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Pulmonary Systemic Pressure Ratio Correlates with Morbidity in Cardiac Valve Surgery

Sarah A. Schubert, MD¹, J. Hunter Mehaffey, MD MSc¹, Alexander Booth, BA², Leora T. Yarboro, MD¹, John A. Kern, MD¹, Jamie L.W. Kennedy, MD³, Gorav Ailawadi, MD¹, and Sula Mazimba, MD³

¹Division of Thoracic and Cardiovascular Surgery, University of Virginia, Charlottesville, Virginia

²University of Virginia School of Medicine, Charlottesville, Virginia

³Division of Cardiovascular Medicine, Department of Medicine, University of Virginia, Charlottesville, Virginia

Abstract

Objectives: Pulmonary hypertension portends worse outcomes in cardiac valve surgery; however, isolated pulmonary artery pressures may not accurately reflect patients' global cardiac function. To better account for the interventricular relationship, we hypothesized that patients with greater pulmonary-systemic ratios [mean pulmonary arterial pressure]/[mean systemic arterial pressure] would correlate with worse outcomes after valve surgery.

Design: Retrospective cohort study

Setting: Single academic hospital

Participants: 314 patients undergoing valve surgery with or without coronary artery bypass grafting (2004–2016) with Society of Thoracic Surgeons predicted risk scores and preoperative right heart catheterization

Interventions: None

Measurements and Main Results: The pulmonary-systemic ratio was calculated as (mean pulmonary arterial pressure/mean systemic arterial pressure); patients were stratified by pulmonary-systemic ratio quartile. Logistic regression assessed the risk-adjusted association between pulmonary-systemic ratio or mean pulmonary arterial pressure. Median pulmonary-systemic ratio was 0.33 (Q1, Q3: 0.23, 0.65); median pulmonary arterial pressure was 29 (21, 30) mmHg. Patients with the highest pulmonary-systemic ratio had the highest rates of morbidity and mortality ($p < 0.0001$). High pulmonary-systemic ratio was associated with longer duration in the intensive care unit ($p < 0.0001$) and hospital ($p < 0.0001$). After risk-adjustment, pulmonary-

Corresponding Author: Sarah Schubert, MD, Division of Thoracic and Cardiovascular Surgery, Department of Surgery, University of Virginia, ss9kw@virginia.edu, Phone: (724)504-4254.

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systemic ratio and pulmonary arterial pressure were independently associated with morbidity and mortality, but the pulmonary-systemic ratio (odds ratio=23.88, $p=0.008$, Wald 7.1) was more strongly associated than pulmonary arterial pressure (odds ratio=1.035, $p=0.011$, Wald 6.5).

Conclusions: The pulmonary-systemic ratio is more strongly associated with risk-adjusted morbidity and mortality in valve surgery than pulmonary arterial pressure. By integrating ventricular interactions, this metric may better characterize the risk of valve surgery.

Keywords

cardiac catheterization/intervention; cardiac function; physiology; pulmonary vascular resistance/hypertension

Introduction

Pulmonary hypertension is known to portend worse outcomes in patients undergoing cardiac surgery.¹ Prior studies have demonstrated increased operative mortality and decreased long-term survival, particularly in patients undergoing valvular heart surgery.^{2,3} Although not currently included in the Society of Thoracic Surgeons' (STS) risk score, the detrimental effects of pulmonary hypertension on surgical outcomes has been recognized and accordingly incorporated in the risk modeling using the EuroSCORE and to grade the severity of pathology in patients with congenital heart disease.⁴⁻⁶

In patients with acquired valvular heart disease, pulmonary hypertension – defined as a mean pulmonary arterial pressure (MPAP) >25 mmHg – generally develops from chronically elevated left atrial pressures. This prolonged elevation of left atrial pressure leads to a cascade of unfavorable pulmonary vascular remodeling processes, resulting in elevated pulmonary vascular resistance (PVR) and a high trans-pulmonary gradient. The ultimate consequence is increased afterload on the right ventricle. This increased afterload leads to undue stress on the right ventricle, a chamber that is not accustomed to pumping blood against high pressures. The net effect of these deleterious changes is right ventricular dilatation and failure. Furthermore, a failing and dilated right ventricle may have unfavorable hemodynamics on the left ventricle. These hemodynamic and structural perturbations may not be fully accounted for by pulmonary artery systolic pressure alone – as used in the EuroSCORE calculation – as it does not account for ventricular interactions.⁴

