Predicting operative mortality in patients who undergo elective open thoracoabdominal aortic aneurysm repair

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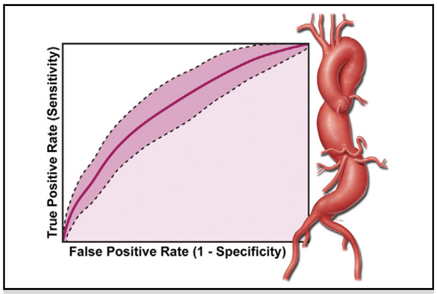
ABSTRACT

Background: We have developed a model aimed at identifying preoperative predictors of operative mortality in patients who undergo elective, open thoracoabdominal aortic aneurysm (TAAA) repair. We converted this model into an intuitive nomogram to aid preoperative counseling.

Methods: We retrospectively analyzed data from 2884 elective, open TAAA repairs performed between 1986 and 2023 in a single practice. Using clinical and selected operative variables, we built 4 predictive models: multivariable logistic regression (MLR), random forest, support vector machine, and gradient boosting machine. Each model's predictive effectiveness was evaluated with the C-statistic. Test C-statistics were computed using an 80:20 cross-validation scheme with 1000 iterations.

Results: Operative death occurred in 200 patients (6.9%). Test set C-statistics showed that the MLR model (median, 0.68; interquartile range [IQR], 0.65-0.71) outperformed the machine learning models (0.61 [IQR, 0.59-0.64] for random forest; 0.61 [IQR, 0.58-0.64] for support vector machine; 0.65 [IQR, 0.62-0.67] for gradient boosting machine). The final MLR model was based on 7 characteristics: increasing age (odds ratio [OR], 1.04/y; $P < .001$), cerebrovascular disease (OR, 1.54; $P = .01$), chronic kidney disease (OR, 1.53; $P = .008$), symptomatic aneurysm (OR, 1.42; $P = .02$), and Crawford extent I (OR, 0.66; $P = .08$), extent II (OR, 1.61; $P = .01$), and extent IV (OR, 0.41; $P = .002$). We converted this model into a nomogram.

Conclusions: Using institutional data, we evaluated several models to predict operative mortality in elective TAAA repair, using information available to surgeons preoperatively. We then converted the best predictive model, the MLR model, into an intuitive nomogram to aid patient counseling. (JTCVS Open 2024;22:95-103)



Receiver operating characteristic curve for predicting operative mortality in TAAA repair patients.

CENTRAL MESSAGE

We built a model using preoperative patient characteristics to predict operative mortality in patients undergoing thoracoabdominal aortic aneurysm repair. This model was converted into a nomogram to aid patient counseling.

PERSPECTIVE

We built a multivariable logistic regression model to predict operative mortality in thoracoabdominal aortic aneurysm repair by using variables known preoperatively: age, symptomatic aortic aneurysm, chronic kidney disease, cerebrovascular disease, and Crawford extent of repair. This model had better predictive effectiveness than machine learning models and was converted to a nomogram that can aid patient counseling.

See Discussion on page 104.

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Abbreviations and Acronyms

CKD = chronic kidney disease
MLR = multivariable logistic regression
OR = odds ratio
TAAA = thoracoabdominal aortic aneurysm

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When counseling a patient deciding whether to undergo elective thoracoabdominal aortic aneurysm (TAAA) repair, a central decision involves weighing the patient's risk of aortic rupture without operation against their risk of death from the repair.[1] Even at experienced centers, the risk of operative mortality in open TAAA repairs is 3.6% to 15.9% overall[2-5] and 1.5% to 6.2% for elective repairs.[2,5,6] Thus, being able to accurately estimate a patient's operative mortality risk from characteristics known preoperatively is vital for patient counseling.

More than 2 decades ago, our research group developed a multivariable logistic regression (MLR) model to predict operative mortality in patients who undergo TAAA repair.[7] Statistical methods and surgical approaches have evolved since then. The aim of the present study was to evaluate parametric and nonparametric statistical models to identify a predictive model for operative death. A secondary aim was to use that model to create a nomogram, an intuitive graphical tool for translating abstract models into an understandable form.

PATIENTS AND METHODS

Study Protocol and Patient Cohort

The Baylor College of Medicine Institutional Review Board approved our clinical research protocol (18095) in 2006. Whenever possible, we obtained informed consent to collect clinical data from patients who underwent their operation after protocol approval. The Institutional Review Board waived the need for consent from patients whose illness prevented them from providing consent or who had no family members available to provide consent for them, as well as for patients who underwent surgery before the protocol was approved. For patients treated before 2006, data were collected retrospectively from medical records. From October 1986 to August 2023, our single service (led by the senior author) performed 2884 consecutive open, elective TAAA repairs.

