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Prognostic Value of Troponin I Levels for Predicting Adverse Cardiovascular Outcomes in Postmenopausal Women Undergoing Cardiac Surgery

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Abstract

BACKGROUND—Adverse cardiac events that follow cardiac surgery are an important source of perioperative morbidity and mortality for women. Troponin I provides a sensitive measure of cardiac injury, but the levels after cardiac surgery may vary between sexes. Our purpose in this study was to evaluate the prognostic value of troponin I levels for predicting cardiovascular complications in postmenopausal women undergoing cardiac surgery.

METHODS—The cohort of this study were women enrolled in a previously reported clinical trial evaluating the neuroprotective potential of 17 β -estradiol in elderly women. In that study, 175 postmenopausal women not receiving estrogen replacement therapy and scheduled to undergo coronary artery bypass graft (with or without valve surgery) were prospectively randomized to receive 17 β -estradiol or placebo in a double-blind manner beginning the day before surgery and continuing for 5 days postoperatively. Serial 12-lead electrocardiograms were performed and serum troponin I concentrations were measured before surgery, after surgery on arrival in the intensive care unit, and for the first four postoperative days. The primary end-point of the present study was major adverse cardiovascular events (MACE) defined as a Q-wave myocardial infarction, low cardiac output state or death within 30 days of surgery. The diagnosis of Q-wave myocardial infarction was made independently by two physicians blinded to treatment and patient outcomes with the final diagnosis requiring consensus. Low cardiac output state was defined as cardiac index $<2.0 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ for $>8 \text{ h}$ regardless of treatment.

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RESULTS—Troponin I levels on postoperative day 1 were predictive of MACE (area under the receiver operator curve = 0.862). A cutoff point for troponin I of >7.6 ng/mL (95% confidence interval, 6.4–10.8) provided the optimal sensitivity and specificity for identifying patients at risk for MACE. The negative predictive value of a troponin I level for identifying a patient with a composite cardiovascular outcome was high (96%) and the positive predictive value moderate (40%). Postoperative troponin I levels were not different between women receiving perioperative 17 β -estradiol treatment compared with placebo and the frequency of MACE was not influenced by 17 β -estradiol treatment.

CONCLUSIONS—In postmenopausal women, elevated troponin I levels on postoperative day 1 are predictive of MACE. Monitoring of perioperative troponin I levels might provide a means for stratifying patients at risk for adverse cardiovascular events.

Operative morbidity and mortality after cardiac surgery are consistently reported to be more frequent for women than for men.^{1–5} A portion of this higher risk can be explained by the older age at which women generally undergo surgery and the associated higher burden of their co-morbid conditions compared with men.⁵ National benchmarking data, however, suggests that female sex is independently associated with mortality after coronary artery bypass graft (CABG) surgery.¹ In that analysis, for any single risk factor, female patients had a statistically higher risk for mortality than did male patients except for the highest risk categories.

Troponin I is a cardiac-specific protein with high sensitivity and specificity for myocardial injury in a number of settings.⁶ Surveillance of troponin levels has become increasingly common after cardiac surgery to identify patients at risk for poor short- and long-term outcomes.^{7–16} However, because of surgery-related cardiac trauma and other perturbations, postoperative serum troponin I levels often far exceed those normally considered as diagnostic for myocardial infarction (MI) in a nonsurgical setting.⁶ Thus, several investigators have proposed troponin I cutoff levels to identify patients at risk for postoperative cardiac complications and mortality.^{7–9,15} Troponin I release after cardiac surgery may differ between men and women, but studies that have examined the predictive value of perioperative troponin I levels have included few women.¹⁷ Consequently, it is presently unknown if increases in troponin I levels after cardiac surgery are associated with adverse cardiovascular events in women.

Previously we reported that low cardiac output state, MI and stroke are the leading causes of mortality for women after cardiac surgery.^{2–4} We have recently evaluated the neuroprotectant potential of 17 β -estradiol in postmenopausal women undergoing cardiac surgery.¹⁸ In that study, perioperative treatment with 17 β -estradiol conferred no benefit for reducing neurological complications compared with placebo. Data on major adverse cardiovascular events (MACE, defined as Q-wave MI, low cardiac output state and all-cause mortality) were collected in that study as a safety end-point to evaluate whether 17 β -estradiol affected the frequency of this outcome compared with placebo. The purpose of this study was to evaluate the prognostic value of troponin I levels for predicting MACE in postmenopausal women after cardiac surgery.

