Post-CABG dyssynchrony $&lt;$ 72 ms and $\geq$ 5 viable segments (n = 44)</td>
<td>3 (7%)</td>
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<tr>
<td>Post-CABG dyssynchrony $\geq$ 72 ms and $\geq$ 5 viable segments (n = 25)</td>
<td>2 (8%)</td>
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<tr>
<td>Post-CABG dyssynchrony $\geq$ 72 ms and $\geq$ 5 viable segments (n = 52)</td>
<td>14 (27%)</td>
</tr>
<tr>
<td>p value (chi-square test)</td>
<td>$&lt;$0.001</td>
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CABG = coronary artery bypass graft.
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Table 5 Baseline and Follow-Up Characteristics in Patients With and Without Significant Post-CABG LV Dyssynchrony

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<th>Post-CABG Dyssynchrony $\geq$72 ms (n = 77)</th>
<th>Post-CABG Dyssynchrony $&lt;$72 ms (n = 107)</th>
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<td></td>
<td>Baseline (n = 77)</td>
<td>Follow-Up (n = 72)*</td>
</tr>
<tr>
<td>Angina pectoris, n (%)*</td>
<td>43 (56)</td>
<td>19 (26)</td>
</tr>
<tr>
<td>NYHA functional class*</td>
<td>2.5 ± 1.0</td>
<td>2.0 ± 1.6</td>
</tr>
<tr>
<td>LV end-diastolic volume, ml*</td>
<td>155 ± 35</td>
<td>149 ± 40</td>
</tr>
<tr>
<td>LV end-systolic volume, ml*</td>
<td>112 ± 36</td>
<td>106 ± 51</td>
</tr>
<tr>
<td>LVEF, %*</td>
<td>31 ± 6</td>
<td>34 ± 11</td>
</tr>
<tr>
<td>Dysfunctional viable segments, n (%)</td>
<td>4.9 ± 3.6</td>
<td>NA</td>
</tr>
<tr>
<td>LV dyssynchrony, ms#</td>
<td>143 ± 60</td>
<td>126 ± 34</td>
</tr>
<tr>
<td>Improved by CABG, n (%)*</td>
<td>43 (56)</td>
<td>NA</td>
</tr>
<tr>
<td>Death from any cause, n (%)</td>
<td>16 (21)</td>
<td>NA</td>
</tr>
<tr>
<td>Heart failure hospitalization, n (%)</td>
<td>26 (34)</td>
<td>NA</td>
</tr>
<tr>
<td>Composite of death from any cause and heart failure hospitalization, n (%)</td>
<td>42 (55)</td>
<td>NA</td>
</tr>
<tr>
<td>Median event-free survival, days</td>
<td>326 (IQR 205–479)</td>
<td>NA</td>
</tr>
</tbody>
</table>

*The 6-month functional follow-up data are reported in 72 patients with significant post-CABG dyssynchrony (≥72 ms) and 106 patients without post-CABG dyssynchrony (<72 ms) who survived the first 6 months after CABG. †p < 0.05, ‡p < 0.01, §p < 0.001: follow-up patients with versus without significant post-CABG dyssynchrony. #Follow-up (post-CABG) LV dyssynchrony was assessed within 1 month after CABG (see Methods section).

Conclusions

The presence of severe pre- and post-CABG LV dyssynchrony was associated with high in-hospital and long-term mortality in patients with ischemic heart failure undergoing myocardial revascularization. Therefore, noninvasive testing to assess LV dyssynchrony should be performed before CABG surgery to guide patient selection. After CABG surgery, assessment of LV dyssynchrony should be repeated to optimize patient management. High-risk patients with severe dyssynchrony might be considered for biventricular pacing before or after CABG surgery. These results call for a prospective study to investigate the effects of cardiac resynchronization therapy in patients with heart failure and severe LV dyssynchrony undergoing CABG surgery.

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