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ORIGINAL RESEARCH

STRUCTURAL

Predicting Outcomes in Patients With Tricuspid Regurgitation Undergoing Transcatheter Edge-to-Edge Repair Using an Artificial Intelligence–Derived Risk Score

The EuroTR Risk Score

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ABBREVIATIONS AND ACRONYMS

AI = artificial intelligence

AUC = area under the curve

EuroSCORE = European System for Cardiac Operative Risk Evaluation

NT-proBNP = N-terminal pro-brain natriuretic peptide

ROC = receiver-operating characteristic

RV = right ventricle/ventricular

sPAP = systolic pulmonary artery pressure

TAPSE = tricuspid annular plane systolic excursion

TR = tricuspid regurgitation

TRIO = Tricuspid Regurgitation Impact on Outcomes

T-TEER = tricuspid valve transcatheter edge-to-edge repair

XGB = extreme gradient boosting

ABSTRACT

BACKGROUND Risk stratification for tricuspid valve transcatheter edge-to-edge repair (T-TEER) is paramount in the decision-making process to appropriately select patients with severe tricuspid regurgitation.

OBJECTIVES The aim of this study was to develop and validate an artificial intelligence–driven risk score, the EuroTR (European Registry of Transcatheter Repair for Tricuspid Regurgitation) score, to predict 1-year mortality in patients undergoing T-TEER.

METHODS The EuroTR score was developed using data from the EuroTR registry, comprising 1,225 patients in the derivation cohort and 601 patients in the validation cohort. On the basis of 18 clinical, laboratory, echocardiographic, and hemodynamic parameters, an extreme gradient boosting algorithm was trained and independently validated against established risk models.

RESULTS Among the entire study cohort (N = 1,826), the overall 1-year survival rate was 82.1% (95% CI: 80.1%-84.2%), with no significant differences between the derivation and validation cohorts. The EuroTR score successfully stratified patients into low-risk and high-risk groups for 1-year mortality after T-TEER (HR: 4.26; 95% CI: 2.71-6.67; $P < 0.001$), and it significantly outperformed established risk scores such as the EuroScore and the TRI-SCORE in the validation cohort. Beyond mortality prediction (Harrell's C index [validation cohort] = 0.741; 95% CI: 0.699-0.783), increasing EuroTR score values were associated with a higher likelihood of a clinically relevant combined endpoint of 1-year mortality, need for heart failure hospitalization, or persistent dyspnea corresponding to NYHA functional class ≥III. The likelihood of poor outcomes increased from 30.6% in patients with the lowest EuroTR scores (EuroTR risk rank <5%) to 85.5% in the highest risk group (EuroTR risk rank ≥95%). The EuroTR score's performance was confirmed in several subgroups (atrial vs nonatrial tricuspid regurgitation, TRILUMINATE-eligible vs TRILUMINATE-noneligible patients, and patients with vs without cardiac implantable electronic device leads).

CONCLUSIONS The EuroTR score offers an easy-to-use, externally validated, accurate risk stratification tool for patients undergoing T-TEER. It supports personalized treatment strategies and the design of future clinical trials, helping optimize patient selection and enhance shared decision-making within multidisciplinary heart teams. (JACC Cardiovasc Interv. 2026;19:631-646) © 2026 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Tricuspid valve transcatheter edge-to-edge repair (T-TEER) has emerged as a promising treatment option for symptomatic patients with right heart failure and severe tricuspid regurgitation (TR).[1-6] Although important advances have been made in understanding the pathophysiology of right heart failure, TR, and the technical aspects of T-TEER, accurate prediction of postprocedural outcomes such as 1-year mortality and symptomatic alleviation remains a major clinical challenge. Accurate risk stratification is crucial for selecting appropriate candidates, guiding clinical decision-making, and optimizing patient outcomes. However, existing risk assessment models, many of which were originally developed for short-term outcomes following cardiac surgery, often rely on a limited number of clinical variables and may not fully capture the complexity and heterogeneity of patients with TR undergoing T-TEER.[7-9]

The EuroTR (European Registry of Transcatheter Repair for Tricuspid Regurgitation) provides a large, contemporary data set with detailed patient

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characteristics, echocardiographic and hemodynamic parameters, as well as procedural and clinical outcomes; thus, the EuroTR registry offers a unique resource for refining our understanding of T-TEER in the treatment of patients with symptomatic TR.[2,10,11] Leveraging large data sets with modern machine learning algorithms offers the opportunity to develop a disease-specific risk prediction model that can identify complex risk patterns that might be missed by traditional statistical methods.

Accordingly, the aim of this study was to develop and validate a robust, reliable risk score for predicting mortality and clinical outcomes in patients undergoing T-TEER, using the extensive EuroTR data set. This novel risk model was designed to surpass the predictive accuracy of existing risk models. By providing enhanced predictive accuracy, the proposed risk score may assist clinicians in identifying high-risk TR patients and offer valuable insights to inform clinical decision-making within interdisciplinary heart teams performing T-TEER on symptomatic TR patients.

METHODS

STUDY COHORT AND PROCEDURAL TECHNIQUE. The EuroTR registry is a retrospective international multicenter registry that includes consecutive patients with severe symptomatic TR undergoing T-TEER (NCT06307262). For the development of a risk score, patients from 23 European heart valve centers were included who underwent T-TEER between 2016 and 2022. Patients undergoing concomitant mitral transcatheter edge-to-edge repair were excluded from this analysis. Patient selection was based on interdisciplinary heart team consensus at each center, considering comorbidities, anatomy, symptoms, and life expectancy, after optimizing medical therapy. T-TEER was performed using the PASCAL device (Edwards Lifesciences) or the MitraClip or TriClip system (Abbott Cardiovascular), as previously described.[12,13] The study adheres to the principles of the Declaration of Helsinki and received approval from the local ethics committee of each participating center.

STUDY VARIABLES. Clinical patient characteristics included demographic data (age, sex, and body mass index), comorbidities (diabetes mellitus, arterial hypertension, coronary artery disease, atrial fibrillation or flutter, and chronic obstructive pulmonary disease), as well as markers of renal and liver function (eg, estimated glomerular filtration rate and bilirubin levels). Baseline levels of N-terminal pro-brain natriuretic peptide (NT-proBNP) and hemoglobin concentration were recorded. Clinical signs of heart failure were assessed, with pleural effusion considered indicative of left heart failure, while peripheral edema, ascites, and jugular vein distension were evaluated as signs of right heart failure. Functional capacity was classified according to the NYHA functional classification. In addition, the presence of a transtricuspid right ventricular (RV) lead was recorded. Medication data including information on diuretic agents, β-blockers, and renin-angiotensin system inhibitors were also collected. Existing risk scores were calculated as previously described.[14]

Right heart catheterization prior to T-TEER was performed according to each center's internal standards and included measurements of pulmonary artery pressures—systolic pulmonary artery pressure (sPAP), diastolic pulmonary artery pressure, and mean pulmonary artery pressure—as well as mean pulmonary capillary wedge pressure, right atrial v-wave, cardiac output, and pulmonary vascular resistance.[11] Transthoracic echocardiography, performed by experienced operators at each center, assessed TR severity using a 5-grade approach, combining quantitative, semiquantitative, and qualitative parameters in line with European guidelines.[15,16] Quantitative measurements included effective regurgitant orifice area and regurgitant volume determined by the proximal isovelocity surface area method, while vena contracta width was used for semiquantitative assessment. Left ventricular function was evaluated by left ventricular ejection fraction, while RV function was assessed using tricuspid annular plane systolic excursion (TAPSE) and RV fractional area change. TR etiology was classified as primary, secondary, or mixed according to Tricuspid Valve Academic Research Consortium guidelines.[17] Atrial secondary TR was defined by a ratio of end-systolic right atrial area to RV area ≥1.5 in the presence of preserved RV function (TAPSE >17 mm).[17,18] RV-to-pulmonary arterial coupling was calculated as the ratio of TAPSE to sPAP.[19-21] TRILUMINATE ineligibility criteria included TR severity <3+, NYHA functional class <II, sPAP >70 mm Hg, mean pulmonary artery pressure >30 mm Hg plus transpulmonary gradient >17 mm Hg, mitral regurgitation ≥3+ or concomitant mitral transcatheter edge-to-edge repair, left ventricular ejection fraction ≤20%, or need for dialysis.[1,4]

ENDPOINTS, FOLLOW-UP, AND DEFINITIONS. The EuroTR patient cohort was divided on a per-site basis into a derivation cohort for score development and a respective validation cohort. Targeting a

patient split ratio of 2:1, the patients from 14 study sites were included in the derivation cohort, while the patients from 9 different study sites were used for score validation. This method allowed an independent, "never-before-seen" data set of sufficient size for validation.