METHODS

One-hundred-eighty-six postmenopausal women undergoing cardiac surgery with cardiopulmonary bypass were enrolled in the trial. Patients were prospectively randomized to receive either 17β -estradiol or placebo perioperatively as previously reported.¹⁸ The study was approved by the IRBs three enrolling sites affiliated with BJC Health Care System, St. Louis, MO. Each participant provided written, informed consent. Patients were excluded for: off-pump surgery; elevated liver function tests or creatinine levels >2 mg/dL; emergency or reoperative surgery; planned use of antifibrinolytics; venous thromboembolism; vaginal bleeding; history of breast or endometrial cancer in the absence of hysterectomy; and preoperative estrogen use within 6 mo of surgery. The patients received transdermal patches containing either 17β -estradiol (Climara, Berlex Laboratories, Wayne, NJ) or placebo (blank patches) in a double-blind manner. Patches were worn from the day before surgery until the fifth postoperative day.

Perioperative Care

Details of the patients' care during and after surgery have been reported.¹⁸ The patients received propofol, fentanyl, midazolam, pancuronium and volatile anesthetic in doses aimed at early tracheal extubation after surgery. Cardiopulmonary bypass was performed with a nonpulsatile system using a membrane oxygenator and a $40\text{-}\mu\text{m}$ in-line arterial filter. Antifibrinolytic drugs were not given due to the unknown potential for prothrombotic interactions between these drugs and 17β -estradiol. Body temperature was maintained between 32°C and 34°C during cardiopulmonary bypass. After surgery, the patients electrocardiograms (ECGs) were monitored continuously with bedside monitoring or telemetry until hospital discharge. Atrial fibrillation was defined as an unusually irregular cardiac rhythm in the absence of P-waves that persisted >1 min regardless of treatment. Twelve-lead ECGs were performed the day before surgery, on arrival in the intensive care unit after surgery, and then daily for the first 4 postoperative days. Blood was obtained to measure serum troponin I concentrations at the same time points. All serum troponin I levels were measured with an enzyme immunoassay (Dade Behring, Newark, DE) in the same central hospital clinical laboratory. The sensitivity of the assay is 0.04 ng/mL, and normal is considered <0.1 ng/mL.

Cardiovascular Complications

Two physicians blinded to treatment and patient outcomes independently reviewed the 12-lead ECGs and the troponin I results. A Q-wave MI was defined as the presence of a new Q-wave >0.04 s or $1/3$ the height of the ECG R-wave on the 12-lead ECG. The diagnosis of a MI was made by consensus; a third cardiologist was consulted when there was disagreement. Low cardiac output state was defined as a cardiac index <2.0 $\text{L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ for >8 h, regardless of treatment. Thermodilution cardiac output was determined using iced saline injection and the average of three measurements recorded. Operative death was defined as that occurring during hospitalization or within 30 days of surgery.

Statistical Analysis

Power analysis for the clinical trial was based on the primary outcome of neurocognitive dysfunction 4–6 wk after surgery as previously described.¹⁸ After the second planned interim analysis, the trial was stopped by the Data and Safety Monitoring Board after enrollment of 187 of a planned 334 women. This decision was based on the unlikelihood that 17 β -estradiol would result in improvements in postoperative neurocognitive function.¹⁸ In this analysis, the primary end-point of the study was the occurrence of MACE at any timepoint after surgery.

Univariate logistic regression models were created with troponin I levels measured on postoperative day 1 considered as a predictor for this outcome. Area under the receiver operator curve (ROC) and corresponding sensitivities, specificities, positive predictive values, and negative predictive values were estimated using optimal troponin I data (Fig. 1). A boot strap simulation was performed in order to estimate the 95% confidence interval for the optimal troponin cut point by creating 1000 random samples, chosen with replacement, from the original data set. For each of these samples, the cut point that minimized the absolute difference between the sensitivity and specificity was determined, and the 95% percent confidence interval calculated as the 2.5th and 97.5th percentiles of these 1000 optimal cut points.

In addition, multivariable logistic regression models were created for the composite outcome using these cutoff points and adjusted for demographic and clinical covariates. Interactions with these variables and treatment assignment (e.g., 17 β -estradiol or placebo) were also considered. Variables with a $P < 0.1$ in the univariate analysis as listed in Table 1, along with those variables deemed to be clinically and scientifically important (e.g., age, smoking, hypertension, carotid bruit, insulin dependent diabetes), were considered in the multivariable regression models.

