

Deterioration of Regional Wall Motion Immediately after Coronary Artery Bypass Graft Surgery Is Associated with Long-term Major Adverse Cardiac Events

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Background: Patients undergoing coronary artery bypass graft (CABG) surgery frequently develop wall motion abnormalities diagnosed by intraoperative transesophageal echocardiography. However, the relation between deterioration in wall motion and postoperative morbidity or mortality is unclear. Therefore, the authors hypothesized that deterioration in intraoperative left ventricular regional wall motion immediately after CABG surgery is associated with a higher risk of adverse cardiac events.

Methods: With institutional review board approval, data were gathered from 1,412 CABG surgery patients. Echocardiographic wall motion score (WMS) was derived using a 16-segment model. Outcomes data were gathered for up to 2 yr after surgery. The primary outcome, major adverse cardiac event, was a composite index of myocardial infarction, need for subsequent coronary revascularization, or all-cause mortality during the follow-up period.

Results: Two hundred twenty-one patients (16%) had 254 primary outcome events during follow-up. Postbypass WMS did not change in 812 patients (58%), deteriorated in 219 patients (16%), and improved in 368 patients (26%). Kaplan-Meier analysis showed that patients with deterioration in WMS after CABG experienced significantly lower major adverse cardiac event-free survival than patients with either no change or improvement in WMS ($P = 0.004$). Cox proportional hazards regression modeling revealed a significant association between deterioration in WMS and the composite adverse outcome (hazard ratio, 1.47 [1.06–2.03]; $P = 0.02$).

Conclusions: The authors confirmed their hypothesis that deterioration in wall motion detected by intraoperative echocardiography after CABG surgery is associated with increased risk of long-term adverse cardiac morbidity. Worsening wall motion after CABG surgery should be considered a prognostic indicator of adverse cardiovascular outcome.

PATIENTS undergoing cardiac surgery frequently develop segmental wall motion abnormalities (WMAs) diagnosed by transesophageal echocardiography (TEE). In

the setting of coronary artery bypass graft (CABG) surgery with cardiopulmonary bypass (CPB), WMAs found after revascularization are multifactorial and dynamic in nature. Although the association of hibernating myocardium detected by dobutamine stress echocardiography or nuclear imaging with adverse outcomes after CABG surgery has been reported, little is known about the extent to which new intraoperative WMAs are predictive of morbidity and mortality after CABG surgery. In a study by Leung *et al.*,¹ post-CPB episodes of worsening wall motion were found to be associated with an increased incidence of myocardial infarction (MI), cardiogenic shock, or death during the period of hospitalization. Similarly, Comunale *et al.*² showed that WMAs occurring at any point intraoperatively in CABG patients conferred an increased risk of developing an early postoperative MI. Both of these studies, however, were limited by the use of short-term outcomes and a single four-segment transgastric midpapillary view of the left ventricle.

Because segmental WMAs have repeatedly been shown to predict long-term mortality in the setting of MI,^{3–5} the current study was designed to characterize the long-term prognostic value of postoperative WMAs in a sample of CABG patients using a 16-segment⁶ wall-motion model. We hypothesized that deterioration in left ventricular regional wall motion immediately after surgical coronary revascularization would be associated with a higher risk of adverse cardiac events.

Materials and Methods

Patient Selection

The study was approved by the institutional review board of Duke University Medical Center (Durham, North Carolina) as a retrospective data review with a waiver of consent. Detailed clinical and TEE data were gathered from prospectively entered databases in all patients older than 18 yr undergoing isolated, primary CABG with CPB between September 1, 2000, and December 31, 2002 at Duke University Medical Center. Patients undergoing redo-sternotomy or CABG combined with valvular or other surgery were excluded. Those on preoperative inotropes or dependent on ventricular pacing and/or intraaortic balloon counterpulsation were also excluded from the study because their baseline wall motion score (WMS) probably did not

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reflect their true baseline. Anesthesia and surgery were managed per institutional protocols and have been described elsewhere.⁷