The primary endpoint for the development of the EuroTR score in the derivation cohort was 1-year all-cause mortality. This same endpoint was used for score validation and comparison with other risk models in the validation cohort. Secondary analyses included a composite of 1-year mortality, heart failure hospitalization, and persistence in NYHA functional class III or IV at 1-year follow-up, allowing a comprehensive evaluation of T-TEER efficacy and futility across EuroTR scores. Follow-up was conducted through regular visits or telephone interviews with patients or their next of kin, depending on each center's protocol, assessing NYHA functional class, survival, and hospitalization status.

STATISTICAL ANALYSIS. Categorical variables are presented as numbers and percentages, while continuous variables are expressed as median (Q1-Q3), given their non-normal distribution. The chi-square or Fisher exact test was applied to assess associations between categorical variables, and the independent-samples Wilcoxon test was used for comparisons of continuous variables. Kaplan-Meier curves were created to illustrate survival data, and Cox proportional hazards models were used to estimate HRs and 95% CIs.

An extreme gradient boosting (XGB) algorithm (R package xgboost) was selected as the machine learning technique of choice for a binary classification task such as mortality prediction, developed on the derivation cohort and validated on an independent data set from 9 institutions within the EuroTR registry (validation cohort, "never-before-seen" data set). Patients without known 1-year survival status after T-TEER were excluded before training of the XGB algorithm. Before searching for potential input parameters to train the prediction model, those parameters with <50% data completeness in the derivation data set were excluded (patterns of missingness were formally assessed using Little's test for missing completely at random using the R package naniar). All other remaining parameters (clinical, echocardiographic, laboratory, hemodynamic, and medication data) were entered into to the XGB algorithm for further selection through recursive feature elimination as described in the following (Supplemental Table 1). To train the algorithm on as many data as possible, missing data points in the derivation cohort were imputed using an established random forest algorithm (R package missForest), which generated a single complete data set through iterative nonparametric prediction[22]; however, these imputed data were not used in subsequent descriptive analyses, such as baseline comparisons. The XGB algorithm was initially trained on all selected input parameters. Those parameters were then ranked according to their feature importance to predict 1-year all-cause mortality. The least important features were progressively discarded, and the model was refitted each time. Hyperparameters of the XGB algorithm (such as learning rate and maximum depth of each tree) were optimized within the derivation cohort by testing various settings in a grid search approach and a 3-fold cross-validation setting. At each iteration, 2 folds were used for training and 1 fold for testing, rotating across folds, with early stopping applied to avoid overfitting. The final model was subsequently refitted on the entire derivation cohort using the optimal hyperparameters and number of boosting rounds as determined during cross-validation (see Supplemental Table 2 for a detailed description of the final hyperparameters of the XGB model). The metric of choice to evaluate the model performance was area under the curve (AUC) from receiver-operating characteristic (ROC) analysis. Shapley additive explanations values were calculated as a state-of-the-art metric derived from cooperative game theory to quantify the contribution of input variables to the model's prediction (R package SHAPforxgboost).[23-25] For the independent external validation cohort, no imputation was performed; instead, the inherent ability of the XGB algorithm to handle missing values by assigning default tree-splitting directions was exploited. Comparisons against other risk scores in the validation cohort were therefore performed on available data only. Finally, a free online calculator was created on the basis of Shiny R (www.EuroTR.eu). HRs for survival according to the EuroTR score were estimated using Cox proportional hazards models. The proportional hazards assumption was tested using Schoenfeld residuals, and no violations were detected. The predictive performance of the EuroTR score was compared with existing risk scores (such as the European System for Cardiac Operative Risk Evaluation II [EuroSCORE II] score, TRI-SCORE, TRIO (Tricuspid Regurgitation Impact on Outcomes) score, TRIVALVE score, and the scores of LaPar et al[9] and Wang et al[7]) using ROC analysis, and statistical differences among the ROC curves were assessed using DeLong's 2-tailed test for correlated ROC curves.

TABLE 1 Comparison of Demographic and Clinical Characteristics

	All Patients (N = 1,826)	Derivation (n = 1,225)	Validation (n = 601)	*P* Value
Age, y	80.0 (76.0-83.3)	80.4 (76.2-83.4)	79.6 (75.3-83.0)	0.003
Male	855/1,826 (46.8)	579/1,225 (47.3)	276/601 (45.9)	0.624
BMI, kg/m^2	25.3 (22.8-28.8)	25.4 (22.9-29.1)	25.1 (22.5-28.3)	0.072
Arterial hypertension	1,351/1,663 (81.2)	855/1,062 (80.5)	496/601 (82.5)	0.343
Diabetes mellitus	416/1,662 (25.0)	266/1,061 (25.1)	150/601 (25.0)	1.0
Dyslipidemia	822/1,659 (49.5)	575/1,061 (54.2)	247/598 (41.3)	<0.001
History of myocardial infarction	186/1,672 (11.1)	114/1,071 (10.6)	72/601 (12.0)	0.452
History of coronary disease	742/1,795 (41.3)	499/1,194 (41.8)	243/601 (40.4)	0.616
History of stroke or TIA	143/1,238 (11.6)	75/637 (11.8)	68/601 (11.3)	0.870
History of COPD	305/1,698 (18.0)	191/1,097 (17.4)	114/601 (19.0)	0.463
AF or atrial flutter	1,630/1,792 (91.0)	1,072/1,191 (90.0)	558/601 (92.8)	0.059
eGFR, mL/min	45 (31.5-59.0)	44 (31-59)	45 (33-59)	0.405
AST, U/L	28 (23-35)	29 (23-35)	28 (23-35)	0.581
ALT, U/L	18 (14-25)	19 (13-25)	18 (14-25)	0.830
Bilirubin, mg/dL	0.8 (0.6-1.2)	0.8 (0.6-1.1)	0.8 (0.6-1.2)	0.036
Alkaline phosphatase, U/L	109 (81-146)	114 (86-151)	102 (76-139)	0.029
Hemoglobin, g/dL	11.9 (10.4-13.2)	12.0 (10.6-13.3)	11.6 (10.3-12.9)	<0.001
NT-proBNP, pg/mL	2,440 (1,329-4,719)	2,515 (1,330-4,807)	2,254 (1,326-4,573)	0.236
NYHA functional class				
I	24/1,810 (1.3)	20/1,217 (1.6)	4/593 (0.7)	0.141
II	267/1,810 (14.8)	201/1,217 (16.5)	66/593 (11.1)	0.003
III	1,274/1,810 (70.4)	851/1,217 (69.9)	423/593 (71.3)	0.575
IV	245/1,810 (13.5)	145/1,217 (11.9)	100/593 (16.9)	0.005
Peripheral edema	924/1,495 (61.8)	512/895 (57.2)	412/600 (68.7)	<0.001
Ascites	199/1,491 (13.3)	98/892 (11.0)	101/599 (16.9)	0.001
Jugular vein distension	115/742 (15.5)	20/352 (5.7)	95/390 (24.4)	<0.001
Any RV lead	539/1,391 (38.7)	364/790 (46.1)	175/601 (29.1)	<0.001
EuroSCORE II, %	4.6 (2.5-7.6)	4.4 (2.3-7.5)	4.8 (2.9-8.1)	0.005
TRI-SCORE, %	6 (4-7)	6 (4-7)	6 (5-7)	0.269
Medications				
Loop diuretic agent	1,550/1,667 (93.0)	985/1,068 (92.2)	565/599 (94.3)	0.132
Thiazide diuretic agent	241/1,164 (20.7)	123/568 (21.7)	118/596 (19.8)	0.478
MRA	711/1,669 (42.6)	425/1,070 (39.7)	286/599 (47.7)	0.002
β-blocker	1,417/1,671 (84.8)	923/1,072 (86.1)	494/599 (82.5)	0.056
RASI	780/1,335 (58.4)	438/740 (59.2)	342/595 (57.5)	0.566
SGLT-2 inhibitor	148/897 (16.5)	100/402 (24.9)	48/495 (9.7)	<0.001

Values are median (Q1-Q3) or n/N (%), unless otherwise indicated.

