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Development and internal validation of a nomogram for predicting 90- day mortality in intensive care unit patients with pressure injury: a retrospective cohort study based on the MIMIC-IV database

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Abstract

Background

Pressure injury (PI) is a frequent and serious complication in critically ill patients and is independently associated with increased mortality; however, dedicated tools for individualized short-term prognosis prediction in intensive care unit (ICU) patients who already have PI remain scarce.

Methods

We retrospectively enrolled 2880 adult patients with PI on their first ICU admission from the MIMIC-IV database and randomly assigned them to a training cohort (n = 2016, 70%) and a validation cohort (n = 864, 30%). Least absolute shrinkage and selection operator (LASSO) Cox regression followed by backward elimination was used for variable selection, and a multivariable Cox model was constructed and visualized as a nomogram. Discrimination was assessed by the Harrell concordance index (C-index) and the area under the receiver operating characteristic curve (AUC); calibration and clinical utility were evaluated by calibration plots and decision curve analysis (DCA). Internal validation used 1000 bootstrap resamples.

Results

Overall 90-day mortality was 41.3%. Six independent predictors were retained: age, body mass index (BMI), Acute Physiology and Chronic Health Evaluation (APACHE) III score, Charlson comorbidity index, red cell distribution width (RDW), and arterial partial pressure of oxygen (PaO2). The nomogram achieved a C-index of 0.699 in the training cohort and 0.690 in the validation cohort (AUC 0.738 and 0.731, respectively). After 1000 bootstrap resamples, the optimism-corrected C-index was 0.694 (95% CI 0.684–0.711), and the calibration slope and calibration-in-the-large (intercept) were 0.959 and 0.001, respectively, indicating excellent calibration. Decision curve analysis demonstrated net clinical benefit across a wide range of threshold probabilities. Compared with the generic APACHE III, SOFA, and SAPS II scores used alone, the nomogram showed superior discrimination (category-free NRI 0.588 and IDI 0.121 vs APACHE III alone in the training cohort). The associations of all six predictors remained stable at 28-, 90-, and 365-day endpoints (all $P < 0.001$).

Conclusions

We developed and internally validated a six-variable nomogram that reliably predicts 90-day mortality in ICU patients with PI, with good discrimination, calibration, and clinical utility, which may support individualized risk stratification and early intervention in this high-risk population. An accompanying free online calculator is provided to facilitate bedside use.

Introduction

Pressure injury, formerly termed pressure ulcer, is localized damage to the skin and/or underlying soft tissue, usually over a bony prominence, resulting from intense or prolonged pressure or pressure in combination with shear [1]. As one of the most common hospital-acquired complications, PI represents a substantial and growing clinical and economic burden: a recent United States analysis of the National Inpatient Sample reported rising prevalence and an inpatient cost that increased from approximately $69,499 to $102,939 per admission between 2009 and 2019 [2]. The international DecubICUs study, which surveyed 13,254 patients in 1,117 ICUs across 90 countries, found an overall PI prevalence of 26.6% and an ICU-acquired PI prevalence of 16.2%, underscoring the particular vulnerability of critically ill patients [3].

Beyond their local consequences, pressure injuries are strong markers and drivers of poor outcome. A systematic review and meta-analysis reported that older patients with PI have an approximately twofold higher risk of death during follow-up compared with those without PI (pooled hazard ratio 1.78, 95% CI 1.46–2.16), with higher stages conferring even greater risk [4]. In ICU populations specifically, PI severity has been shown to mediate the relationship between illness severity and mortality [5], and the graded association between PI stage and ICU mortality is well documented [3]. Critically ill patients with PI are therefore a high-risk group in whom accurate risk estimation is clinically valuable.

Most existing research on PI has, however, concentrated on its incidence, staging, prevention, and treatment [6], rather than on the prognosis of patients who already have it. Although a few recent studies based on the MIMIC-IV database have explored machine-learning models or single nutritional markers for mortality in ICU patients with PI [7, 8], a simple, readily interpretable prognostic tool—such as a nomogram built from routinely available variables—is still lacking for this population.