Study Definitions and Follow-up

All data were collected using standard definitions.[2,8] Elective repairs were planned after a preoperative clinical assessment. We defined operative mortality as any patient death before final hospital discharge (including any hospital transfer) or within 30 days of surgery after discharge; all patients without operative death were considered early survivors. Chronic kidney disease (CKD) was defined as an estimated glomerular filtration rate of <60 mL/min/1.73 m^2. Symptoms of aortic aneurysm included any self-reported finding (acute or chronic, mild or severe) associated with the aneurysm, including hoarseness, dysphagia, back pain, and other symptoms identified during formal preoperative patient evaluation.

Surgical Techniques

Preoperatively, elective patients were optimized as much as possible in several key clinical areas (eg, neurologic, cardiovascular, respiratory, renal) according to their baseline comorbidities.[9] Our multimodal surgical approach has been largely standardized since 2005 and is based on the Crawford extent of repair.[8,10,11] Since our previous model was derived,[7] our practice has adopted key innovations (eg, cerebrospinal fluid drainage, balloon-expandable branch artery stents) that were not used in the original cohort.

Regardless of the extent of repair, our operations generally involved moderate systemic heparinization (1.0-1.5 mg/kg), mild permissive hypothermia (32-34 °C, nasopharyngeal), cold (4 °C solution) perfusion of the renal arteries, and whenever possible, segmental artery reattachment (between T8 and L1). Patients undergoing extent I or II repair generally received cerebrospinal fluid drainage, left heart bypass, and selective visceral perfusion. Visceral artery lesions were managed with endarterectomy, balloon-expandable stents, or bypass grafts as needed. Incidental splenectomy was performed if the spleen was damaged and believed to present a risk for bleeding when assessed at the end of the repair.[12]

Statistical Analysis

Statistical analyses were performed with R version 4.2.2 (R Project for Statistical Computing). Continuous variables were evaluated for normality and are presented as median (interquartile range), as appropriate. Categorical variables are presented as number (percentage). Data were stratified by operative death (early survivors vs nonsurvivors); univariate comparisons were conducted with the Pearson χ^2 test or nonparametric Wilcoxon rank-sum test as appropriate. Continuous variables were assessed for normality with the Shapiro-Wilk test. Missing data were imputed with regression imputation based on the methods of Blackburn and colleagues.[13]

We tested the predictive effectiveness of an MLR model and 3 machine learning models: random forest, support vector machine, and gradient boosting machine. Only variables available preoperatively were used in modeling. For the MLR model, all variables underwent univariate testing first; variables associated with operative mortality at $P < .1$ were included in the multivariable model (Table E1). Variables were then removed using backward selection until all remaining variables were significant at $P < .1$, forming the final predictive model.

For the random forest, support vector machine, and gradient boosting machine models, several hyperparameters were varied to tune the model to its optimal form. Hyperparameters (Table E2) were chosen as described by Ostberg and colleagues.[14] The hyperparameters for each model were evaluated using the test set C-statistic over 10 iterations; the hyperparameters with the highest C-statistic were selected as the final model form for each. The final model for each of the model types was created from the full dataset, using the same variables used to build the MLR model.

Once each model was in its final form, the models were compared in terms of predictive effectiveness using an 80:20 cross-validation scheme—meaning that 80% of the data were used to train the models, and the remaining 20% were used to test the model's effectiveness. This process was repeated 1000 times. The validation cohorts were used only to evaluate model predictive effectiveness, not to create the final model. The final comparison outcome was the median test C-statistic. The final MLR model was converted into a nomogram to aid patient counseling.

RESULTS

Operative Death

There were 200 operative deaths (6.9%), of which 131 occurred within 30 days of surgery (4.5% of the cohort; 65.5% of operative deaths). There were 2684 early survivors (93.1%).