AF = atrial fibrillation; ALT = alanine aminotransferase; AST = aspartate aminotransferase; BMI = body mass index; CAD = coronary artery disease; COPD = chronic obstructive pulmonary disease; eGFR = estimated glomerular filtration rate; EuroSCORE = European System for Cardiac Operative Risk Evaluation; MRA = mineralocorticoid receptor antagonist; NT-proBNP = N-terminal pro-brain natriuretic peptide; RASI = renin-angiotensin system inhibitor; RV = right ventricular; SGLT-2 = sodium-glucose cotransporter-2; TIA = transient ischemic attack.

P values ≤0.05 were considered to indicate statistical significance. All statistical analyses were performed using R version 4.3.2 (R Foundation for Statistical Computing; see Supplemental Table 3 for a complete list of R packages used in this study).

RESULTS

OVERALL STUDY COHORT. This analysis included 1,826 patients who underwent T-TEER for severe TR across 23 sites. The derivation cohort included 1,225 patients from 14 centers, while the validation cohort consisted of 601 patients from the remaining 9 centers, resulting in a 2:1 split ratio (**Table 1**). The derivation and validation cohorts were comparable in terms of center experience (derivation cohort, 9 [Q1-Q3: 4-21] patients/y; validation cohort, 7 [Q1-Q3: 4-11] patients/y; $P = 0.179$). The average age was 78.9 ± 7.2 years, 46.8% were men, and 91.0% had atrial fibrillation. Diuretic use was high, including loop diuretic

TABLE 2 Comparison of Echocardiographic Characteristics

	All Patients (N = 1,826)	Derivation (n = 1,225)	Validation (n = 601)	*P* Value
TR etiology				
Primary	106/1,780 (6.0)	80/1,182 (6.8)	26/598 (4.3)	0.053
Secondary	1,527/1,780 (85.8)	1,019/1,182 (86.2)	508/598 (84.9)	0.518
Mixed	147/1,780 (8.3)	83/1,182 (7.0)	64/598 (10.7)	0.010
TR severity				
Severe	863/1,826 (47.3)	571/1,225 (46.6)	292/601 (48.6)	0.457
Massive	598/1,826 (32.7)	420/1,225 (34.3)	178/601 (29.6)	0.052
Torrential	365/1,826 (20.0)	234/1,225 (19.1)	131/601 (21.8)	0.197
TR EROA, cm^2	0.57 (0.41-0.80)	0.57 (0.40-0.80)	0.57 (0.42-0.80)	0.869
TR RegVol, mL	48 (36-64)	49 (37-65)	45 (34-60)	0.005
TR vena contracta, mm	11 (8-14)	11 (8-14)	10 (8-14)	0.197
TAPSE, mm	17 (14-20)	17 (14-20)	17 (14-20)	0.046
RV FAC, %	39 (32-47)	40 (33-48)	37 (30-44)	<0.001
RVEDA, cm^2	26 (20-32)	25 (20-32)	27 (21-33)	0.005
RVESA, cm^2	16 (12-21)	15 (12-20)	17 (13-21)	<0.001
RV midventricular diameter, mm	40 (35-46)	40 (34-46)	40 (35-46)	0.693
RV length, mm	70 (63-78)	68 (60-77)	71 (64-79)	<0.001
TV annulus diameter, mm	44 (39-49)	44 (38-48)	45 (41-50)	<0.001
RAA, cm^2	34 (28-43)	34 (28-42)	34 (29-44)	0.413
$sPAP_{echo}$, mm Hg	41 (33-51)	42 (33-52)	40 (32-50)	0.023
Coaptation gap, mm	6 (4-8)	6 (4-8)	6 (4-8)	0.848
Tenting height, mm	7 (5-10)	7 (6-9)	8 (5-11)	0.017
Tenting area, cm^2	1.7 (1.2-2.5)	1.7 (1.1-2.4)	1.7 (1.2-2.6)	0.540
LVEF, %	55 (47-60)	55 (45-60)	55 (50-60)	0.027
LVEDD, mm	48 (43-53)	48 (43-53)	48 (43-53)	0.441

Values are n/N (%) or median (Q1-Q3), unless otherwise indicated.

EROA = effective regurgitant orifice area; FAC = fractional area change; LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; RAA = right atrial area; RegVol = regurgitant volume; RV = right ventricular; RVEDA = right ventricular end-diastolic area; RVESA = right ventricular end-systolic area; $sPAP_{echo}$ = systolic pulmonary artery pressure on echocardiography; TR = tricuspid regurgitation; TV = tricuspid valve.

agents, thiazides, aldosterone antagonists, and sodium-glucose cotransporter-2 inhibitors. Severe, massive, and torrential TR was diagnosed in 863 (47.3%), 598 (32.7%), and 365 (20.0%) patients, respectively, with 85.8% of cases attributed to secondary etiology. Patient demographic, clinical, laboratory, echocardiographic, and hemodynamic characteristics are summarized in **Tables 1, 2, and 3**. Importantly, 80.1% of patients achieved reductions in TR severity to moderate or less TR (≤2+) at discharge (**Table 4**).

The median follow-up duration for the entire study population was 12.1 months (Q1-Q3: 3.4-23.3 months; derivation cohort, 10.8 months [Q1-Q3: 2.6-21.2 months]; validation cohort, 13.6 months [Q1-Q3: 7.2-23.9 months]). Survival rates at 1 and 2 years after T-TEER for all patients were 82.1% (95% CI: 80.1%-84.2%) and 69.3% (95% CI: 66.5%-72.3%), respectively, with no statistically significant differences between the derivation and validation cohorts (Supplemental Figure 1).

Within the entire study population, a total of 209 patients (11.4%) were rehospitalized for heart failure after T-TEER, occurring after a median time of 4.7 months (Q1-Q3: 1.8-9.2 months). Importantly, no significant differences in time to rehospitalization for heart failure were observed when comparing derivation and validation cohorts ($P = 0.259$).

At baseline, 83.9% of patients presented with exertional dyspnea corresponding to NYHA functional class III or IV (derivation cohort, 81.8%; validation cohort, 88.2%; $P = 0.001$). Among those patients presenting at 1-year follow-up after T-TEER, a significant improvement in NYHA functional class was observed, with only 37.1% of patients from the entire study cohort persistently in NYHA functional class ≥ III (derivation cohort, 39.8%; validation cohort, 34.3%; $P = 0.111$).

DERIVATION OF THE EuroTR SCORE. Within the derivation cohort (1,225 patients from 14 study sites), recursive feature elimination reduced 60 potential

input parameters to 18 final parameters, covering a comprehensive set of clinical, laboratory, echocardiographic, and hemodynamic data. Overall, data completeness across these parameters before imputation was 87.1% (10,925 of 12,546 data points available). Some variables were fully complete (eg, age), while others showed moderate missingness (eg, TAPSE, 9.6%; tricuspid annular diameter, 14.3%; estimated glomerular filtration rate, 6.0%), and a few had higher rates of missingness (eg, right atrial area, 33%; mean pulmonary artery pressure, 33%; "any sign of right heart failure," 32%). Patterns of missingness are shown in Supplemental Figure 2. Inspection of patterns shows that missing values were not evenly distributed but tended to occur in specific echocardiographic and hemodynamic parameters. Missing values in the derivation cohort were imputed using a dedicated random forest algorithm. This procedure generated a single complete data set through iterative nonparametric imputation for subsequent training of the XGB model for mortality prediction. In the following, the 1-year mortality prediction of the XGB algorithm is referred to as the EuroTR score. The algorithm was trained until reaching an AUC of 0.838 (95% CI: 0.803-0.873) in the derivation cohort (Supplemental Figure 3). The 5 parameters with the strongest contribution to 1-year mortality prediction were (in order of predictive importance) hemoglobin, NT-proBNP, TAPSE, the presence of pleural effusion, and TR vena contracta width (**Central Illustration**). Balancing sensitivity and specificity in terms of 1-year mortality prediction, a Youden index-based cutoff value of the 64th score percentile was identified. Dichotomizing the study population according to this threshold allowed optimized differentiation of surviving and nonsurviving patients, with those patients classified as high risk by their EuroTR scores showing an HR of 5.64 (95% CI: 3.92-8.10; $P < 0.001$) for 1-year mortality after T-TEER (Supplemental Figure 4). Nonsurvivors were detected with sensitivity of 73.2% (95% CI: 66.0%-80.3%) and specificity of 73.4% (95% CI: 69.7%-77.1%) (Supplemental Table 4).