RESULTS

Eleven women were dropped from further study due to change in surgical plan ($n = 2$), withdrawal of consent ($n = 4$), previously undisclosed history of breast cancer ($n = 1$), difficulty with vision precluding psychometric testing ($n = 2$), previously undisclosed history of hormone replacement therapy within 6 mo of surgery ($n = 1$) and research staff error ($n = 1$). Thus, data from 175 women were available for the current study. Demographics and operative characteristics for the patients are listed in Table 1. Troponin I levels (mean $\pm$ SD) on the first postoperative day were not different between patients receiving 17 β -estradiol or placebo (8 ± 12 ng/mL vs 7 ± 10 ng/mL, $P = 0.788$) or at any time after surgery. Nearly 70% of patients underwent isolated CABG surgery, whereas approximately 14% and 18% underwent combined CABG and valvular surgery or isolated valvular surgery, respectively. Variables that were statistically associated with MACE after controlling for treatment assignment included race ($P = 0.019$), history of transient ischemic attack (TIA) ($P = 0.013$), history of congestive heart failure (CHF) ($P = 0.021$), duration of cardiopulmonary bypass time in minutes ($P = 0.001$), and duration of aortic cross-clamping in minutes ($P = 0.007$). Treatment assignment was not statistically associated with MACE.

Perioperative complications are listed in Table 2. The median duration of hospitalization in the intensive care unit was 2.0 (inter-quartile range, 1–3) days. The median length of hospitalization was 7.0 (inter-quartile range, 5–9) days. Of the 11 operative deaths, 2 occurred before blood was obtained for troponin I measurement on the first postoperative day. The composite MACE outcome occurred in 29 (16.6%) patients.

The troponin I cutoff level and associated predictive values for the primary study outcome is listed in Table 3 and Figure 1. Troponin I data from the first postoperative day were available for 27 of 29 patients with an adverse cardiovascular event. A cutoff point for troponin I of >7.6 ng/mL (95% confidence interval, 6.4–10.8) provided the optimal classification of a patients having or not having a MACE as shown in Figure 2 (area under the ROC = 0.862). The distribution of troponin levels above and below 7.6 ng/mL for women with and without MACE is shown in Figure 3. The sensitivity for troponin I >7.6 ng/mL for identifying risk for a MACE was 82% and specificity of 77%. The negative predictive value of a troponin I level for identifying a patient with a MACE (96%) was high, but the positive predictive value was moderate (40%). We repeated the sensitivity and specificity calculations assuming the 2 patients who died before troponin I measurement had a troponin level below or above 7.6 ng/mL. If the level was <7.6 ng/mL, the sensitivity decreases to 75.9% and negative predictive value decreases to 94%. If we assume that the people who died before troponin I measurements had levels >7.6 ng/mL, the sensitivity increases to 82.8% and positive predictive value increases from 40% to 42%. We examined the interaction of troponin I by cut-point of 7.6 ng/mL and found no significant 17β -estradiol treatment-troponin interaction ($P = 0.141$). That is, the cut-point of 7.6 ng/mL was not different for patients receiving 17β -estradiol or placebo.

Variables independently associated with MACE or at least trended towards significance ($P < 0.1$) based on multivariate logistic regression analysis are listed in Table 4. A troponin I level >7.6 ng/mL on postoperative day 1 was independently associated with risk for a MACE even after adjusting for race, history of CHF and prior TIA (adjusted odds ratio, 16.3; 95% confidence interval, 5.3–49.8; $P < 0.001$). A good fit of the model to the data is indicated by a Hosmer-Lemeshow P value equal to 0.943, and an area under the ROC = 0.8608 (Fig. 1). Duration of cardiopulmonary bypass and aortic cross-clamping, though significantly associated with the outcome after adjusting for treatment, were no longer statistically significant once we adjusted the variables listed in Table 4. A sub-analysis of the data using only those patients undergoing CABG surgery gave a multivariable model similar to that reported in Table 4 with the same variables being significant and estimated adjusted odds ratios of a similar magnitude.

DISCUSSION

In this study of postmenopausal women, our results show that troponin I measurement after cardiac surgery is predictive of risk for developing a composite adverse cardiovascular outcome of Q-wave MI, low cardiac output state or death. A value of <7.6 ng/mL had a high negative predictive value (96%), indicating that a patient was unlikely to experience an adverse event below this level. A value >7.6 ng/mL was moderately predictive (40%) that a patient would develop an adverse cardiovascular complication. Other variables found to be

independently associated with an adverse cardiovascular outcome included African American race, history of CHF, and history of TIA.