Preoperative Characteristics

Compared with patients who survived repair, nonsurvivors were older (median age, 71 [IQR, 64-76] years vs 67 [IQR, 58-72] years; $P < .001$), were less likely to have heritable thoracic aortic disease (10.0% vs 17.1%; $P = .009$), were more likely to have a symptomatic aneurysm (63.0% vs 55.2%; $P = .03$), and had a larger median maximum distal aortic diameter (6.4 [IQR, 5.6-7.0] cm vs 6.1 [IQR, 5.5-7.0] cm; $P = .03$) (Table 1). Nonsurvivors also had higher rates of cerebrovascular disease (27.5% vs 17.0%; $P < .001$), chronic obstructive pulmonary disease (57.0% vs 47.0%; $P = .006$), peripheral vascular disease (31.5% vs 24.9%; $P = .04$), and CKD (54.0% vs 38.1%; $P < .001$).

Operative Details

Nonsurvivors were more likely than survivors to undergo Crawford extent II repair (51.0% vs 33.9%) and less likely to undergo Crawford extent IV repair (10.0% vs 20.1%) (Table 1). Nonsurvivors also were more likely to need endarterectomy, stenting, or bypass grafting of the visceral or renal arteries (53.0% vs 42.2%; $P = .003$), as well as incidental splenectomy (18.5% vs 11.0%; $P = .002$) (Table E3).

TABLE 1. Preoperative and selected operative characteristics of patients who underwent elective, open TAAA repair

Characteristics	All (N = 2884)	Early survivors (N = 2684)	Operative deaths (N = 200)	*P* value
Age, y, median (IQR)	67 (59-73)	67 (58-72)	71 (64-76)	**<.001**
Male sex, n (%)	1851 (64.2)	1730 (64.5)	121 (60.5)	.3
Heritable thoracic aortic disease, n (%)*	479 (16.6)	459 (17.1)	20 (10.0)	**.01**
Aortic dissection, n (%)	1039 (36.0)	972 (36.2)	67 (33.5)	.5
Prior distal aortic repair, n (%)†	750 (26.0)	694 (25.9)	56 (28.0)	.6
Maximum distal aortic diameter, cm, median (IQR)	6.1 (5.5-7.0)	6.1 (5.5-7.0)	6.4 (5.6-7.0)	**.03**
Aortic diameter ≥7.0 cm, n (%)	710 (24.6)	655 (24.4)	55 (27.5)	.3
Aortic diameter unknown, n (%)	307 (10.6)	284 (10.6)	23 (11.5)	—
Coronary artery disease, n (%)	1026 (35.6)	947 (35.3)	79 (39.5)	.3
Prior myocardial infarction, n (%)	545 (18.9)	497 (18.5)	48 (24.0)	.07
Symptomatic aneurysm, n (%)	1607 (55.7)	1481 (55.2)	126 (63.0)	**.04**
Acute	85 (2.9)	78 (2.9)	7 (3.5)	.8
Chronic	1531 (53.1)	1412 (52.6)	119 (59.5)	.07
Hypertension, n (%)	2499 (86.7)	2320 (86.4)	179 (89.5)	.3
Hyperlipidemia, n (%)	968 (33.6)	895 (33.3)	73 (36.5)	.4
Diabetes, n (%)	227 (7.9)	205 (7.6)	22 (11.0)	.1
Cerebrovascular disease, n (%)	512 (17.8)	457 (17.0)	55 (27.5)	**<.001**
Body mass index, kg/m^2, median (IQR)	26.0 (23.1-29.2)	26.1 (23.1-29.3)	25.8 (22.5-29.0)	.4
Body mass index unknown, n (%)	246 (8.5)	221 (8.2)	25 (12.5)	—
COPD, n (%)	1375 (47.7)	1261 (47.0)	114 (57.0)	**.008**
Tobacco use, n (%)	2304 (79.9)	2135 (79.5)	169 (84.5)	.1
Peripheral vascular disease, n (%)	732 (25.4)	669 (24.9)	63 (31.5)	**.05**
Chronic kidney disease, n (%)‡	1130 (39.2)	1022 (38.1)	108 (54.0)	**<.001**
Chronic kidney disease unknown, n (%)	175 (6.1)	161 (6.0)	14 (7.0)	—
Crawford extent of repair, n (%)				**<.001**
Extent I	750 (26.0)	716 (26.7)	34 (17.0)	
Extent II	1012 (35.1)	910 (33.9)	102 (51.0)	
Extent III	562 (19.5)	518 (19.3)	44 (22.0)	
Extent IV	560 (19.4)	540 (20.1)	20 (10.0)	

Bold type indicates statistical significance. *IQR*, Interquartile range; *COPD*, chronic obstructive pulmonary disease. *Heritable thoracic aortic disease is defined as a known syndromic (eg, Marfan syndrome) or nonsyndromic (eg, familial thoracic aortic aneurysm and dissection) genetic disorder or the presence of aortic disease at age ≤50 years. †Any previous open or endovascular repair of the descending thoracic, thoracoabdominal, or abdominal aorta. ‡An estimated glomerular filtration rate of <60 mL/min/1.73 m^2.