Because of the known significance of preoperative pulmonary hypertension on postoperative morbidity and mortality, we hypothesized that a simple index that incorporated interactions between the ventricles would be associated with adverse outcomes in patients undergoing cardiac surgery. We therefore evaluated the pulmonary-systemic pressure ratio (PSR), range 0–1, defined as [mean pulmonary artery pressure]/[mean systemic arterial pressure]. We hypothesized that a greater PSR would be associated with increased morbidity and mortality in patients undergoing single valve surgery with or without coronary artery bypass surgery (CABG).

Patients and Methods

Patient Selection and Outcomes

The study included consecutive patients with a preoperative right heart catheterization (RHC) who underwent single valve surgery (aortic replacement and mitral repair or replacement) with or without CABG between January 2004 and June 2016 at an academic medical center. Clinical data including the STS predicted risk of morbidity or mortality (PROMM) score was extracted from an institutional STS database. The STS data of these 314 patients was paired with their RHC values, including MPAP and MAP, obtained from a clinical data repository (Figure 1). The study was approved by the University of Virginia Institutional Review Board (IRB) with a waiver of patient consent (IRB protocol #19247).

The primary outcomes of interest were risk-adjusted morbidity and mortality. Outcomes reported were standard STS measures of morbidity and mortality, which included permanent stroke, cardiac arrest, renal failure, atrial fibrillation, prolonged ventilation, pneumonia, packed red blood cell (PRBC) transfusion, readmission, discharge to a facility, and operative mortality. STS major morbidity was used as a composite measure of morbidity, and that metric included permanent stroke, prolonged ventilation, deep sternal wound infection, renal failure, and reoperation for any reason. Reoperation included any patient who was taken back to the operating room for bleeding with or without tamponade, graft occlusion, valve dysfunction, or any other cardiac reason. Operative mortality was defined as death within 30-days or prior to discharge.

Measuring the Pulmonary-Systemic Ratio

The PSR was calculated as a ratio of MPAP to MAP, both of which were obtained at the time of patients' preoperative RHC. Patients were then stratified into quartiles based on their PSR: quartile 1: 0.23, quartile 2: 0.24 – 0.33, quartile 3: 0.34 – 0.45, and quartile 4: >0.45.

Statistical Analysis

Categorical variables are presented as a count (percentage) and continuous variables as either mean $\pm$ standard deviation or median [25th, 75th percentile] depending on normality. Categorical variables were analyzed by Chi-Square Test or Fisher Exact Test as indicated. Continuous variables were analyzed by Students T-test or Mann-Whitney U Test as appropriate. Missing variable information was excluded from the respective unpaired univariate analysis. Finally, logistic regression modeling was used to assess the risk-adjusted associations between PSR or isolated MPAP on morbidity and mortality in two different models adjusting for the STS PROMM and year of surgery. All analyses were performed with SAS Version 9.4 (SAS Institute, Cary, NC) and significance was determined by $p < 0.05$, two-sided.

Results

Patient Demographics and Preoperative Characteristics

From January 2004 to June 2016, a total of 314 consecutive patients underwent valve repair or replacement with or without coronary artery bypass grafting (CABG) with complete

hemodynamic data on a preoperative RHC. The median PSR was 0.33 [0.23–0.65], and the median MPAP was 29 [21, 30] mmHg of the entire cohort. The median age of the cohort was 72 [62, 79] years, and the age of patients in each quartile of PSR decreased as the ratio increased, with quartile 4 having the youngest patients (64 [54, 77] years). The prevalence of diabetes, hypertension, endocarditis, immunosuppression, prior myocardial infarctions, dialysis-dependent renal failure, and peripheral vascular disease were similar between each of the patient quartiles (Table 1). Heart failure rates were statistically different between each quartile ($p=0.03$) and increased with each quartile increase, with 68% of patients in quartile 1, 74.7% of quartile 2, 79.5% of quartile 3, and 87.3% of quartile 4, demonstrating heart failure symptoms within two weeks of surgery.