Accordingly, in the present study we used the MIMIC-IV database to develop and internally validate a nomogram for predicting 90-day mortality in ICU patients with pressure injury, integrating variables selected by LASSO Cox regression, and to evaluate its discrimination, calibration, and clinical utility.

Methods

Study design and data source

This retrospective cohort study was performed using the Medical Information Mart for Intensive Care (MIMIC)-IV database (version 2.2, the version available at the time of data extraction), a large, publicly available, single-center database comprising de-identified data from patients admitted to the intensive care units of the Beth Israel Deaconess Medical Center in Boston, Massachusetts [9], the successor to the earlier MIMIC-III database [10]. Because the database is fully de-identified and publicly available, the requirement for informed consent and institutional review board approval was waived. The study was conducted and reported in accordance with the Transparent Reporting of a Multivariable Prediction

Model for Individual Prognosis or Diagnosis (TRIPOD) statement [11] and its updated TRIPOD + AI guidance [12].

Study population

In MIMIC-IV v2.2, there were 73,181 adult (aged ≥ 18 years) ICU admissions, of which 50,920 were first ICU admissions. Among these, 2880 patients had a recorded diagnosis of pressure injury—defined by International Classification of Diseases codes ICD-9 707.0x or ICD-10 L89.x—on their first ICU admission and were enrolled (Fig. 1). Time zero was defined as ICU admission (the index time), at which point all candidate predictors were extracted; the severity scores (APACHE III, SOFA, SAPS II, OASIS) were computed from the first 24 hours of ICU admission by their original definitions, and the 90-day outcome was anchored at ICU admission. No patient was excluded solely because of missing data; instead, missing covariate values were handled by median imputation and variables with ≥ 40% missingness were excluded a priori, as described below.

Candidate variables and outcome

Candidate predictors were selected a priori based on clinical relevance and previous literature, and included demographic characteristics (age, sex, BMI), severity-of-illness scores (SOFA [13], APACHE III [14], SAPS II [15], OASIS [16], and Glasgow Coma Scale [17]), the Charlson comorbidity index [18], vital signs, and laboratory parameters (complete blood count, coagulation, biochemistry, arterial blood gas, and lactate). The primary outcome was 90-day all-cause mortality. To test robustness, 28-day and 365-day all-cause mortality were evaluated as sensitivity endpoints.

Statistical analysis

Continuous variables were summarized as median (interquartile range) and compared using the Mann–Whitney U test; categorical variables were summarized as counts (percentages) and compared using the χ^2 or Fisher exact test. Continuous variables were winsorized at the 0.5th and 99.5th percentiles derived from the training cohort to limit the influence of extreme values while avoiding information leakage from the validation cohort; a sensitivity analysis using thresholds derived from the full dataset yielded identical results. Missing values among candidate predictors were imputed using the training-cohort median; variables with ≥ 40% missingness (e.g., serum albumin 57.4% and C-reactive protein 94.8%) were excluded a priori to avoid unstable imputation, and the missingness rates of all candidate predictors are summarized in Supplementary Table S1. Patients were randomly divided into a training cohort (70%) and a validation cohort (30%) stratified by outcome. To avoid overfitting, LASSO Cox regression with 5-fold cross-validation was applied to select predictors from the candidate set [19]; the one-standard-error rule selected a penalty λ_1 of 0.06 (the selected value on a pre-specified penalty grid from 0.005 to 0.20; retaining seven predictors), after which backward elimination (retention criterion $P < 0.10$) removed the SAPS II score owing to collinearity with APACHE III, leaving six predictors. A

multivariable Cox proportional hazards model was then fitted to estimate hazard ratios (HRs) with 95% confidence intervals (CIs) [20], and a nomogram was constructed. Model discrimination was quantified by the Harrell C-index [21] and the 90-day time-dependent (cumulative/dynamic) AUC; the median follow-up time and the censoring proportion were also reported. To evaluate incremental value over generic severity scores, the nomogram was compared with the APACHE III, SOFA, and SAPS II scores used alone in terms of C-index and time-dependent AUC, and the integrated discrimination improvement (IDI), the category-free net reclassification improvement (NRI), and the category-based NRI at 10% and 20% risk thresholds were computed relative to APACHE III alone [22, 23, 24, 25]. Calibration was assessed graphically and quantified by the calibration slope and the calibration-in-the-large (intercept), and clinical utility was evaluated by decision curve analysis [26]. Internal validation was performed using 1000 bootstrap resamples, from which optimism-corrected performance and 95% percentile CIs were derived [22, 23]. All analyses were performed in Python (version 3.12) using lifelines, scikit-learn, and pandas. A two-sided $P < 0.05$ was considered statistically significant.