VALIDATION OF THE RISK SCORE. In the validation cohort (601 patients from 9 study sites), the EuroTR score yielded an AUC of 0.763 (95% CI: 0.715-0.811) for 1-year mortality prediction (**Figure 1**). Harrell's C index confirmed good discrimination when accounting for censoring of survival data at 1 year (0.741; 95% CI: 0.699-0.783). Moreover, calibration plots demonstrated good agreement between predicted and observed 1-year mortality in the low- to intermediate-risk range. At higher predicted risk levels, the EuroTR score tended to overestimate mortality, particularly in the validation cohort (Supplemental Figure 5). Applying the aforementioned EuroTR score threshold classified 356 patients (59.2%) as lower risk and 245 patients (40.8%) as higher risk for 1-year mortality (**Figure 2**). Kaplan-Meier curves demonstrated 1-year survival rates of 90.9% (95% CI: 87.7%-94.4%) in the low-risk group and 66.8% (95% CI: 60.8%-73.4%) in the high-risk group, with an HR of 4.26 (95% CI: 2.72-6.67; $P < 0.001$) (**Figure 3**). The predictive accuracy for 1-year mortality was 65.9% (95% CI: 61.4%-70.4%), with sensitivity, specificity, positive predictive value, and negative predictive value of 73.5% (95% CI: 64.7%-82.2%), 63.7% (95% CI: 58.5%-68.8%), 37.3% (95% CI: 30.5%-44.1%), and 89.1% (95% CI: 85.1%-93.0%), respectively (Supplemental Table 5).

COMPARISON OF THE EuroTR RISK SCORE WITH OTHER SCORES. The predictive performance of the EuroTR score regarding 1-year mortality prediction following T-TEER was then compared with that of 6 other risk scores in the validation cohort. The EuroTR risk score significantly outperformed 4 risk scores that are used for the prediction of early surgical mortality (EuroSCORE II, TRI-SCORE, TRIO score,

TABLE 3 Comparison of Characteristics From Right Heart Catheterization

	All Patients (N = 1,826)	Derivation (n = 1,225)	Validation (n = 601)	*P* Value
Systolic PAP, mm Hg	44 (35-55)	44 (35-55)	43 (35-55)	0.894
Diastolic PAP, mm Hg	18 (13-23)	17 (13-22)	19 (14-24)	<0.001
Mean PAP, mm Hg	28 (23-35)	28 (23-35)	29 (23-35)	0.901
Mean PCWP, mm Hg	18 (14-23)	18 (14-23)	18 (13-24)	0.330
PVR, WU	2.4 (1.6-3.6)	2.8 (1.8-3.9)	2.3 (1.6-3.3)	0.002
RA v-wave, mm Hg	17 (11-22)	16 (12-23)	17 (11-22)	0.511
CO, mL/min	3.9 (3.2-4.9)	3.7 (3.0-4.5)	4.1 (3.5-5.2)	<0.001

Values are median (Q1-Q3), unless otherwise indicated.
CO = cardiac output; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; RA = right atrial; WU = Wood units.

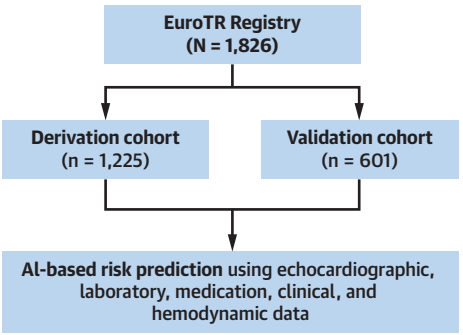
TABLE 4 Comparison of Residual Tricuspid Regurgitation Severity

	All Patients (N = 1,826)	Derivation (n = 1,225)	Validation (n = 601)	*P* Value
Moderate or less	1,427/1,781 (80.1)	938/1,181 (79.4)	489/600 (81.5)	0.330
Severe	291/1,781 (16.3)	204/1,181 (17.3)	87/600 (14.5)	0.153
Massive	47/1,781 (2.6)	30/1,181 (2.5)	17/600 (2.8)	0.835
Torrential	16/1,781 (0.9)	9/1,181 (0.8)	7/600 (1.2)	0.555

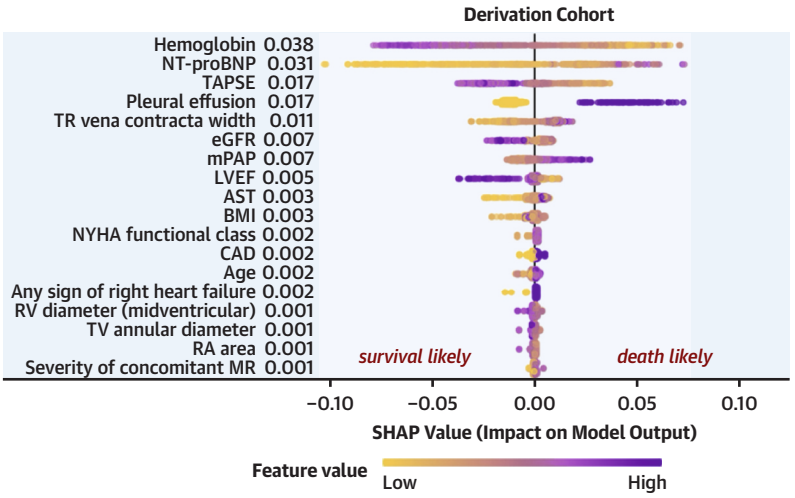
Values are n/N (%), unless otherwise indicated.

CENTRAL ILLUSTRATION **The EuroTR Score: An AI-Derived Risk Score to Predict 1-Year Mortality and Poor Clinical Outcome in Patients Undergoing T-TEER**

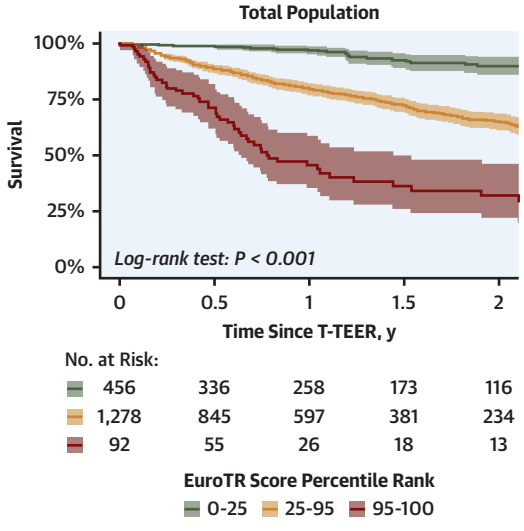
Study Flow



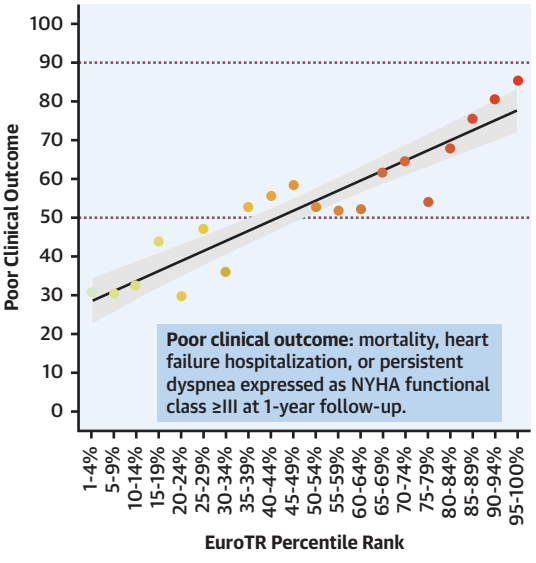
Contributing Parameters



Survival Stratified by EuroTR Score Rank



EuroTR Score and Poor Clinical Outcome

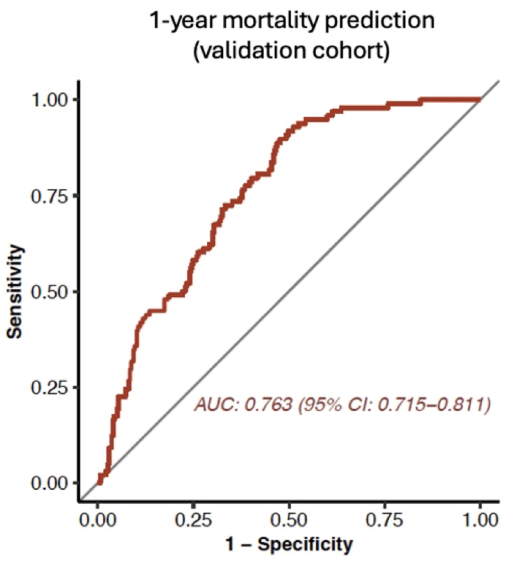


- The EuroTR Score successfully stratified patients into low-risk and high-risk groups for 1-year mortality after T-TEER (HR: 4.26, 95% CI: 2.71-6.67; $P < 0.001$).
- The EuroTR Score outperformed established risk scores in the validation cohort.
- Beyond mortality prediction, increasing EuroTR score values were associated with a higher likelihood of poor clinical outcome.
- The EuroTR Score performance was confirmed in several subgroups.
- An online calculator is available at www.EuroTR.eu.