Rates of aortic stenosis were significantly greater among patients in the lower quartiles, including 87.2% of patients in quartile 1, 74.7% of quartile 2, 59% of quartile 3, and 53.2% of quartile 4. Rates of severe aortic insufficiency, severe mitral regurgitation, and severe mitral stenosis, however, were significantly greater in the higher PSR quartiles. Finally, as PSR increased, both PROM and PROMM also increased (Table 1).

Operative Factors

Total cardiopulmonary bypass (CPB) time and cross-clamp time were both significantly different between each quartile, with both CPB and cross-clamp times increasing with each quartile increase. A majority of the entire cohort underwent AVR, with or without CABG (48.7% isolated AVR, 23.6% AVR with CABG). A majority of the patients undergoing mitral valve procedures were concentrated in the third and fourth quartiles.

Postoperative Outcomes

Patients in the fourth quartile had the greatest rates of STS major morbidity, (6.4%, 16.5%, 16.7%, 34.2%, $p<0.0001$). Overall mortality of the cohort was 2.5%, compared to the STS PROM of 4.1% (Observed: Expected: 0.61), and all deaths were in the fourth PSR quartile (Table 3). Because of relatively low event numbers, reoperation was the only postoperative complication included in the STS major morbidity metric that was statistically different between the lowest and highest PSR quartiles (1.3%, 5.1%, 7.7%, 12.7%, $p=0.03$).

Resource Utilization

Increasing PSR was also associated with increasing duration in the intensive care unit (46.5 hours, 53.2 hours, 69.5 hours, 115 (48–192) hours, $p<0.0001$) and with increasing length of hospitalization (6 days, 6 days, 7 days, 8 days, $p<0.0001$).

Risk-Adjusted Outcomes

The PSR demonstrated a positive linear relationship ($m=1.7$, $R^2=0.79$) with observed morbidity or mortality to expected STS PROMM (Figure 2), with only patients in the fourth quartile having higher observed than expected morbidity or mortality. After risk-adjustment, both PSR and MPAP were independently associated with morbidity or mortality, but higher PSR (OR=23.88, $p=0.008$, Wald 7.1) was more strongly associated with morbidity or mortality than increasing MPAP (OR=1.035, $p=0.011$, Wald 6.5).

Comment: The present study evaluated the association of the hemodynamic index PSR with outcomes among patients undergoing single valve surgery with or without CABG surgery. First, we found that PSR was significantly associated with adverse outcomes independent of established surgical risk scores. In addition, PSR was more strongly associated with adverse outcomes compared to mean pulmonary arterial pressure alone. Although prior studies have investigated the idea of using both the MPAP and the MAP together to capture ventricular dysfunction and pulmonary hypertension, this study uniquely found that a ratio of MPAP to MAP obtained before any surgical intervention could predict adverse postoperative outcomes and was positively correlated with increased resource utilization for patients undergoing cardiac valve surgery.

In this study cohort, pulmonary hypertension was most likely the result of valvular pathology – that is, pulmonary hypertension secondary to left ventricular dysfunction. It has been reported that 100% and 65% of symptomatic patients with mitral and aortic valve pathology, respectively, have some element of pulmonary hypertension.⁷⁻⁹ With the progression of valvular pathology, there is a passive increase in left atrial pressure that leads to pulmonary vascular remodeling and pulmonary hypertension which in turn leads to unfavorable hemodynamic effects on the right ventricle. Under normal anatomic conditions, the right and left ventricles are related via their interlacing adjacent muscle fibers. In addition, a shared septum and pericardial sac allows for the physiological coupling between the two chambers in a way that facilitates optimal cardiac performance.¹⁰ In the presence of pulmonary hypertension, increased RV afterload is associated with a decrease in RV function, in turn leading to an increase in RV end-diastolic volumes. The negative consequences of these hemodynamic changes (diminished cardiac output and systemic hypotension) are mediated via negative interventricular septal diastolic interactions and increased RV free wall stiffness.¹¹ The left ventricle is known to undergo atrophic remodeling, decreased contractility and hypotension as a result of worsening of RV failure.¹² Conceptually, there may be a threshold at which the PSR would decrease in the end stages of pulmonary hypertension when MPAP decreases as a result of a failing RV. Even so, this advanced stage of pulmonary hypertension may also be balanced by low systemic pressures due to an impaired left ventricle from a failing RV.