Data Sources and Collection

Clinical data were gathered from the Duke Databank for Cardiovascular Diseases, a large, quality-assured data repository for patients undergoing cardiovascular procedures at Duke University Medical Center that has been previously described.⁸ Perioperative clinical data gathered included age, sex, preoperative ejection fraction, left main occlusion greater than 90%, presence of congestive heart failure, unstable angina, diabetes, chronic obstructive pulmonary disease, dialysis dependence or MI within 7 days, and aortic cross clamp time. These were used to calculate a modified score based on the index developed and validated by Hannan *et al.*^{9,10} for each patient. To determine postoperative duration of hospital stay while taking into account the mortality rates, we calculated the number of hospital-free days within 30 days after CABG surgery.¹¹ Patients who died were given a score of 0 hospital-free days.

Follow-up was conducted by the Duke Clinical Research Institute Follow-up Services Group, which is responsible for collecting annual follow-up mortality data and nonfatal endpoint information for the Duke Databank for Cardiovascular Diseases. The annual surveys collect data on general health, hospitalizations, MI, stroke, cardiac procedures, and medication use. Patients are surveyed 6 months after an index visit and yearly thereafter with a mailed, self-administered survey with a phone-administered survey to nonresponders. Follow-up is 95% complete for mortality, and patients who are lost to follow-up (2%) or who have asked to be withdrawn (3%) are submitted for an annual search of the National Death Index. Death information is collected from next-of-kin interviews; hospital discharge summaries; death certificates; and *International Classification of Diseases*, 10th Revision, cause of death provided from the National Death Index. Cause of death is assigned after agreement from independent reviews by a death committee. Specific outcomes for this study were monitored for a period of up to 2 yr after initial surgery in all patients and included MI, need for subsequent revascularization, and all-cause mortality. The Duke Databank for Cardiovascular Diseases uses the Society of Thoracic Surgeons data definitions (version 2.41 during the study period), which defines postoperative MI if two of the following four criteria are met: (1) prolonged (> 20 min) typical chest pain not relieved by rest and/or nitrates; (2) elevation of either creatinine kinase-isoenzyme MB by more than 5% of total creatinine kinase, creatinine kinase greater than 2× normal, lactate dehydrogenase subtype 1 greater than lactate dehydrogenase subtype 2, or troponin greater than 0.2 µg/l; (3) new postoperative WMAs; (4) serial electrocardiograms (at least two) show-

ing changes from baseline or serially in ST-T and/or Q waves that are 0.03 s in width and/or greater than or equal to one third of the total QRS complex in two or more contiguous leads. Subsequent revascularization was defined as the need for percutaneous or surgical coronary intervention during the follow-up period.

Echocardiographic data were obtained from the Duke intraoperative TEE database that contains prospectively collected intraoperative TEE data for more than 7,000 patients.

Echocardiographic Assessment of Wall Motion

Intraoperative TEE was performed using a multiplane, phased array TEE probe (T6210 Omniplane II transducer; Phillips Medical Systems, Andover, MA). Images were digitally acquired on a Phillips Sonos 5500 or 7500 Ultrasound Imaging System (Phillips Medical Systems) and stored for off-line analysis. A comprehensive TEE evaluation was performed on each patient after induction (before sternotomy) and once again after successful separation from CPB (before chest closure) according to the sequence and number of views recommended by the American Society of Echocardiography–Society of Cardiovascular Anesthesiologists guidelines.⁶ A one-beat loop was recorded unless the heart rate was greater than 90–100 beats/min or during electrical diathermy interference, in which case a two-second loop was recorded. Loops were recorded for each standardized view. Left ventricular wall motion assessed by TEE was graded in a 16-segment model. For each segment, scores were assigned as normal = 1, hypokinesis = 2, akinesis = 3, and dyskinesis = 4. Systolic thickening greater than 30% was graded as normal, 10–30% thickening was graded as hypokinesis, less than 10% thickening was graded as akinesis, and paradoxical motion without thickening was graded as dyskinesis.