Hausleiter J, et al. JACC Cardiovasc Interv. 2026;19(5):631-646.

The EuroTR (European Registry of Transcatheter Repair for Tricuspid Regurgitation) score offers an easy-to-use, externally validated, accurate risk stratification tool for patients undergoing tricuspid valve transcatheter edge-to-edge repair (T-TEER). AI = artificial intelligence; AST = aspartate aminotransferase; BMI = body mass index; CAD = coronary artery disease; eGFR = estimated glomerular filtration rate; LVEF = left ventricular ejection fraction; mPAP = mean pulmonary artery pressure; MR = mitral regurgitation; NT-proBNP = N-terminal pro-brain natriuretic peptide; RA = right atrial; RV = right ventricular; SHAP = Shapley additive explanations; TAPSE = tricuspid annular plane systolic excursion; TR = tricuspid regurgitation; TV = tricuspid valve.

FIGURE 1 Receiver-Operating Characteristic Curve for 1-Year Mortality Prediction Using the EuroTR Risk Score



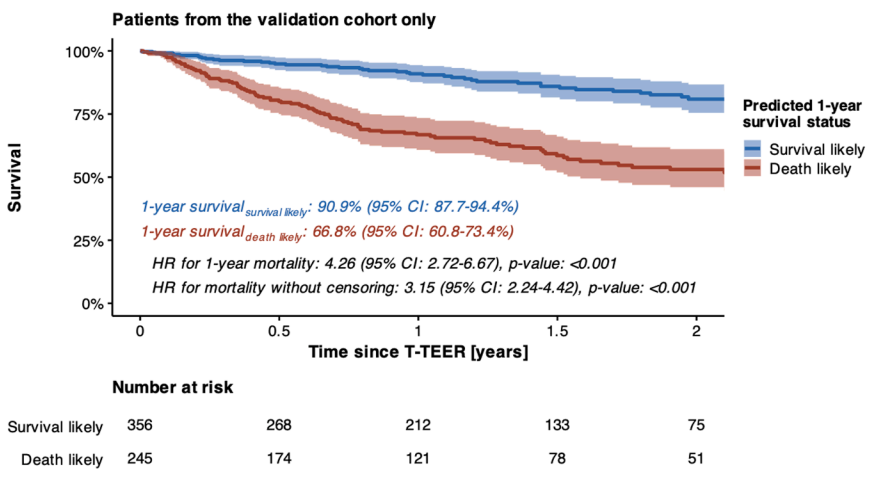
The area under the curve for prediction of 1-year mortality in the validation cohort was 0.763 (95% CI: 0.715-0.811).
AUC = area under the curve; EuroTR = European Registry of Transcatheter Repair for Tricuspid Regurgitation.

and the scores of LaPar et al[9]), as displayed in the respective ROC curves in Figure 3 and according to Harrell's C index in Supplemental Tables 6 and 7. Furthermore, the EuroTR score exceeded the performance of the score of Wang et al,[7] which predicts long-term mortality in patients with isolated TR (Figure 3). Finally, the EuroTR score also outperformed the TRIVALVE score, which is the only score to date that has been derived from patients treated percutaneously for severe TR (Figure 3).

EARLY MORTALITY PREDICTION. The entire EuroTR cohort of 1,826 patients (both derivation and validation cohorts) was ranked according to EuroTR score (Figure 4). Among patients from the EuroTR registry, 30-day mortality after T-TEER was low at 1.3%. Despite being developed for 1-year outcome prediction, the EuroTR score also identified patients at increased risk for early mortality. Kaplan-Meier curves showed 30-day survival rates of 99.2% (95% CI: 98.7%-99.8%) in the low risk group and 97.8% (95% CI: 96.7%-98.9%) in the high-risk group, with an HR of 2.86 (95% CI: 1.20-6.81; $P = 0.018$) (Figure 5).

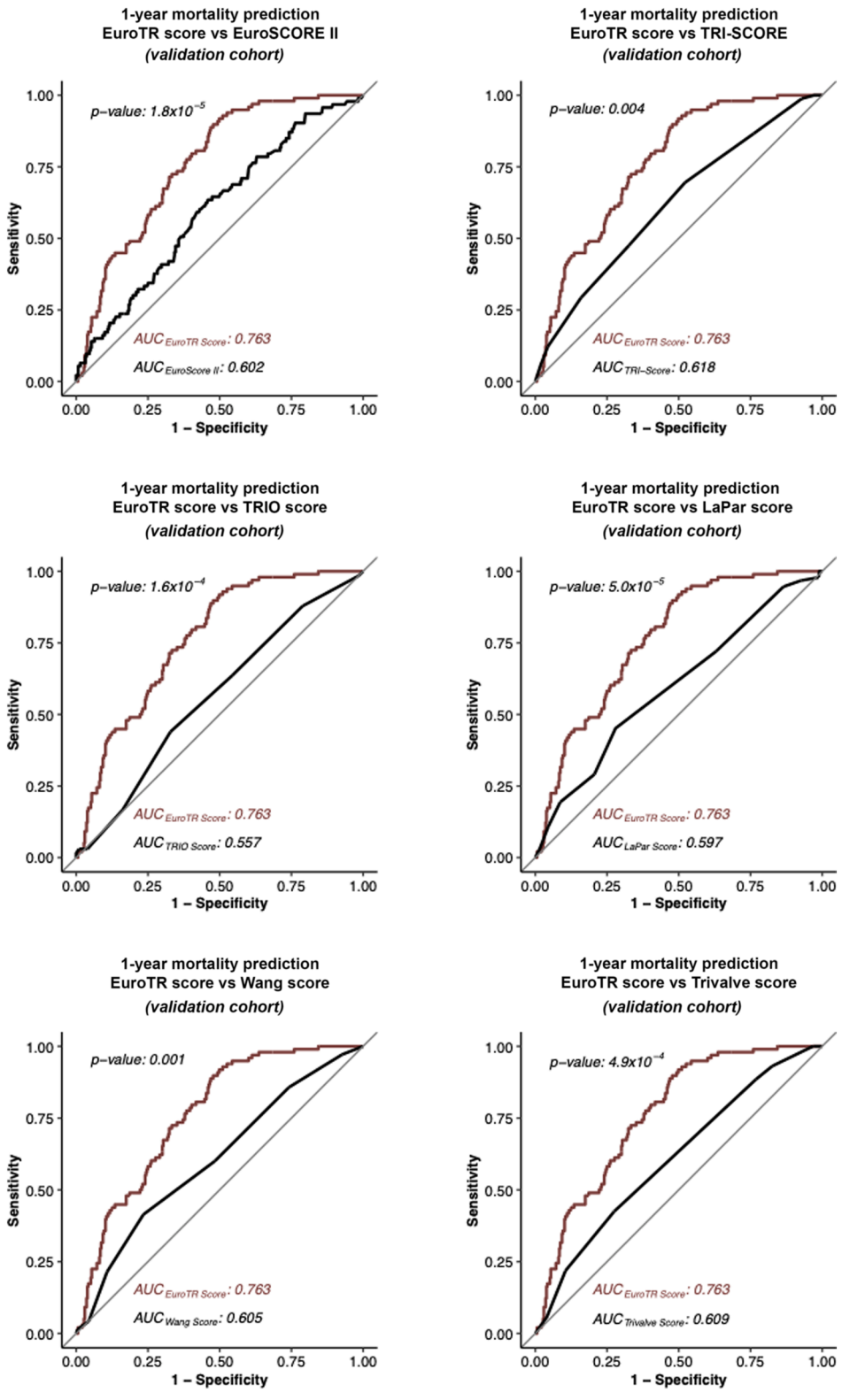
ASSESSMENT OF T-TEER UTILITY AND RISK STRATIFICATION. The entire EuroTR cohort (both derivation and validation cohorts, N = 1,826) was categorized into risk groups according to their EuroTR scores, with increasing risk for 1-year mortality: low risk (<25th score percentile), intermediate risk (25th to 95th score percentile), and high risk (≥95th score percentile) (Central Illustration). One- and 2-year survival rates were 97.0% and 90.0% in low-risk patients, 79.6% and 64.9% in

FIGURE 2 2-Year Survival According to the EuroTR Risk Score



Applying an optimized EuroTR (European Registry of Transcatheter Repair for Tricuspid Regurgitation) score threshold to the validation cohort yielded prognostic significance regarding 1- and 2-year survival.