Imaging and RHC studies have demonstrated that left ventricular peak filling rate and MPAP decrease proportionally to decreases in right ventricular ejection fraction in patients with pulmonary hypertension.¹³ Another study of relative MPAP affirmed that this ratio correlates with the eccentricity index, a measure of the ventricular pressure on either side of the interventricular septum.¹⁴ Given these physiological considerations, PSR – a metric that integrates MPAP and MAP – takes into account the interactions between the two ventricles; thus, a high ratio signals advanced disease status.

This advanced disease status is reflected within the increasing quartiles of PSR, as each increase in PSR quartile is associated with a concomitant increase in patients' STS PROMM. Most notably, the percentage of patients in each PSR quartile with recent heart failure increased significantly with each quartile increase, thus indicating that the PSR index correlates – as expected – with worsening disease status. This relationship was evident on univariate analysis which demonstrated that increasing PSR was associated with worse

outcomes. With each quartile increase, there was significantly greater composite major morbidity, longer intensive care unit (ICU) stays, and longer duration of hospitalization for patients. Notably, all mortalities occurred among patients with the highest PSR; however, there were only 8 mortalities overall.

Moreover, risk-adjusted analysis yielded a Wald score for the PSR that was greater than the Wald score for MPAP, indicating that increasing PSR was more strongly associated with adverse postoperative outcomes than is increasing MPAP. Simply with the inclusion of MAP, the PSR adds left ventricular function into the metric, thereby accounting for left ventricular function in patients' postoperative outcomes. Left ventricular function, as quantified with MAP, is well-known to correlate with multiple postoperative outcomes. Prior studies have demonstrated the associations between decreased perioperative MAP with adverse postoperative outcomes, including acute kidney injury, stroke, prolonged ventilation, and mortality.^{15, 16} Although the measurement of MAP used in this study was taken at the time of the RHC, the findings of this study reiterate the overarching importance of a patient's systemic blood pressure on surgical outcomes.

Although the concept of using relative MPAP is not new, this study is different from preexisting research on this topic because it relies upon measurements that are taken well-before a patient is in the operating room to predict outcomes.^{17, 18} By having this hemodynamic parameter available before proceeding to the operating room, it could be a useful tool for deciding whether or not to operate on a patient, what operation is appropriate, and what postoperative care adjuncts – such as inhaled nitric oxide – to have available. Furthermore, separation of PSR values into strata adds granularity to this index, another unique attribute of this study. Separation of patients into these strata based on PSR then allows some correlation with outcomes, as seen in this study, thus making the index a useful predictor of postoperative complications.

Finally, although pulmonary arterial pressure can be estimated with Doppler echocardiography, the accuracy of those methods has been questioned, thus the use of preoperative RHC in estimating MPAP in this study. Fisher et al. found that Doppler echocardiography does not accurately predict pulmonary artery pressure in patients with pulmonary hypertension, with Doppler echocardiography both overestimating and underestimating pulmonary hypertension. Of note, underestimation of pulmonary hypertension by the Doppler method was of greater magnitude, leading to more significant misclassification of a patient by grade of pulmonary hypertension. Despite RHC not being a standard component of the preoperative evaluation of patients prior to valve surgery and carrying its own set of risks compared to non-invasive Doppler echocardiography, the findings of this study emphasize the need for careful assessment of patients' biventricular function prior to surgery.

Limitations

This retrospective analysis of data from a single institution may not be generalizable to all populations or institutions. Additionally, the chronicity of MAP and MPAP measurements may introduce bias, as they have been taken prior to surgery. All of the measurements represent a single point in time prior to surgery and without many of the confounding

variables such as general anesthetic use, protamine administration, positive end-expiratory pressure, and the systemic inflammatory response to cardiopulmonary bypass, that are present during cardiac surgery and are known to affect PVR.¹ In addition, the exact mechanism of elevated MPAP in each patient was not investigated, and different etiologies of pulmonary hypertension and the physiologic response to stress may influence how severely MPAP is elevated. Although there were a range of valve procedures represented in each quartile, patients undergoing mitral valve surgery were concentrated in the third and fourth quartiles; this unequal distribution of procedures across the quartiles is a limitation of the data that could confound the final results. Also, because there are multiple outcomes being analyzed, there is a risk of a type 1 error. Finally, a selection bias is likely present, as a RHC is not part of the standard preoperative work-up for patients undergoing cardiac surgery, and these patients were likely undergoing a RHC because they were suspected to have pulmonary hypertension and/or right ventricular dysfunction.