Definition of Wall Motion Score

A WMS was then derived by summing the scores for each segment and dividing by the total number of segments (WMS of 1 = normal motion in all segments).⁶ A WMS value greater than 1 indicates abnormal wall motion in at least one segment. In cases of postoperative ventricular pacing, septal segments were excluded from both pre- and post-CPB WMS calculations. In cases when only a subset of 16 segments could be visualized, the nonvisualized segments were eliminated from WMS calculation both pre- and post-CPB. When the value for a segment was missing in the preoperative study, the value for the same segment was set to missing for the postoperative study. The same missing data protocol was also followed when postoperative segment values were missing. This ensured that no measurement bias with respect to missing observations confounded comparisons between preoperative and postoperative TEE measurements. Although missing observations may increase the

variance of estimates of mean WMS, we expect our protocol would not introduce bias in measurements of change in mean WMS. Deterioration in wall motion was defined as a post-CPB WMS greater than pre-CPB WMS; similarly, improvement in wall motion was defined as a post-CPB WMS less than pre-CPB WMS. Change in WMS (Δ WMS) between the pre- and post-CPB TEE examinations was also examined as a continuous variable and defined as the difference between post-CPB WMS and pre-CPB WMS expressed as a fraction of pre-CPB WMS: $((\text{post-CPB WMS} - \text{pre-CPB WMS})/\text{pre-CPB WMS})$. For example, if a patient had no WMAs pre-CPB (WMS = 1) and four-segment hypokinesia post-CPB (WMS = 1.25), the Δ WMS would be $(1.25 - 1.0)/1.0 = 0.25$. Therefore, a positive Δ WMS signifies postoperative deterioration in wall motion, whereas a negative value indicates improvement. For missing wall motion data, we assumed that missing segments are missing at random with respect to WMS. Therefore, in the above example, if 4 segments were hypokinetic among 12 available segments, we expect the loss of 4 segments to yield on average 3 of 12 segments exhibiting hypokinesia, resulting in an unchanged post-CPB WMS of 1.25. Deterioration in WMS as a binary variable assigns equal weight to all degrees of deterioration, whereas Δ WMS as a continuous variable assigns greater weight to greater degrees of deterioration.

All TEE studies were performed and dictated by an attending cardiac anesthesiologist in concert with a cardiac anesthesia fellow in a dedicated perioperative echocardiography training program. All attending anesthesiologists had received formal training in perioperative echocardiography. All studies were reviewed off-line by a single, independent, board-certified (National Board of Echocardiography, Raleigh, North Carolina) anesthesiologist (J.P.M.) with more than 10 yr of experience in TEE, who was blinded to the outcome. Because all wall motion assessment was performed by one individual who was overreading the intraoperative examination, concordance with the intraoperative anesthesiologist was not assessed.

Statistical Analysis

Patients were grouped according to whether they showed deterioration in WMS (post-CPB WMS > pre-CPB WMS). Characteristics were compared between groups using the Pearson chi-square test for categorical variables and Wilcoxon rank sum test for continuous variables. The primary outcome measure was defined as major adverse cardiac event (MACE), a composite index of MI, need for subsequent surgical or percutaneous coronary revascularization, or death (all-cause mortality) during the follow-up period. An uncensored observation represented "complete data": A MACE had occurred and the time to MACE after CABG surgery was known. In contrast, the absence of any MACE event during the