FIGURE 3 Prognostic Performance of the EuroTR Risk Score Compared With Existing Scores



The EuroTR risk score significantly outperformed 4 risk scores that are used for the prediction of early surgical mortality. EuroSCORE = European System for Cardiac Operative Risk Evaluation; TRIO = Tricuspid Regurgitation Impact on Outcomes; other abbreviations as in Figure 1.

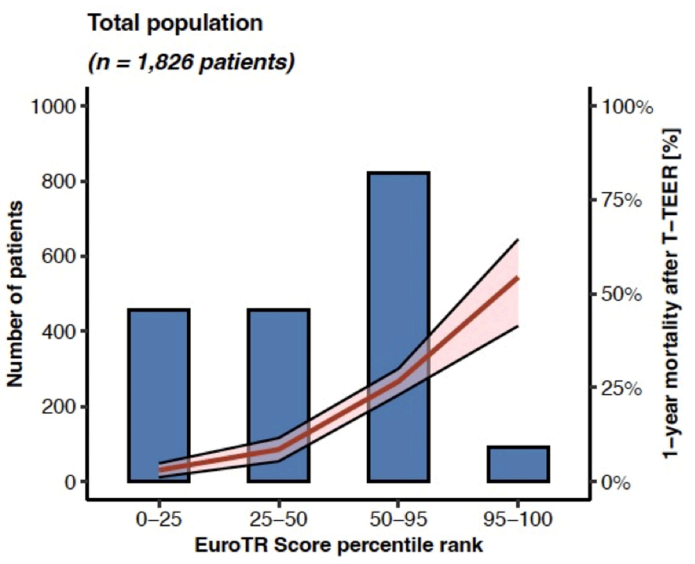
intermediate-risk patients, and 45.6% and 32.0% in high-risk patients.

As depicted in the **Central Illustration**, the EuroTR score not only predicts 1-year mortality after T-TEER but additionally stratifies patients by their likelihood of poor clinical outcomes, defined as mortality, heart failure hospitalization, or persistent dyspnea expressed as NYHA functional class ≥III at 1-year follow-up. Poor clinical outcome correlated with increasing EuroTR scores. The likelihood of poor outcomes increased from 30.6% in patients with the lowest EuroTR scores (EuroTR risk rank <5%) to 85.5% in the highest risk group (EuroTR risk rank ≥95%).

PERFORMANCE OF THE EuroTR RISK SCORE IN SUBGROUPS. The performance of the EuroTR risk score was further evaluated in several subgroups, including secondary vs primary or mixed TR etiology, atrial vs nonatrial secondary TR, absence vs presence of transtricuspid RV leads, lower vs higher TAPSE, lower vs higher pulmonary artery pressure, and lower vs higher pulmonary vascular resistance. The EuroTR score performed comparably well across all subgroups, with significantly elevated HRs for high-risk patients in each subgroup. Detailed results are presented in Supplemental Figures 6 to 8. Furthermore, subgroup analyses demonstrated robust discrimination across RV lead status and TR etiologies, with particularly high Harrell's C index for atrial TR; Harrell's C index was 0.732 (95% CI: 0.684-0.780) in patients without and 0.759 (95% CI: 0.716-0.802) in those with any RV leads. Among patients with secondary TR, atrial TR showed higher discrimination (0.810; 95% CI: 0.753-0.868) compared with nonatrial TR (0.730; 95% CI: 0.691-0.768). By etiology, the C index was 0.793 (95% CI: 0.730-0.856) in primary and mixed TR and 0.763 (95% CI: 0.735-0.791) in secondary TR.

IMPACT OF TRILUMINATE ELIGIBILITY ON 1-YEAR SURVIVAL PREDICTION. Among patients from the EuroTR registry, the 7 main TRILUMINATE eligibility criteria were available for 737 patients. On the basis of these eligibility criteria, the entire EuroTR cohort was divided into TRILUMINATE-eligible (41.7%) and TRILUMINATE-noneligible (58.3%) patients (**Figure 7**). TRILUMINATE-noneligible patients displayed significantly lower 1-year survival of 73.5% (95% CI: 69.1%-78.2%) compared with TRILUMINATE-eligible patients (1-year survival 85.6%; [95% CI: 81.3%-90.2%]; HR for 1-year mortality: 2.03 [95% CI: 1.38-2.98]; $P < 0.001$). Importantly, stratification by the EuroTR score proved effective in both TRILUMINATE-eligible and TRILUMINATE-noneligible patients (**Figure 7**).

FIGURE 4 1-Year Mortality Risk According to the EuroTR Risk Score



The relationship of EuroTR (European Registry of Transcatheter Repair for Tricuspid Regurgitation) risk score values and survival probability. T-TEER = tricuspid valve transcatheter edge-to-edge repair.

ONLINE RISK CALCULATOR. To facilitate the use of the EuroTR score in future clinical or research settings, an online risk calculator has been developed and is freely accessible at www.EuroTR.eu.

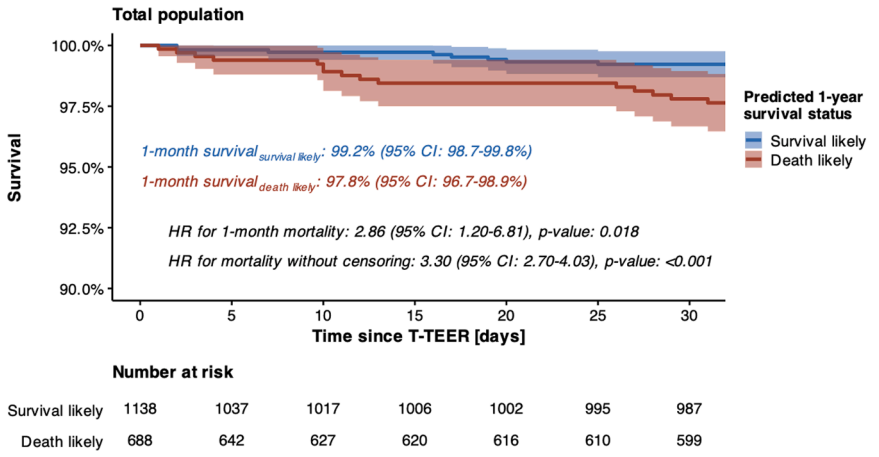
DISCUSSION

The EuroTR score, which was developed from the largest available, international, multicenter EuroTR registry, is an artificial intelligence (AI)-driven comprehensive risk model for predicting mortality and futility in patients with severe symptomatic TR undergoing T-TEER. The main findings of this study can be summarized as follows.

First, the EuroTR score, which was validated in a large separate contemporary patient cohort, allows significantly improved 1-year mortality prediction compared with multiple established risk scores by integrating clinical, laboratory, echocardiographic, and hemodynamic data.