Conclusion:

The pulmonary-systemic ratio is independently associated with risk-adjusted morbidity and mortality in patients undergoing valve surgery. This ratio accounts for the relationship between the left and right ventricles and provided better predictive power compared to pulmonary artery pressure alone. By integrating biventricular interactions, this metric may better characterize the overall risks associated with valve surgery and should be considered in high risk patients with known pulmonary hypertension. Additional hemodynamic data should be captured in the STS database to further demonstrate the risk-adjusted effects of PSR on cardiac surgery outcomes in a large national cohort.

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Figure 1:

CONSORT diagram depicting patient selection. STS = Society of Thoracic Surgeons; PROMM = Predicted Risk of Morbidity or Mortality; AVR = aortic valve replacement; MV = mitral valve; CABG = coronary artery bypass grafting; RHC = right heart catheterization.

Figure 2:

The pulmonary-systemic ratio (PSR) demonstrated a positive linear relationship ($m=1.7$, $R^2=0.79$) with observed morbidity or mortality to expected STS Predicted Risk of Morbidity or Mortality.

Table 1:

Preoperative patient characteristics of the entire cohort and stratified by pulmonary-systemic ratio quartile.

Characteristics	Q1 (0.23) n=78	Q2 (0.24–0.33) n=79	Q3 (0.34–0.45) n=78	Q4 (>0.45) n=79	p value
Age	72 (64–79)	74 (66–79)	75 (66–81)	64 (54–77)	<0.001
(median, Q1,Q3) MPAP/MAP	0.20 (0.17–0.22)	0.27 (0.25–0.29)	0.38 (0.36–0.41)	0.54 (0.48–0.59)	<0.001
MPAP (mmHg)	18 (15–20)	24 (22–28)	34.5 (31–37)	44 (40–53)	<0.001
Female	43 (55.1%)	30 (38%)	33 (42.3%)	30 (38%)	0.098
Smoker	34 (43.6%)	23 (29.1%)	20 (25.6%)	36 (45.6%)	0.016
Hypertension	60 (76.9%)	65 (82.3%)	64 (82.1%)	55 (69.6%)	0.186
Diabetes	23 (29.5%)	27 (34.2%)	33 (42.3%)	25 (31.6%)	0.353
Dialysis dependent renal failure	1 (1.3%)	3 (3.8%)	4 (5.1%)	6 (7.6%)	0.280
Preoperative atrial fibrillation	4 (5.1%)	8 (10.1%)	15 (19.2%)	3 (3.8%)	0.004
Heart failure within 2 weeks	53 (68%)	59 (74.7%)	62 (79.5%)	69 (87.3%)	0.030
Endocarditis					0.132
None	76 (97.4%)	76 (96.2%)	72 (92.3%)	69 (87.3%)	
Active/Treated	2 (2.6%)	3 (3.8%)	6 (7.7%)	10 (12.7%)	
Aortic stenosis	68 (87.2%)	59 (74.7%)	46 (59.0%)	42 (53.2%)	<0.001
Mitral stenosis	1 (1.3%)	3 (3.8%)	11 (14.1%)	13 (16.5%)	0.001
Severe aortic insufficiency	0.0%	3 (3.8%)	4 (5.1%)	7 (8.9%)	0.060
Severe mitral insufficiency	7 (9.0%)	18 (22.8%)	20 (25.6%)	23 (29.1%)	0.013
Severe tricuspid insufficiency	0.0%	0.0%	1 (1.3%)	1 (1.3%)	0.570
Preoperative inotropic support	1 (2.6%)	1 (1.3%)	1 (1.3%)	6 (7.6%)	0.074
Cardiogenic shock	1 (1.3%)	2 (2.5%)	4 (5.1%)	13 (16.4%)	<0.001
Status					<0.001
Elective	63 (80.8%)	57 (72.2%)	43 (55.1%)	29 (36.7%)	
Urgent/Emergent	15 (19.2%)	22 (27.8%)	35 (44.9%)	50 (63.3%)	
PROMM (%)	18.1 (12.5–26.7)	24.6 (15.8–32.1)	29.0 (20.6–44.5)	31.1 (19.2–47.5)	<0.001
PROM (%)	2.9 (1.7–4.9)	3.6 (1.7–6.0)	5.5 (2.9–10.1)	5.7 (2.5–10.3)	<0.001

Q = quartile; Q1,Q3 = quartile 1, quartile 3 as 25th,75th percentile; MPAP = mean pulmonary arterial pressure; MAP = mean systemic arterial pressure; IABP = intra-aortic balloon pump; PROMM = Society of Thoracic Surgeons' Predicted Risk of Morbidity and Mortality; PROM = Society of Thoracic Surgeons' Predicted Risk of Mortality.