follow-up period represented a censored observation. Freedom from MACE was assessed by constructing survival curves in both groups (deterioration *vs.* no deterioration), using the Kaplan-Meier method. Individual MACE-free survival curves were compared using the log-rank test. We used Cox regression methods to remove variability in MACE that is accounted for by the Hannan index of in-hospital mortality.^{9,10} The index includes most traditional risk variables for cardiac-related mortality, such as age, sex, hemodynamic state, ventricular function, extent of coronary disease, and other relevant comorbidities. We chose to use this index, which was developed independently in different patients, to remove the risk of overfitting our data with a high-dimensional multivariable model using concurrent covariates. The relation between WMS and risk of MACE was evaluated using a Cox proportional hazards regression model with Hannan risk score and aortic cross clamp time as covariates. The primary analysis assessed the association of deterioration in wall motion as a binary outcome (post-CPB WMS > pre-CPB WMS) with risk of MACE. A secondary Cox proportional hazards regression analysis was performed to determine whether deterioration in wall motion was independently associated with risk of MACE when represented as a continuous variable (positive Δ WMS). The secondary analysis was considered necessary for a comprehensive assessment of wall motion because, unlike deterioration in WMS as a binary variable, Δ WMS assigns greater weights to greater degrees of deterioration. The effect of WMS on risk of MACE was expressed as the hazard ratio and its 95% confidence interval. Wall motion grading was repeated in a random sample of 50 patients for assessment of intraobserver variability. For purposes of internal validation, we fit the multivariable models to 5,000 bootstrap samples of the data set. The 95% confidence intervals for the hazard ratio of deterioration in WMS and of change in mean WMS were obtained by bootstrap resampling. For each bootstrap sample, patient records were randomly sampled from the data set with replacement and a Cox proportional hazard model was fit to the sample. A point estimate of the hazard ratio was obtained for each bootstrap sample. A 95% confidence interval for the hazard ratio for WMS was computed from the 2.5 and 97.5 percentiles of the hazard ratio distribution resulting from 5,000 bootstrap samples. Analyses were conducted using SAS (version 9.1; Cary, NC); a two-tailed *P* value less than 0.05 was considered significant.

Results

Of 1,412 patients who met inclusion criteria, 13 patients (0.9%) were lost to follow-up. The remaining 1,399 patients in the study cohort had a mean follow-up time of 451 ± 260 days. There was no significant asso-

Table 1. Characteristics of Patients Exhibiting Deterioration and Patients Exhibiting No Change/Improvement in Wall Motion Score

Patient Characteristic (Total n = 1,399)	Change in Wall Motion Score*		P Value§
	Deterioration (n = 219)	No Change/Improvement (n = 1,180)	
Preoperative			
Mean age ± SD, yr	64.2 ± 11.3	63.5 ± 10.7	0.435
Age >75 yr	37 (17%)	158 (13%)	0.202
Male sex	150 (69%)	849 (72%)†	0.288
Congestive heart failure	9 (4%)	24 (2%)	0.085
Left ventricular ejection fraction >35%	171 (81%)	989 (86%)†	0.057
Myocardial infarction	28 (13%)	171 (15%)	0.530
Diabetes	61 (28%)	295 (25%)	0.398
Hypertension	112 (51%)	585 (50%)	0.713
Previous percutaneous coronary angioplasty	28 (13%)	111 (9%)	0.139
Cross clamp time, min	65 (62–71)	66 (64–67)	0.451
Hannan score × 10	0.23 (0.20–0.29)	0.22 (0.21–0.23)	0.013
Postoperative			
Renal failure	7 (3%)	8 (1%)†	0.004
Stroke	9 (4%)	18 (2%)†	0.017
In-hospital mortality	11 (5%)	13 (1%)	<0.001
Hospital-free days, 30 days after CABG	24 (22–25)‡	25 (23–26)‡	<0.001
MACE	48 (22%)	173 (15%)	0.009

* Values are number of patients with percentage of group total in parentheses, or median with 95% confidence interval for median in parentheses. For age, values are expressed as mean ± SD. † Male sex (n = 1,175), left ventricular ejection fraction >35% (n = 1,148), renal failure (n = 1,179), stroke (n = 1,179). ‡ Values for hospital-free days (30 days after coronary artery bypass graft [CABG]) are expressed as median with interquartile range in parentheses. § P values for comparison of deterioration with no change/improvement in wall motion score; continuous variables compared by Wilcoxon rank sum test, except age compared by t test; binary variables compared by Pearson chi-square test with exact P values.