Model assumptions were examined as follows. The proportional hazards (PH) assumption was tested with Schoenfeld residuals for each predictor. Because the primary outcome was a fixed 90-day window with complete follow-up (no loss to follow-up), a multivariable logistic regression with the same six predictors was additionally fitted to confirm the robustness of the associations, and a time-varying coefficient model (APACHE III × ln(time)) was used to characterize the non-proportionality of the APACHE III effect. Potential non-linear associations between the continuous predictors and the log hazard were evaluated using restricted cubic splines with three knots at the 10th, 50th, and 90th percentiles and likelihood-ratio tests for non-linearity. Multicollinearity was assessed with the variance inflation factor (VIF) and pairwise Pearson correlations. Baseline balance between the training and validation cohorts was summarized with standardized mean differences (SMDs), and a missing-indicator analysis tested whether missingness itself was associated with mortality.

Results

Patient characteristics

A total of 2880 patients were included (median age 72 years; 56.5% male), with a 90-day mortality of 41.3% (n = 1189). Compared with survivors, non-survivors were significantly older (median 77 vs 69 years), had higher severity scores (APACHE III 59 vs 49; SOFA 6 vs 5; SAPS II 46 vs 39), a higher Charlson comorbidity index (7 vs 6), higher RDW (16.4% vs 15.4%), and lower BMI (25.2 vs 27.0 kg/m²) and PaO2 (75 vs 90 mmHg) (all $P < 0.001$). The prevalence of congestive heart failure, atrial fibrillation, renal disease, malignant cancer, and the need for renal replacement therapy were all higher among non-survivors. Baseline characteristics stratified by 90-day survival are summarized in Table 1. All non-decedents were followed for the full 90 days with no loss to follow-up (censoring was administrative at 90 days); the 90-day censoring proportion was 58.7%, and among the 1189 decedents the median time to death was 21 days (IQR 9.0–42.0). The cohort was predominantly older (median age 72 years) with

high illness severity (median APACHE III 52), consistent with a population enriched for advanced or complicated pressure injuries, although PI stage was not recorded in the extract.

Table 1
Baseline characteristics of the study population stratified by 90-day survival.