Table 2:

Operative Characteristics of the Entire Patient Cohort and Patients Stratified by Pulmonary-Systemic Ratio Quartile

Characteristics	Q1 (0.23) n=78	Q2 (0.24 to 0.33) n=79	Q3 (0.34 to 0.45) n=78	Q4 (>0.45) n=79	p value
Cross-clamp time (minutes; median, Q1,Q3)	69.5 (56.5–85.5)	75.5 (61–95)	78 (66–91)	86 (64–102)	0.039
CPB time (minutes; median, Q1,Q3)	99 (82–118)	110 (90–132)	113.5 (93–130)	123 (93–151)	0.002

Procedure Type	Q1 (0.23) n=78	Q2 (0.24 to 0.33) n=79	Q3 (0.34 to 0.45) n=78	Q4 (>0.45) n=79	p value
AVR	53 (68%)	40 (50.6%)	30 (38.5%)	30 (38%)	0.0002
AVR with CABG	17 (21.8%)	20 (25.3%)	20 (25.6%)	17 (21.5%)	
MV repair	5 (6.4%)	9 (11.4%)	8 (10.3%)	7 (8.9%)	
MV repair with CABG	1 (1.3%)	1 (1.3%)	6 (7.7%)	3 (3.8%)	
MV replacement with CABG	0 (0%)	3 (3.8%)	4 (5.1%)	1 (1.3%)	
MV replacement	2 (2.6%)	6 (7.6%)	10 (12.8%)	21 (26.6%)	

Q = quartile; Q1,Q3 = quartile 1, quartile 3 as 25th,75th percentile; CPB = cardiopulmonary bypass; AVR = aortic valve replacement; CABG = coronary artery bypass grafting; MV = mitral valve

Table 3:

Postoperative Outcomes, Stratified by Pulmonary-Systemic Ratio Quartile

Outcomes	Q1 (0.23) n=78	Q2 (0.24 to 0.33) n=79	Q3 (0.34 to 0.45) n=78	Q4 (>0.45) n=79	p value
STS operative mortality	0 (0%)	0 (0%)	0 (0%)	8 (10.1%)	< 0.0001
STS major morbidity	5 (6.4%)	13 (16.5%)	13 (16.7%)	27 (34.2%)	0.0001
STS morbidity or mortality	5 (6.4%)	13 (16.5%)	13 (16.7%)	28 (35.4%)	< 0.0001
Permanent stroke	1 (1.3%)	2 (2.5%)	2 (2.6%)	4 (5.1%)	0.5450
Atrial fibrillation	17 (21.8%)	23 (29.1%)	18 (23.1%)	23 (29.1%)	0.6020
Pneumonia	1 (1.3%)	7 (8.9%)	3 (3.8%)	5 (6.3%)	0.1603
Renal failure	3 (3.9%)	2 (2.5%)	2 (2.6%)	8 (10.1%)	0.0774
Deep sternal wound infection	0 (0%)	1 (1.3%)	0 (0%)	0 (0%)	0.3941
Reoperation for any reason	1 (1.3%)	4 (5.1%)	6 (7.7%)	10 (12.7%)	0.0347
Discharge to facility	25 (32.1%)	25 (31.6%)	35 (44.9%)	25 (31.6%)	0.2196
ICU stay (hours; median, Q1,Q3)	46.5 (23–94)	53.2 (25–93)	69.5 (41.6–141)	115 (48 192)	< 0.0001
Length of stay (days; median, Q1,Q3)	6 (5–8)	6 (5–8)	7 (5–11)	8 (6–13)	< 0.0001
Observed to ExpectedPROMM	0.353	0.671	0.576	1.13	

Q = quartile; Q1,Q3 = quartile 1, quartile 3 as 25th,75th percentile; STS = Society of Thoracic Surgeons; ICU = intensive care unit; PROM = STS Predicted Risk of Mortality.