Risk stratification of patients for complications after cardiac surgery might allow for a focused implementation of monitoring and treatment strategies that could lead to improved patient outcomes. Several investigations have shown that postoperative troponin I levels measured within 24 h of cardiac surgery are an independent predictor of morbidity and mortality 30 days and up to 3 yr after cardiac surgery.^{7–16} Compared with men, women undergoing cardiac surgery are more likely to have diabetes and hypertension, but fewer coronary artery stenoses and less frequent left ventricular dysfunction.¹ Coronary atherosclerosis, even in the absence of epicardial obstructive stenosis, may alter the coronary microcirculation and limit myocardial blood flow.¹⁹ This limitation in blood flow might be particularly marked in elderly women as a result of endothelial dysfunction that develops after menopause.²⁰ Moreover, spontaneous myocardial ischemia or ischemia that occurs during aortic cross-clamping may induce microvascular injury and result in reduced myocardial flow despite normal epicardial coronary artery flow (“no reflow” phenomenon).²¹ Nonetheless, it is widely assumed clinically that elevated troponin levels after cardiac surgery have the same implications for women and men, even though women represented <30% of the patients in these studies and despite the differences in cardiac risk profiles between sexes. Furthermore, other data suggest that there are sex-based differences in regard to the extent of myocardial injury assessed with troponin I measurements. In an age-, Body Mass Index- and case-matched study, men were found to have a threefold higher peak troponin I level than women (18.5 ± 5.7 vs 6.4 ± 1.0 ng/mL) after CABG surgery.¹⁷ Thus, it is not clear whether susceptibility to adverse cardiovascular outcomes including mortality occur with a similar extent of myocardial damage perioperatively in women as in men.

The troponin I value of $>7.6 \mu\text{g/L}$ that we found to be prognostic of cardiovascular events after surgery in women is lower than that reported in studies that included mostly men. For example, Lasocki et al.⁸ and Fellahi et al.⁹ found that a troponin I level >13 ng/mL measured within 24 h after cardiac surgery predicted in-hospital mortality and death over a 2-yr follow-up period. Jacquet et al.¹⁰ reported that troponin I levels >13.1 ng/mL had 90% sensitivity and 100% specificity for identifying Q-wave MI after CABG surgery. Other smaller studies found that postoperative troponin I levels of 9 to 15 ng/mL predicted Q-wave MI in patients after CABG surgery.^{11–12} The ability to compare absolute troponin I levels among studies is confounded by the multiple surgical and patient factors that can influence myocardial injury, including type of surgery, number of CABGs performed, quality of the distal anastomosis and choice of cardioplegia.^{7,8,22} Of more concern for comparing data among studies is recent evidence of marked variation in troponin I reference values among assays from different manufacturers.²³ The troponin assay used in this study, though, is the same as that used in the studies by Lasocki et al.⁸ and Jacquet et al.¹⁰ Regardless, our methods are in accordance with laboratory guidelines recommended by the National Academy of Clinical Biochemistry as the levels were obtained using a single assay.²³

National benchmarking data from the multicenter Society of Thoracic Surgeons National Cardiac Surgery Database report that mortality after isolated CABG surgery is twice as high

for women than men: 4.52% compared with 2.61%.¹ Our study included patients undergoing CABG and/or valvular surgery, and thus, is representative of a broad array of contemporary practices. This fact and the inclusion of elderly, high-risk postmenopausal women in this study likely explains the mortality rate (6.3%) observed in this cohort.

Estrogens have been consistently found experimentally to have cardioprotective properties via multiple mechanisms, including enhancement of endothelial- and nonendothelial-dependent vasodilation, inhibition of cytokine-mediated increases in endothelial adhesion molecule expression, and activation of ischemic preconditioning pathways, such as the adenosine triphosphate-sensitive potassium (K_{ATP}) channels.^{20,24–31} Estrogen improves endothelial-dependent blood flow in postmenopausal women, and its vascular protective effects might reduce ischemia-induced microvascular injury.^{25–27} Despite the potential beneficial effects, we failed to demonstrate differences in troponin I levels after surgery between women who received 17 β -estradiol and those who received placebo. Our findings suggest that estrogen's protective mechanisms might not benefit women undergoing cardiac surgery, which has a highly complex etiology of myocardial injury.