Second, the EuroTR score identifies patients at higher mortality risk and estimates the likelihood of T-TEER futility, aiding heart team decision-making and informing patients of procedural benefits,

FIGURE 5 Early Outcome Prediction Using the EuroTR Risk Score



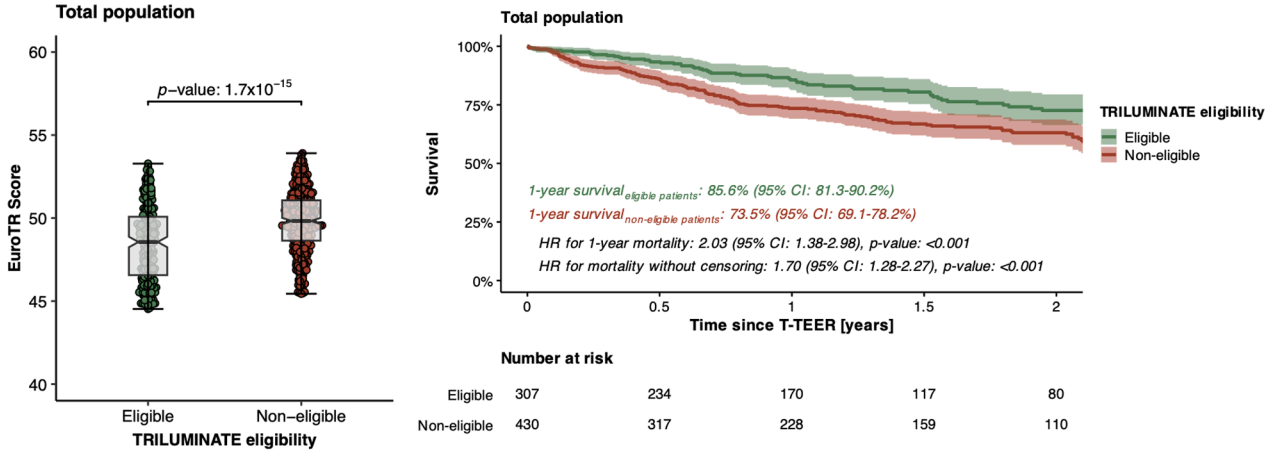
Despite being developed for 1-year outcome prediction, the EuroTR score also enabled the identification of patients at increased risk for early mortality. Abbreviations as in Figure 4.

ultimately enabling a more informed and personalized approach to medical care.

Third, the EuroTR score enhances characterization of TR patient cohorts undergoing T-TEER, which may help better understand outcomes from contemporary clinical trials.

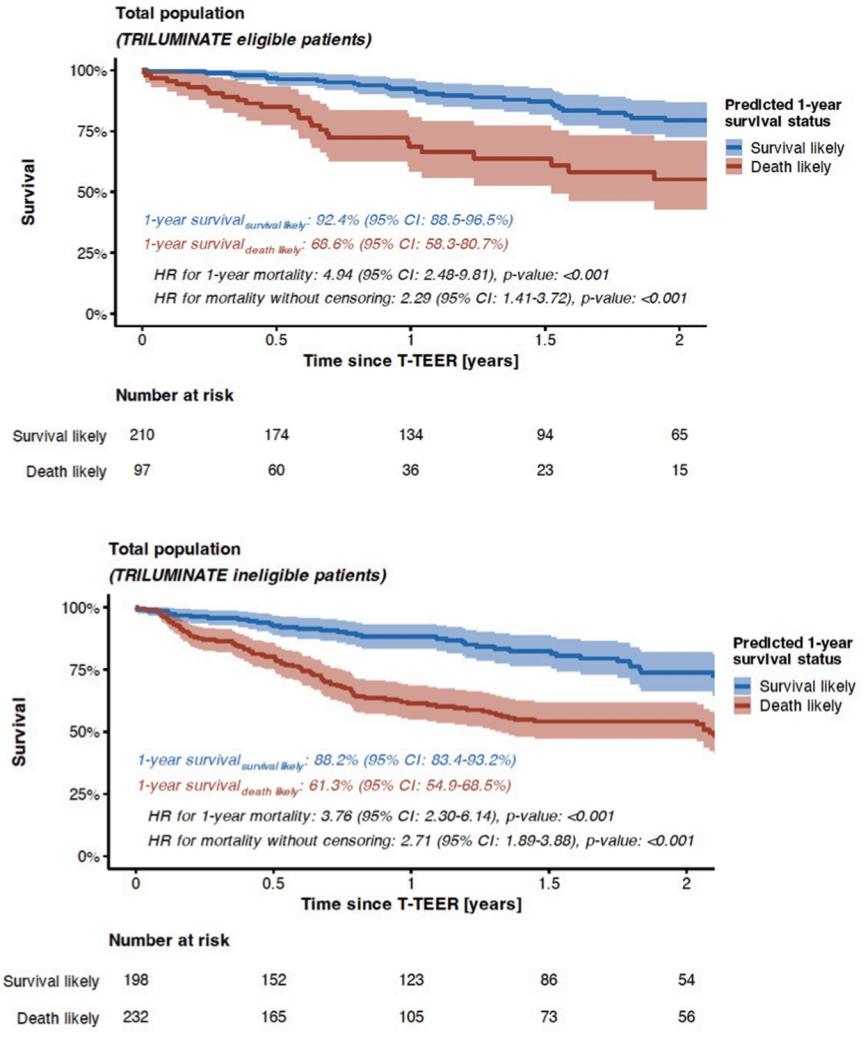
As transcatheter tricuspid valve procedures are rapidly emerging in response to an unmet clinical need, critical questions about optimal patient selection and timing of interventions arise. Traditional risk scores are often applied in clinical practice to guide clinical decision-making. However, these scores, including the EuroSCORE II, TRI-SCORE, and others, were designed primarily for short-term mortality prediction of cardiac and tricuspid valve surgery, and different models, such as the TRIO score and the score of Wang et al,[7] were developed for long-term mortality prediction, without accounting for interventional repair strategies. Thus, these scores suffer from low accuracy for outcome

FIGURE 6 The EuroTR Risk Score in TRILUMINATE-Eligible and TRILUMINATE-Ineligible Patients



TRILUMINATE-noneligible patients showed significantly higher EuroTR score ranks than TRILUMINATE-eligible patients. Abbreviations as in Figure 4.

FIGURE 7 Predictive Value of the EuroTR Risk Score Stratified by TRILUMINATE Eligibility



TRILUMINATE-noneligible patients displayed a significantly lower 1-year survival compared with TRILUMINATE-eligible patients. Abbreviations as in Figure 4.

prediction of T-TEER procedures, which are currently the most often applied valve repair technique for isolated TR. The EuroTR score addresses this gap by incorporating a broad range of clinical, echocardiographic, laboratory, and hemodynamic parameters and leveraging machine learning to capture complex predictor variable interactions. Validated in an independent large multicenter cohort, the EuroTR score significantly outperformed all currently available risk models for 1-year survival prediction after T-TEER, indicating a major advancement in patient risk stratification. Importantly, calibration analysis indicated that the EuroTR score provided reliable mortality predictions in the clinically most relevant low- to intermediate-risk range. At higher predicted risk levels, the model tended to overestimate mortality. From a clinical perspective, this conservative calibration seems justifiable, as high-risk patients are less likely to be underestimated and may therefore be more reliably identified for intensified follow-up and management.

Identifying patients who would benefit most from tricuspid interventions is essential for optimizing patient selection, particularly in high-risk groups in which T-TEER may offer limited benefit. In this regard, the EuroTR risk score provides valuable

information for heart teams and patients alike, using a clinically relevant combined endpoint of mortality, heart failure hospitalization, and persistence of NYHA functional class ≥III at 1-year follow-up. The almost linear relationship between EuroTR score deciles and the combined clinical endpoint indicates that the AI-derived risk assessment offers meaningful risk estimates across a wide spectrum of patient profiles. This is of particular importance, as available data from the TRILUMINATE and Tri.FR trials have demonstrated that the main benefit of T-TEER might be observed in the reduction of heart failure symptoms and annular heart failure hospitalizations.[26] Accordingly, the combined prediction of symptomatic alleviation and survival after a tricuspid procedure is of utmost value for the consideration of potential treatment futility, a difficult task that is currently not adequately addressed by the aforementioned risk scores. Thus, the EuroTR risk score will facilitate better communication among cardiologists, referring physicians, and patients, and this improved transparency will support shared decision-making, ensuring that patients are fully informed about their treatment options and expectations.

Especially within a high-risk cohort, for which no randomized controlled data are currently available, an open discussion with the patient is important to discuss the individually expected benefit of the procedure and to weigh whether this therapeutic goal aligns with the patient's values and preferences. Specifically for this decision and discussion, the EuroTR score could make an important contribution.

The analyses indicate that the EuroTR risk score also provides robust risk estimates for dedicated patient subgroups, including patients with atrial secondary TR phenotypes, transtricuspid cardiac implantable electronic device leads, elevated pulmonary artery pressures, and reduced RV function indexes. Interestingly, anatomical parameters of the tricuspid valve (coaptation gap, tenting height and area) did not significantly contribute to the final prediction model.