MACE = major adverse cardiac event.

ciation between deterioration in WMS and loss to follow-up ($P = 0.10$). A description of the study sample is shown in table 1. There were 256 incidents of a MACE in 221 (16%) patients during the follow-up period. Of these 256 events, all-cause mortality accounted for 100 (39%), MI was implicated in 66 (26%), and subsequent need for surgical or percutaneous revascularization was identified in 90 (35%). Within the 100 mortality events, 56 were cardiac causes, whereas 44 cases included medical, trauma-related, or unobserved deaths. Of the 221 patients, 187 had a single event, 33 had two events, and 1 had three events. In the 34 patients who had more than one MACE outcome, time to the first MACE was recorded for the survival analysis. The group with deterioration in WMS had a higher apparent percentage of patients with a MACE outcome compared with the group that showed improvement or had no change in WMS (table 1).

An analysis of missing segment counts showed that 98% of patients had a mean WMS based on 12 or more segments. There were only 33 patients with missing information on more than 4 segments. A majority of patients with missing wall motion data did not have a score in three missing segments. This is likely explained by our scoring protocol, which required that patients with ventricular pacing had septal segment observations (apical, mid, and basal) set to “missing.” WMS did not change in 812 patients (58%), deteriorated in 219 patients (16%), and improved in 368 patients (26%). Although estimates of mean WMS suggest a greater post-

operative increase in nonnormal segments in 34 patients with more than one MACE event than in 187 patients with a single MACE event, the difference was not significant (data not shown).

Kaplan-Meier survival curves showed that patients with worse WMS immediately after CABG had significantly lower probability of MACE-free survival (hazard ratio, 1.59 [1.15–2.19]; $P = 0.004$) compared with patients in whom the WMS did not deteriorate (fig. 1). Cox proportional hazards regression modeling that included aortic cross-clamp time and Hannan risk score as covariates revealed a significant positive association between deterioration in WMS and MACE (hazard ratio, 1.50 [1.06–2.03]; $P = 0.02$; table 2). In the secondary analysis, deterioration in wall motion (positive Δ WMS) as a continuous variable was also significantly associated with risk of MACE (hazard ratio, 1.94 [1.00–3.74]; $P = 0.049$; table 2). In both analyses, Hannan score accounts for a large part of the variability in MACE. Change in WMS remained significantly associated with MACE even after inclusion of Hannan score in the Cox regression model. Bootstrap estimates of a 95% confidence interval for the hazard ratio yielded 1.026–2.047 for deterioration in WMS as a binary response and 0.941–3.902 for change in mean WMS. The bootstrap 95% confidence interval for deterioration in WMS does not include 1, indicating that the significant effect inferred for this binary measure of wall motion is robust to minor variation in the data. For change in mean WMS, the bootstrap

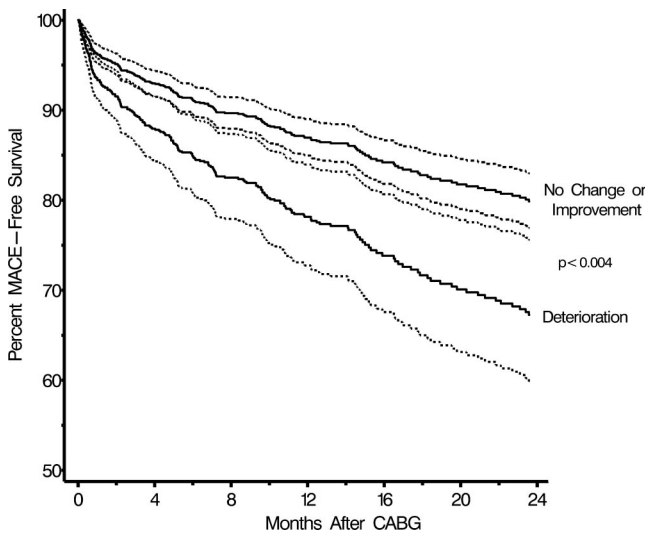


Fig. 1. Kaplan-Meier survival curve showing major adverse cardiac event (MACE)-free survival in the group of patients with deterioration in wall motion score (WMS) compared with the group that showed either no change or improvement in WMS up to 24 months after surgery. *P* value indicates between-group difference. “Deterioration” is represented as a binary variable (post-cardiopulmonary bypass WMS > pre-cardiopulmonary bypass WMS). Hashed lines on either side of the solid line for each group indicate the 95% confidence interval for MACE-free survival. CABG = coronary artery bypass graft.