The strengths of this study were that the data were prospectively collected and outcomes were determined in a blinded, adjudicated fashion. We acknowledge limitations, however, including the small sample size that may limit the assessment of the relative risk associated with troponin I elevation and mortality. The sample size of our study was determined based on the primary outcome of neurocognitive dysfunction and not the cardiovascular outcomes we examined in this analysis. Thus, the clinical implications of our findings require confirmation in a study involving a larger sample size. Our study was focused on operative events and does not provide information regarding the long-term prognostic value of elevated troponin I levels in postmenopausal women after cardiac surgery.

Compliance with evidence-based guidelines known to lower mortality from coronary heart disease is less after CABG surgery than in nonsurgical populations.³² Furthermore, performance measures for secondary prevention of atherosclerosis have been shown to be less frequently implemented in women than in men.³² Our study and those of others show that in the first 24 h after cardiac surgery, elevation of troponin levels is predictive of additional cardiovascular complications that impact short- and long-term mortality. Thus, in patients with increased postoperative troponin levels, particular diligence should be applied in implementing evidence-based medical therapies, including antiplatelet drugs, statins, β -adrenergic receptor blockers, and/or angiotensin converting enzyme inhibitors. Other strategies might also be worthy of consideration to aggressively assess completeness of revascularization, such as postoperative stress testing or coronary angiography.

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Figure 1.

Estimated sensitivities and specificities for various cut-points for troponin I levels obtained on postoperative day 1. The optimal cutoff with the highest sensitivity and specificity was 7.6 ng/mL.

Figure 2.

Receiver operating characteristic (ROC) curve for the detection of the primary outcome of a major adverse cardiovascular events (MACE) (consisting of postoperative Q-wave myocardial infarction, low cardiac output state, or operative death) versus troponin I levels determined on postoperative day 1. The area under the ROC curve for a troponin cutoff value of 7.6 ng/mL on postoperative day 1 was 0.8623, provided the optimal sensitivity and specificity for detection of the composite outcome.

Figure 3.

Distribution of troponin values obtained on postoperative day 1 (POD 1) for women with (open bars) and without (solid bars) a major adverse cardiovascular event (MACE). The best cut-point is specified by the arrow at 7.6 ng/mL. The open bars to the left of the arrow represent the false negatives, while the solid bars to the right of the arrow represent the false positives. Four troponin POD 1 values between 108 and 294 ng/mL, all of each were for patients with a MACE are not shown in the Figure.

Table 1

Patient Demographic and Operative Data

Variable	All patients (n = 175)	Patients without MACE (n = 146)	Patients with MACE (n = 29)	P
Treatment assignment 17 β -estradiol	86 (49.4%)	69 (47.6%)	17 (58.6%)	0.278 ^a
Mean (SD) age	70.3 (8.7)	70.4 (8.7)	69.8 (8.8)	0.781
Race				
Caucasian	155 (88.6%)	133 (91.1%)	22 (75.9%)	0.019
African American	20 (11.4%)	13 (8.9%)	7 (24.1%)	
History of CVA (yes)	23 (13.1%)	17 (11.6%)	6 (20.7%)	0.222
History of TIA (yes)	15 (8.6%)	9 (6.2%)	6 (20.7%)	0.013
COPD (yes)	25 (14.3%)	20 (13.7%)	5 (17.2%)	0.689
Congestive heart failure	41 (23.4%)	29 (19.9%)	12 (41.4%)	0.021
Left ventricular function				
Normal	101 (57.7%)	86 (58.9%)	15 (51.7%)	0.717 ^b
Mildly reduced	37 (21.1%)	30 (20.6%)	7 (24.1%)	
Moderately reduced	14 (8.0%)	10 (6.9%)	4 (13.8%)	
Severely reduced	16 (9.1%)	13 (8.9%)	3 (10.3%)	
Unknown	7 (4.0%)	7 (4.8%)	0 (0%)	
Hypertension (yes)	151 (86.3%)	125 (85.6%)	26 (89.7%)	0.597
Previous MI (yes)	72 (41.1%)	59 (40.4%)	13 (44.8%)	0.743
Peripheral vascular disease	30 (17.1%)	25 (17.1%)	5 (17.2%)	0.995
Insulin dependent diabetes	31 (17.7%)	26 (17.8%)	5 (17.2%)	0.943
noninsulin dependent diabetes	46 (26.4%)	35 (24.0%)	11 (39.3%)	0.079
Carotid bruit	12 (6.9%)	8 (5.5%)	4 (13.8%)	0.135
Smoking status				
Never	96 (54.9%)	82 (56.2%)	14 (48.3%)	0.652 ^b
Current	31 (17.7%)	24 (16.4%)	7 (24.1%)	
Prior	48 (27.4%)	40 (27.4%)	8 (27.6%)	
Bypass time (min)				
Mean (SD)	124.7 (55.8)	117.8 (47.3)	159.7 (78.9)	0.001
Median	118	113	141	
Clamp time (min)				
Mean (SD)	76.9 (163.0)	70.5 (175.8)	107.9 (62.6)	0.007
Median	85	83.5	98	
Surgery type				
CABG only	120 (68.6%)	102 (69.9%)	18 (62.1%)	0.082 ^b
CABG w/AVR	11 (6.3%)	10 (6.9%)	1 (3.5%)	
CABG w/MVR	13 (7.4%)	9 (6.2%)	4 (13.8%)	
AVR only	19 (10.9%)	18 (12.3%)	1 (3.5%)	
MVR only	12 (6.9%)	7 (4.8%)	5 (17.2%)	