Model Comparisons

For the MLR model, all predictors significant on univariate analysis, along with the complete multivariable model, are shown in Table E1. The final model after backward selection (Table 2) uses 5 predictors: increasing age (odds ratio [OR], 1.04/y; $P < .001$), cerebrovascular disease (OR, 1.54; $P = .01$), CKD (OR, 1.53; $P = .008$), symptomatic aneurysm (OR, 1.42; $P = .02$), and Crawford extent I (OR, 0.66; $P = .08$), II (OR, 1.61; $P = .01$), and IV (OR, 0.41; $P = .002$). We also developed 3 machine learning models: a random forest model, a support vector machine model, and a gradient boosting machine model. For the final hyperparameter selection, for the random forest model, we used 1000 trees with 10 variables considered per node; for the support vector machine, we used a cost of 1000 and a gamma of 10^{-5}; and for the gradient boosting machine, we built a model with 400 trees, a learning rate of 0.01, a maximum tree depth of 3, and a column sampling per tree of 0.4.

According to the median test set C-statistics from the 80:20, 1000-iteration cross-validation scheme, the MLR model was the most predictive (C-statistic, 0.68; IQR, 0.65-0.71). Of the machine learning models, the gradient boosting machine model was the most predictive (C-statistic, 0.67; IQR, 0.64-0.70), compared with 0.61 (IQR, 0.59-0.64) for random forest and 0.61 (IQR, 0.58-0.64) for support vector machine.

Nomogram

Based on its intuitive structure and superior predictive performance, the MLR model was converted into a nomogram (Figures 1 and 2). A given patient's characteristics can be mapped onto each of the 7 predictor variable rows (age through extent IV TAAA repair), and each variable yields a point contribution, which can be obtained by identifying the corresponding point value on the top row (labeled "points"). These 7 contributions are then summed and mapped on the "total points" line, and the corresponding position on the "predicted value" line is that patient's estimated risk of operative death.

TABLE 2. Predictors of operative mortality on multivariable logistic regression

Characteristic	OR	P value
Increasing age, y	1.04	**<.001**
Cerebrovascular disease	1.54	**.01**
Chronic kidney disease	1.53	**.008**
Symptomatic aneurysm	1.42	**.02**
Extent I TAAA repair	0.66	.08
Extent II TAAA repair	1.61	**.01**
Extent IV TAAA repair	0.41	**.002**

Bold type indicates significance at $P < .05$. *OR*, Odds ratio; *TAAA*, thoracoabdominal aortic aneurysm.

DISCUSSION

Although we evaluated several machine learning models to determine whether we could improve on the predictive effectiveness of MLR modeling with more modern computational approaches, we found that an updated MLR model had the greatest predictive utility. The 5 factors used in the final MLR model were age, cerebrovascular disease, CKD, symptomatic aortic aneurysm, and Crawford extent of repair. This final model was converted into a nomogram, a diagram used to depict predictive statistical models in a more intuitive format useful for patient counseling.

The risk factors that we identified generally follow from our previous work and the work of others. Previous studies have individually identified age,[2,3,7,15] CKD,[4,15-17] symptomatic aortic aneurysm,[18] and Crawford extent of repair[2] as strong predictors of operative mortality. Regarding the Crawford extent, the least mortal procedure was extent IV repair, followed by extent I repair; extent II repair was the most dangerous. Extent IV and extent I repairs usually involve a single body cavity (thoracic and abdominal, respectively); thus, these repairs have generally been found to be safer than extent III and extent II repairs, which require entering both cavities.[2,19] Although MLR models are not explicitly intended to be predictive, they are the models most commonly used to control for covariates when assessing operative mortality.[2-4,16,20]

In contrast, novel machine learning approaches for prediction have become more common in recent years. Pirruccello and colleagues[21] used least absolute shrinkage and selection operator regression to predict ascending aortic diameters from routine clinical data. Ostberg and colleagues[14] successfully leveraged several machine learning approaches to predict aortic rupture in patients with descending thoracic and thoracoabdominal aortic aneurysms.