95% confidence interval is wider and includes 1, suggesting marginal robustness consistent with the *P* value of 0.049 observed for this continuous measure of wall motion. The analyses were repeated with cardiac-only causes of mortality, and similar results were obtained. Intraobserver variability in grading wall motion was 4%.

Discussion

Approximately 500,000 surgical coronary revascularizations are performed annually in the United States¹² and are associated with 1-yr mortality rates of 4–5%.¹³

Table 2. Cox Proportional Hazards Regression Model Showing the Effect of Perioperative Variables on Risk of MACE

Variable	Hazard Ratio (95% Confidence Interval)	<i>P</i> Value
Cox regression model 1		
Deterioration in WMS*	1.47 (1.06–2.03)	0.020
Cross clamp time	1.02 (0.96–1.07)	0.545
Hannan score	1.82 (1.50–2.20)	<0.001
Cox regression model 2		
Deterioration in WMS, ΔWMS†	1.94 (1.00–3.74)	0.049
Cross clamp time	1.01 (0.96–1.07)	0.597
Hannan score	1.83 (1.52–2.21)	<0.001

Hazard ratio for cross clamp time is for a 10-min interval, and hazard ratio for Hannan score is a 0.10-unit increase.

* Deterioration in wall motion score (WMS) is represented as a binary variable in model 1: post-cardiopulmonary bypass (CPB) WMS > pre-CPB WMS.

† Deterioration in WMS is represented as a continuous variable in model 2: ΔWMS = (post-CPB WMS – pre-CPB WMS)/pre-CPB WMS.

MACE = major adverse cardiac event.

The identification of perioperative factors associated with long-term adverse outcome is therefore an important step in reducing this mortality risk. In the current study, we confirmed our hypothesis that deterioration in left ventricular wall motion diagnosed after CPB during CABG surgery was significantly associated with long-term cardiac morbidity and mortality, even after controlling for comorbidities (Hannan index), and a measure of intraoperative ischemic injury (aortic cross clamp time). Our results indicate that worsening of regional wall motion immediately after separation from CPB is prognostic of long-term adverse outcome.

Abnormal myocardial wall thickening has been previously validated as an early and sensitive marker of myocardial ischemia, compared with electrocardiographic and hemodynamic indices.^{14–16} In the nonsurgical MI population, numerous studies^{3,17} have confirmed the strong association between WMAs and mortality up to 7 yr after infarction. This relation was observed even after thrombolytic therapy⁵ or stent implantation after MI.¹⁸ A recent report from the Strong Heart Study group demonstrated that even in a large population of patients without clinically evident cardiovascular disease, WMAs were associated with more than double the risk of long-term cardiovascular events or cardiac death.¹⁹ In the surgical population, Sellier *et al.*²⁰ showed, in a prospective trial of 2,065 subjects designed to risk-stratify post-CABG patients, that a low wall motion index, based on a nine-segment model, defined a group of patients at higher risk for 1-yr death or cardiovascular events (MI, unstable angina, stroke, congestive heart failure) requiring hospitalization (hazard ratio, 1.97; 95% confidence interval, 1.10–3.51). This study did not evaluate any dynamic changes in wall motion, but rather simply assessed baseline postoperative WMAs and summarized them by calculating a WMS. Although the goal of the study by Sellier *et al.* was to develop better prediction of post-CABG adverse outcomes, only patients participating in cardiac rehabilitation and able to perform treadmill stress testing were enrolled. Therefore, even in a relatively healthy group of CABG patients nearly 1 month after surgery, the presence of WMAs predicted a poorer prognosis.