P-values are given for the association between the given variable and the composite major adverse cardiovascular events (MACE) outcome after adjusting for treatment assignment using ordinary logistic regression modeling. The definition of MACE was either postoperative Q-wave myocardial infarction (MI), or low cardiac output state, or operative mortality.

MACE = major adverse cardiovascular events; MI = myocardial infarction; CVA = cerebral vascular accident; TIA = transient ischemic attack; COPD = chronic obstructive pulmonary disease; MI = myocardial infarction; AVR = aortic valve replacement; MVR = mitral valve replacement; CABG = coronary artery bypass graft surgery.

^a χ^2 test.

^b *Post hoc* global Wald test that all the dummy parameters representing all categories of the variable are equal to 0.

Table 2

The Number and Percentage of Patients With Perioperative Complications, Including the Composite Cardiovascular End-Point of Q-Wave Myocardial Infarction (MI), Low Cardiac Output State, and/or Operative Death

Complication	No. of patients ^a
New onset postoperative atrial fibrillation	73 (42.2%)
Clinical stroke	8 (4.6%)
Low cardiac output state	6 (3.5%)
Postoperative Q-Wave MI	15 (8.7%)
Operative death	11 (6.3%)
Composite MACE outcome ^b	29 (16.6%)

MACE = major adverse cardiovascular events; MI = myocardial infarction.

^aTotal number of patients in study sample was 175.

^bThis includes 1 patient with death and Q-wave MI and 1 patient with death and low cardiac output state. The latter patients were only considered once in the major adverse cardiovascular events (MACE) outcome.

Table 3

The Predictive Capacity of a Troponin I Threshold of 7.6 ng/mL Measured on Postoperative Day 1 for Identifying Risk for a Major Adverse Cardiovascular Event (MACE) Consisting of Postoperative Q-Wave Myocardial Infarction (MI), Low Cardiac Output State^a, or Death Within 30 Days of Surgery

Sensitivity	82% (62%, 94%)
Specificity	77% (70%, 84%)
Positive predictive value	40% (27%, 54%)
Negative predictive value	96% (90%, 99%)
<i>P</i> -value for model	<0.001
Area under ROC	0.862
Hosmer-Lemeshow <i>P</i> -value	0.146

Values in parenthesis are 95% confidence interval.

ROC = receiver operating characteristic; MI = myocardial infarction.

^aLow cardiac output was defined as cardiac index $<2.0 \text{ L} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$ for $>8 \text{ h}$ regardless of treatment.

Table 4

Variables Independently Associated With Major Adverse Cardiovascular Events (MACE) (Postoperative Q-Wave Myocardial Infarction, Low Cardiac Output State, or Death Within 30 days of Surgery) and Their Associated Adjusted Odds Ratios (95% confidence intervals) Based on Multivariate Logistic Regression Analysis

Variable	Odds ratio (95% CI)	P
Troponin I >7.6 ng/dL on postoperative day 1	16.3 (5.3–49.8)	<0.0001
African American race	4.9 (1.2–20.6)	0.030
CHF history	3.9 (1.4–11.5)	0.012
Prior TIA	4.3 (0.9–20.1)	0.062

ROC = 0.861 Hosmer-Lemeshow *p*-value = 0.943.

TIA = transient ischemic attack; CHF = congestive heart failure; CI = confidence interval.