Machine learning models are inherently nonparametric in that they are not based on assumptions of parametric model structures, such as the linear relationship between the independent variables and the log odds in an MLR model. This allows greater modeling flexibility and potentially can increase predictive effectiveness, especially for complex, nonlinear relationships between variables. Given this trend in statistical modeling, it seems counterintuitive that our simpler MLR model outperformed the machine learning models. However, a recent meta-analysis from Christodoulou and colleagues[22] showed that machine learning models do not always outperform more traditional MLR models for clinical prediction. This analysis included studies from many clinical disciplines, most commonly oncology and cardiovascular medicine. Our findings, although seemingly surprising given modern expectations,

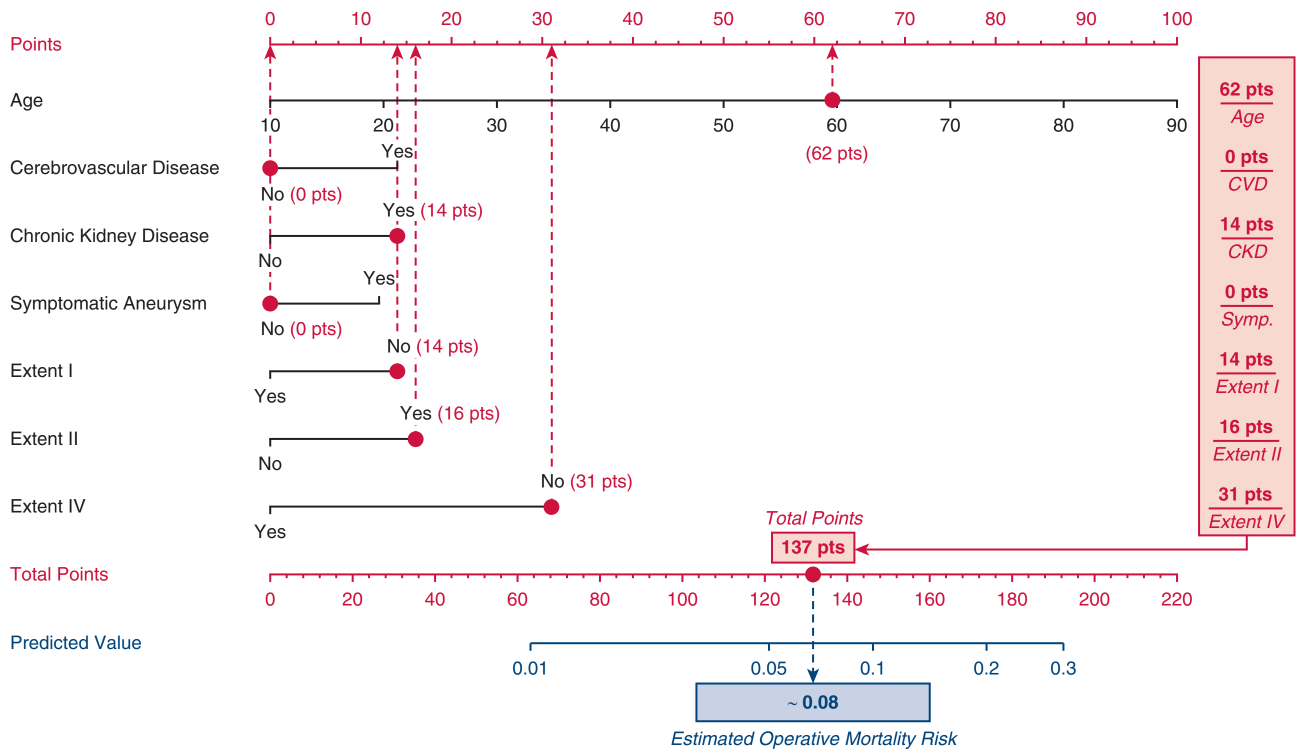


FIGURE 1. Nomogram constructed from the predictive multiple logistic regression model. The number of points each predictor contributes can be determined from the corresponding point count on the top line. For binary variables, the "no" value serves as the reference value. Regarding the 4 Crawford extents of repair, extent III serves as the reference value. The sum of these points is then mapped onto the "total points" line, and the corresponding location on the "predicted value" line corresponds to the patient's predicted risk of operative death. For example, a patient age 60 years (62 points) with chronic kidney disease (CKD; 14 points) undergoes an extent II repair (16 points, plus 14 points for not undergoing an extent I repair and 31 points for not undergoing an extent IV repair). This produces a total score of 137 points, which corresponds to an approximate operative mortality risk of 8%. *CVD*, Cerebrovascular disease.

are in line with the results of Christodoulou and colleagues and others.