The ability to improve characterization of patient cohorts may also be an important additional implication of the EuroTR score. For example, the lack of superior outcomes in terms of mortality after T-TEER in the randomized TRILUMINATE trial have been attributed to many potential factors, including patient selection. Indeed, the application of the EuroTR score indicates that patients from the EuroTR registry, who would have been TRILUMINATE eligible, carry a significant lower risk for 1-year mortality than noneligible patients. This reduced risk translated into a very favorable 1-year survival rate of 85.6% in TRILUMINATE-eligible patients compared with a survival rate of 73.5% in noneligible patients. This 12% absolute difference in survival is substantial considering that the overall 1-year mortality rate of the entire EuroTR study group was only 17.9%. Although it remains speculative whether future randomized clinical trials for transcatheter tricuspid interventions will show differences in mortality, this will be influenced by patient selection and whether high-, intermediate-, or low-risk patients will be included in such trials. Although including low-risk patients might lead to endpoint rates that are too low to demonstrate a significant treatment effect within a reasonable time frame, including high-risk patients could dilute a potential treatment effect because of a too advanced disease state and comorbidities.

Importantly, the EuroTR risk calculator (www.EuroTR.eu) has the ability to handle missing variables; however, the accuracy and reliability of the risk assessment are naturally enhanced with more complete information. The availability of an online EuroTR risk calculator with a user-friendly interface will facilitate the widespread use in daily clinical practice for an individual risk assessment.

STUDY LIMITATIONS. Traditional scores such as EuroSCORE II and TRI-SCORE, although definitely of high value, fall short in specific T-TEER populations because of their generalized nature. Accuracy of surgical risk scores has been reported to be suboptimal in interventionally treated patients.[27,28] The EuroTR score, designed specifically for TR patients undergoing T-TEER, addresses these limitations by incorporating a broader range of relevant variables and leveraging machine learning to uncover complex interactions among predictors. Data on the newly developed Society of Thoracic Surgeons tricuspid score were not available within the EuroTR study population.

The AI-driven model's ability to integrate and analyze a wide range of clinical, laboratory, echocardiographic, and hemodynamic parameters signifies a considerable improvement over traditional risk stratification tools. It may help address 2 important issues: 1) to understand who may benefit from percutaneous therapies, even if procedural risk is low (patient selection); and 2) to assist the heart team in determining the optimal timing for intervention. In fact, for those patients indicated to a "watch and wait" strategy, the 5 main parameters (hemoglobin, NT-proBNP, TAPSE, the presence of pleural effusion, and TR vena contracta width) included in the present risk score might help

understand when the clinical conditions are worsening to avoid "too late" interventions. Furthermore, the EuroTR score does not directly account for adverse events.

Beyond that, the model was derived and validated in patients who were selected for T-TEER treatment. Therefore a certain degree of selection bias cannot be excluded.

Data on the exact cause of death (cardiovascular vs noncardiovascular) are not available because of the retrospective nature of this registry. However, all-cause mortality is a robust and objective endpoint routinely used in clinical trials.

Given the retrospective design and site-reported data collection of this registry, echocardiographic assessments were not supervised by a core laboratory. Nevertheless, the EuroTR score demonstrated solid performance in an independent validation cohort, which supports the reliability of echocardiographic measurements across different centers.

CONCLUSIONS

The EuroTR score is the first AI-driven risk score for predicting 1-year mortality and symptomatic outcomes in T-TEER patients. The score was derived from a large real-world registry and externally validated using data from 9 independent study centers. With an easy-to-use online risk calculator available (www.EuroTR.eu), the score can support patient selection for future studies and trials, as well as the shared decision-making process on the basis of prognosis and the utility of T-TEER on an individual patient level.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Among the full cohort of patients in the registry, data collection for the Hamburg patients was supported by a grant from the German Heart Foundation. Dr Stolz has received speaker honoraria from Edwards Lifesciences. Dr Kresoja has received travel expenses from Edwards Lifesciences. Dr von Stein has received lecture honoraria from Edwards Lifesciences. Dr Rottbauer has received speaker honoraria from Edwards Lifesciences and Abbott Laboratories. Dr Denti has served as a consultant for InnovHeart, Pi-Cardia, HVR, and Approxima; and has received speaker honoraria from Abbott Laboratories and Edwards Lifesciences. Dr Rassaf received speaker honoraria and consulting fees from AstraZeneca, Bayer, Pfizer, and Daiichi-Sankyo. Dr Barreiro-Perez has received speaker fees from Abbott Cardiovascular, Edwards Lifesciences, and Venus Medtech. Dr Adamo has received consulting fees in the past 3 years from Abbott Structural Heart and Edwards Lifesciences. Dr Toggweiler has received personal honoraria from Medtronic, Boston Scientific, Biosensors, Abbott Cardiovascular, Medira, Shockwave Medical, Teleflex, atHeart Medical, Cardiac Dimensions, Polares Medical, Amarin, Sanofi, AstraZeneca, ReCor Medical, and Daiichi-Sankyo; has received institutional research grants from Edwards Lifesciences, Boston Scientific, Fumedica, Novartis, and Boehringer Ingelheim; and holds equity in Hi-D Imaging. Dr Metra has received consulting fees in the past 3 years from Abbott Structural Heart, AstraZeneca, Bayer, Boehringer Ingelheim, Edwards Lifesciences, and Roche Diagnostics. Dr Geisler has received speaker honoraria and research grants from AstraZeneca, Bayer, Bristol Myers Squibb/Pfizer, Ferrer/Chiesi, Medtronic, and Edwards Lifesciences (all unrelated to this study). Dr Estévez-Loureiro has received speaker fees from Abbott Cardiovascular, Edwards Lifesciences, Boston Scientific, and Venus Medtech. Dr Lüdike has received speaker honoraria and consulting fees from AstraZeneca, Bayer, Pfizer, and Edwards Lifesciences; and has received research honoraria from Edwards Lifesciences. Dr Maisano has received grant and/or research institutional support from Abbott Laboratories, Medtronic, Edwards Lifesciences, Biotronik, Boston Scientific, NVT, Terumo, and Venus Medtech; has received consulting fees and personal and institutional honoraria from Abbott Laboratories, Medtronic, Edwards Lifesciences, Xeltis, Cardiovalve, Occlufit, Simulands, Mtex, Venus Medtech, Squadra, and Valgen; has received royalty income from and has intellectual property rights with Edwards Lifesciences; and is a shareholder (including share options) in Magenta, Transseptal Solutions, and 4Tech. Dr Praz has received travel expenses from Edwards Lifesciences, Abbott Cardiovascular, Polares Medical, Medira, and Siemens Healthineers. Dr Kessler has received speaker honoraria from Edwards Lifesciences and Abbott Laboratories. Dr Kalbacher has received personal fees from Abbott Laboratories, Edwards Lifesciences, Pi-Cardia, and Medtronic. Dr Rudolph has received research grants from Abbott Laboratories, Boston Scientific, and Edwards Lifesciences. Dr Iliadis has received consulting fees and travel expenses from Abbott Laboratories and Edwards Lifesciences. Dr Sticchi has served on the advisory board for Edwards Lifesciences. Dr Lurz has received institutional grants from Edwards Lifesciences; and has received honoraria from Innoventric. Dr Hausleiter has received research grant support and speaker honoraria from Edwards Lifesciences. Dr Tarantini has received speaker fees from Abbott Cardiovascular and Edwards Lifesciences. Dr Mahabadi has received speaker fees from and/or is an advisory board member for Amgen, Berlin Chemie, Daiichi-Sankyo, Edwards Lifesciences, Novartis, and Sanofi; and has received research funding from Daiichi-Sankyo and Edwards Lifesciences (all outside the submitted work). Dr Lachmann has received funding from the Technical University of Munich (Clinician Scientist Grant), the Else Kröner-Fresenius Foundation (Clinician Scientist Grant), the German Center for Cardiovascular Research (Postdoc Start-Up Grant on Advancing Digital Aspects), and the German Heart Foundation ("Machine Learning in Severe Mitral Regurgitation"). All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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PERSPECTIVES

WHAT IS KNOWN? Existing risk assessment models for patients undergoing T-TEER are subject to several limitations and might not fully cover the full complexity of patients with severe TR presenting for treatment evaluation.

WHAT IS NEW? The EuroTR risk score, which was derived and independently validated in a large real-world cohort of T-TEER patients, offers the opportunity to predict 1 year mortality and poor clinical outcomes.

WHAT IS NEXT? With an online risk calculator available, the score might support patient selection for future studies and trials, as well as the shared decision-making process within the heart team and with the patient.

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KEY WORDS artificial intelligence, edge-to-edge repair, machine learning, mortality prediction

APPENDIX For supplemental tables and figures, please see the online version of this paper.