In a study of 50 patients undergoing elective CABG surgery, Leung *et al.*¹ examined the relation between intraoperative echocardiographic (TEE) and electrocardiographic evidence of ischemia and in-hospital adverse cardiac events, defined as MI, ventricular failure, or death. These investigators reported that the incidence of electrocardiographic- and TEE-defined ischemia was highest in the post-CPB period, and whereas electrocardiographic changes were not associated with adverse outcome, post-CPB WMAs were related to adverse clinical outcome. None of the 32 patients without TEE evidence of ischemia had an adverse outcome, whereas one third of patients with TEE changes had adverse

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outcomes ($P = 0.001$). Similarly, using data from an investigational drug trial in 351 patients undergoing CABG surgery, Comunale *et al.*² hypothesized that intraoperative ischemia monitoring would predict which patients were at risk for developing MI within the first 24 h postoperatively. After adjusting for the confounding effects of sex, age older than 70 yr, previous CABG surgery, history of MI, diabetes, unstable angina, and low ejection fraction, they reported that TEE-detected WMAs were an independent predictor of perioperative MI (odds ratio, 2.35 [1.30–4.20]). More recently, Zimarino *et al.*²¹ reported the influence of intraoperative ischemia on long-term MACE in 92 patients undergoing minimally invasive CABG surgery. Intraoperative TEE was used to assess regional wall motion using a 16-segment model. Although multivessel disease was significantly predictive of adverse outcome, intraoperative ischemia was not. In this study, MACE occurred in only 8 patients, precluding a comprehensive assessment of intraoperative ischemia as a prognostic indicator. Our study, using a comprehensive assessment of wall motion in a larger sample of patients with long-term follow-up, supports earlier studies in demonstrating the prognostic importance of intraoperative, new-onset WMAs after coronary revascularization.

The strengths of our study include the use of a 16-segment model for wall motion analysis, low intraobserver variability, a quality-assured perioperative cardiac surgery database, rigorous follow-up data, robust statistical models, and generalizable results. However, our results are limited by the fact that TEE evaluation of WMAs was performed at only one time point before and after revascularization. The hours after CABG surgery represent a labile period during which WMAs may follow a dynamic course.²² Nonetheless, two thirds of our patients did not incur any new WMAs. In addition, our study reflects clinical practice, during which wall motion is likely to be evaluated only once or twice after CPB in a majority of patients. A second limitation is that all TEEs were interpreted offline by a single observer; however, intraobserver variability in grading wall motion was only 4% and any potential bias in interpretation would be equally applied to all subjects in this study. While the Hannan risk index reflects the risk of mortality after CABG surgery, it does not include other covariables such as diabetes and obesity that have also been reported as predictive of adverse postoperative outcome. However, the Hannan risk index was developed from an original set of 40 risk factors that were truncated to the 10 most significant index variables in a stepwise multivariable regression model. Diabetes and obesity are not observed in this final index because their effects on mortality are not as significant as the effects of the other 10 variables. Indeed, obesity is predictive of adverse outcome after CABG surgery only in association with diabetes, whereas the influence of diabetes on postoperative outcome is limited to the subset with poor intraoperative glucose

control.^{23,24} Finally, there are inherent limitations of the retrospective use of a data repository for hypothesis testing. However, despite the retrospective nature of this study, TEE examinations were prospectively interpreted on or shortly after the day of surgery. Furthermore, the hypothesis of this study was prospectively defined at the inception of the Duke intraoperative TEE database as the primary study question to be answered from the database.

In summary, patients with worse regional wall motion after surgical coronary revascularization had a twofold increased risk of death, MI, or need for additional revascularization within the subsequent 2 yr after surgery. Despite the dynamic nature of wall motion immediately after CPB, patients with echocardiographically detected deterioration in wall motion should be considered to be at higher risk for developing subsequent adverse cardiovascular events.

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