This phenomenon has several potential causes. Christodoulou and colleagues[22] hypothesized that it involves the signal-to-noise ratio—a statistical concept that identifies the amount of random variation relative to variation driven by given predictors. Interestingly, machine learning models have proven effective for handling datasets with high signal-to-noise ratios but less effective for datasets with lower ratios,[23] exhibiting more sensitivity to noise and overfitting than more rigid, parametric models. Because medical research data tend to have a low signal-to-noise ratio, machine learning models may prove less effective for predicting clinical outcomes.[24] In addition, the flexibility of machine learning methods comes at the cost of having substantially more parameters to estimate compared to parametric models. This typically requires a large dataset for model training to achieve good generalizability, which might not be always available in clinical prediction problems. Indeed, in many contexts, there is often no significant difference in performance between more complex methods and simpler models such as logistic regression, particularly when analyzing structured data with naturally meaningful features,[25] as is common in medical settings. In our other study,[26] which involved a different disease and included follow-up analyses, we found that logistic regression yielded noninferior predictive performance compared with optimally weighting a range of machine learning methods. Overall, our findings and others suggest that parametric methods, featuring interpretability, can yield greater accuracy than more complex methods in medical settings.

Our findings suggest some key future opportunities. First, as mentioned earlier, Ostberg and colleagues[14] produced a model to predict rupture and dissection of descending aortic aneurysms—the risk that serves as the other prong of the risk management calculus for patients considering TAAA repair. Combining their results with ours could provide a unique opportunity to provide patients and surgeons with a dual risk calculation (risk of rupture/dissection vs operative mortality) to aid decision making, including the timing

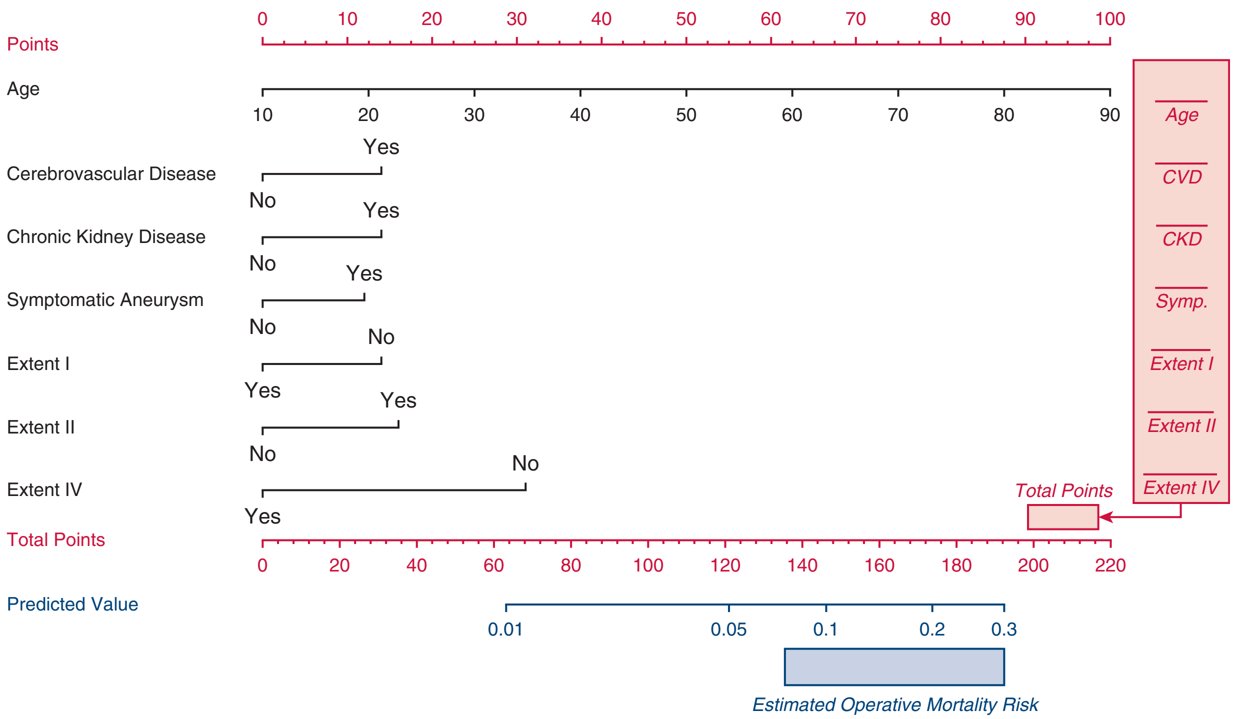


FIGURE 2. Unmarked nomogram for the predictive multiple logistic regression model for use in patient counseling. The number of points that each predictor contributes can be determined from the corresponding point count on the top line. The sum of these points is then mapped onto the "total points" line, and the corresponding location on the "predicted value" line corresponds to the patient's predicted risk of operative death. *CVD*, Cerebrovascular disease; *CKD*, chronic kidney disease.

of operations. Second, operative death is not the only potential surgical complication of TAAA repair, and producing risk models to predict other complications (paraplegia, prolonged ventilation, acute kidney injury necessitating dialysis) is important for patient counseling. Third, developing a dynamic predictive model that incorporates intraoperative variables (eg, transfusions, ischemic times) and postoperative variables (eg, urine output, serum creatinine, lactate, transfusions) ultimately could provide real-time risk prediction as an early warning system.

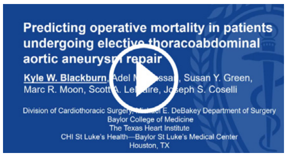
Our study has several limitations. First, as with any study of risk factors in surgical patients, some operative variables had to be simplified. Thus, our data do not fully reflect the unique presentations of the individual patients, each of whom received a tailored approach. Second, the preoperative variables that we studied were largely binary, with a limited number of continuous variables that may have favored a machine learning model; moreover, it is possible that lack of certain preoperative variables that are not directly captured in our database (eg, frailty) could have influenced the model. Third, our findings represent the experience of a single, high-volume tertiary care practice; thus, our results may be susceptible to selection bias and might not be generalizable to the average patient undergoing TAAA repair, especially repair performed by a low-volume team. Finally, this analysis examined repairs performed over a lengthy timespan (1986-2023) during which operative methods evolved[8] and the patient population may have changed. Therefore, some trends that we identified could have been affected by time; however, when we redid the analysis to include only patients treated in 2005 and after, the results remained largely unchanged (Tables E4 and E5).

CONCLUSIONS

In this study, we used a multivariable logistic regression model to identify 5 factors predictive of operative mortality: age, symptomatic aortic aneurysm, CKD, cerebrovascular disease, and Crawford extent of repair. A rigorous cross-validation scheme showed this model to be effective in predicting mortality, and the model was converted into an intuitive nomogram to aid surgeons in patient counseling. The next step in this work will be to validate the model, either through prospective data collection or by validating the model on cohorts from other institutions. Additionally, to aid holistic patient counseling, we plan to develop nomograms for other key postoperative complications of TAAA repair.

Webcast

You can watch a Webcast of this AATS meeting presentation by going to: https://www.aats.org/resources/predicting-operative-mortality-7077.



Conflict of Interest Statement

Dr Chatterjee has served on advisory boards for Edwards Lifesciences, Eagle Pharmaceuticals, La Jolla Pharmaceutical, and Baxter Lifesciences. Dr Moon advises Medtronic and Edwards Lifesciences. Dr LeMaire consults for Cerus and has served as a principal investigator for clinical studies sponsored by Terumo Aortic and CytoSorbents. Dr Coselli serves as principal investigator, consults for, and receives royalties and a departmental educational grant from Terumo Aortic; consults and participates in clinical trials for Medtronic and W.L. Gore & Associates; and participates in clinical trials for Abbott Laboratories, CytoSorbents, Edwards Lifesciences, and Artivion. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: aortic aneurysm, operative mortality, clinical decision rules, prognosis, nomograms, patient counseling, surgical risk, outcome assessment, health care

TABLE E1. Preliminary predictors of operative mortality after univariate and multivariable logistic regression

Characteristics	Univariate analysis OR	Univariate analysis P value	Multivariable analysis OR	Multivariable analysis P value
Increasing age, y	1.04	<**.001**	1.04	<**.001**
Heritable thoracic aortic disease	0.53	**.01**	1.79	.08
Genetic disorder*	0.61	.08	—	—
Marfan syndrome*	0.60	.09	—	—
Maximum distal aortic diameter	1.13	**.04**	1.07	.3
Diabetes	1.49	**.09**	1.46	.1
Cerebrovascular disease	1.85	<**.001**	1.44	**.04**
Previous myocardial infarction	1.39	.06	1.28	.2
Chronic kidney disease	2.08	<**.001**	1.51	**.01**
Chronic obstructive pulmonary disease	1.50	**.007**	1.22	.2
Tobacco use	1.40	.09	0.94	.8
Symptomatic aneurysm	1.38	**.03**	1.40	**.03**
Peripheral vascular disease	1.39	**.04**	1.16	.4
Extent I TAAA repair	0.56	**.003**	0.70	.1
Extent II TAAA repair	2.03	<**.001**	1.67	**.008**
Extent IV TAAA repair	0.44	<**.001**	0.40	**.001**

Bold type indicates statistical significance. Variables were selected for model entry if they were significantly associated with operative mortality at an alpha of 0.1 in the univariate logistic regression analysis. This preliminary model precedes backward selection (Table 2). *OR*, Odds ratio; *TAAA*, thoracoabdominal aortic aneurysm. *To prevent issues with collinearity, only 1 genetic variable could be included in the multivariate model; thus, the most significant predictor—heritable thoracic aortic disease—was selected.

TABLE E2. Hyperparameters evaluated for the machine learning models

Model type	Hyperparameters
Random forest	Number of variables considered: 2, 3, 4, 5, 10 Number of trees grown: 50, 100, 250, 500, 750, 1000
Support vector machine	Cost: 1, 10, 100, 1000 Gamma: 10^{-1}, 10^{-2}, 10^{-3}, 10^{-4}, 10^{-5}
Gradient boosting machine	Learning rate: 0.001, 0.005, 0.01, 0.05, 0.1 Number of trees: 100, 200, 300, 400, 500 Maximum tree depth: 2-8 Column sampling per tree: 0.4, 0.6, 0.8, 1.0

TABLE E3. Operative details of patients who underwent elective, open TAAA repair

Variable	All (N = 2884)	Early survivor (N = 2684)	Operative death (N = 200)	*P* value
Aortic clamp time, min, median (IQR) (N = 2827)	52 (39-68)	51 (38-67)	62 (45-77)	**<.001**
Unknown, n (%)	15 (0.5)	11 (0.4)	4 (2.0)	—
Management of visceral or renal arteries, n (%)				
Endarterectomy, stenting, or bypass graft	1239 (43.0)	1133 (42.2)	106 (53.0)	**.004**
Endarterectomy	740 (25.7)	677 (25.2)	63 (31.5)	.06
Stenting	234 (8.1)	208 (7.7)	26 (13.0)	**.01**
Bypass graft	799 (27.7)	724 (27.0)	75 (37.5)	**.002**
Adjuncts				
Hypothermic circulatory arrest, n (%)	42 (1.5)	35 (1.3)	7 (3.5)	**.03**
Left heart bypass, n (%)	1382 (47.9)	1266 (47.2)	116 (58.0)	**.004**
Left heart bypass time, min, median (IQR)	24 (18-29)	24 (18-29)	24 (19-29)	—
Cerebrospinal fluid drainage, n (%)	1429 (49.5)	1312 (48.9)	117 (58.5)	**.01**
Cold renal perfusion, n (%)	1748 (60.6)	1635 (60.9)	113 (56.5)	.2
Selective visceral perfusion, n (%)	777 (26.9)	699 (26.0)	78 (39.0)	**<.001**
Segmental artery reattachment, n (%)	1530 (53.1)	1413 (52.6)	117 (58.5)	.1
Other				
Incidental splenectomy, n (%)	331 (11.5)	294 (11.0)	37 (18.5)	**.002**

Bold type indicates statistical significance. *IQR*, Interquartile range; *TAAA*, thoracoabdominal aortic aneurysm.

TABLE E4. Predictors of operative mortality after multivariable logistic regression for the cohort restricted to patients treated in 2005 and later

Characteristics	OR	*P* value
Age, y	1.05	**<.001**
Cerebrovascular disease	1.62	.08
Chronic kidney disease	1.53	.09
Symptomatic aneurysm	NS	NS
Extent I TAAA repair	0.73	.4
Extent II TAAA repair	2.21	**.009**
Extent IV TAAA repair	0.16	**.001**

Bold type indicates statistical significance. *OR*, Odds ratio; *NS*, not significant; *TAAA*, thoracoabdominal aortic aneurysm.

TABLE E5. Evaluation of models for the cohort restricted to patients treated in 2005 and later

Model type	C-statistic, median (IQR)
Multivariable logistic regression	0.70 (0.66-0.74)
Random forest	0.62 (0.59-0.67)
Support vector machine	0.59 (0.55-0.64)
Gradient boosting machine	0.64 (0.60-0.68)

The test set C-statistic for each of the models evaluated with 1000 iterations of a cross-validation scheme. *IQR*, Interquartile range.