Variable	Survivors (n = 1691)	Non-survivors (n = 1189)	P
Age, years	69.0 [59.5–79.0]	77.0 [67.0–85.0]	< 0.001
BMI, kg/m²	27.0 [22.9–32.8]	25.2 [21.2–29.5]	< 0.001
SOFA score	5.0 [3.0–7.0]	6.0 [4.0–9.0]	< 0.001
APACHE III score	49.0 [38.0–62.0]	59.0 [46.0–74.0]	< 0.001
SAPS II score	39.0 [31.0–47.0]	46.0 [38.0–56.0]	< 0.001
OASIS score	33.0 [28.0–39.0]	36.0 [30.0–43.0]	< 0.001
Glasgow Coma Scale	15.0 [13.0–15.0]	14.0 [11.0–15.0]	< 0.001
Charlson comorbidity index	6.0 [4.0–8.0]	7.0 [5.0–9.0]	< 0.001
Heart rate, beats/min	90.0 [77.0–104.0]	94.0 [79.0–108.0]	< 0.001
Mean arterial pressure, mmHg	80.0 [69.0–91.0]	77.0 [66.0–89.0]	< 0.001
Respiratory rate, breaths/min	19.0 [15.0–23.0]	20.0 [16.0–25.0]	< 0.001
Temperature, °C	36.7 [36.4–37.1]	36.6 [36.2–37.0]	< 0.001
SpO_2, %	98.0 [95.0–100.0]	97.0 [95.0–100.0]	0.001
WBC, ×10⁹/L	11.5 [7.9–15.9]	12.1 [8.2–17.8]	0.001
Hemoglobin, g/dL	9.7 [8.5–11.4]	9.4 [8.1–10.8]	< 0.001
Platelet, ×10⁹/L	217.0 [151.2–296.0]	206.0 [133.0–297.0]	0.009
RDW, %	15.4 [14.1–17.0]	16.4 [14.9–18.3]	< 0.001
Creatinine, mg/dL	1.1 [0.7–1.9]	1.4 [0.8–2.5]	< 0.001
BUN, mg/dL	26.0 [16.0–45.0]	35.0 [21.0–59.0]	< 0.001
Glucose, mg/dL	127.0 [102.2–167.0]	128.0 [102.0–174.0]	0.409
Lactate, mmol/L	1.6 [1.2–2.6]	1.9 [1.3–3.1]	< 0.001
INR	1.3 [1.2–1.6]	1.4 [1.2–1.9]	< 0.001
pH	7.4 [7.3–7.4]	7.4 [7.3–7.4]	0.190
$PaCO_2$, mmHg	42.0 [36.0–50.0]	41.0 [34.0–49.0]	0.034
PaO_2, mmHg	90.0 [51.0–171.0]	75.0 [45.0–130.0]	< 0.001
Base excess, mmol/L	0.0 [-4.0–2.0]	-1.0 [-6.0–2.0]	0.006
ICU length of stay, days	3.0 [1.6–7.7]	3.2 [1.6–7.9]	0.915

Variable	Survivors (n = 1691)	Non-survivors (n = 1189)	P
Hospital length of stay, days	14.9 [7.9–28.8]	12.7 [6.8–22.5]	< 0.001
Male, n (%)	962 (56.9)	666 (56.0)	0.668
Hypertension, n (%)	1143 (67.6)	798 (67.1)	0.819
Diabetes, n (%)	719 (42.5)	502 (42.2)	0.903
Congestive heart failure, n (%)	643 (38.0)	548 (46.1)	< 0.001
Atrial fibrillation, n (%)	611 (36.1)	528 (44.4)	< 0.001
Renal disease, n (%)	537 (31.8)	481 (40.5)	< 0.001
Chronic pulmonary disease, n (%)	467 (27.6)	322 (27.1)	0.784
Malignant cancer, n (%)	198 (11.7)	244 (20.5)	< 0.001
Cerebrovascular disease, n (%)	294 (17.4)	230 (19.3)	0.196
Peripheral vascular disease, n (%)	255 (15.1)	220 (18.5)	0.017
Mechanical ventilation, n (%)	750 (44.4)	467 (39.3)	0.007
Renal replacement therapy, n (%)	46 (2.7)	57 (4.8)	0.004

Variable selection and construction of the nomogram

LASSO Cox regression initially selected seven predictors; after backward elimination for collinearity, six independent predictors remained in the final model: age, BMI, APACHE III score, Charlson comorbidity index, RDW, and PaO2 (Fig. 2). Higher age (HR 1.015, 95% CI 1.009–1.021), APACHE III score (HR 1.019, 95% CI 1.016–1.022), Charlson comorbidity index (HR 1.102, 95% CI 1.072–1.133), and RDW (HR 1.120, 95% CI 1.091–1.149) were independently associated with increased 90-day mortality, whereas higher BMI (HR 0.972, 95% CI 0.962–0.982) and PaO2 (HR 0.998, 95% CI 0.997–0.999) were protective (all P < 0.001; Table 2, Fig. 3). A nomogram incorporating these six variables was constructed to estimate 90-day mortality (Fig. 4).

Table 2
Multivariable Cox regression for 90-day mortality.

Variable	HR (95% CI)	P
Age	1.015 (1.009–1.021)	< 0.001
BMI	0.972 (0.962–0.982)	< 0.001
APACHE III	1.019 (1.016–1.022)	< 0.001
Charlson comorbidity index	1.102 (1.072–1.133)	< 0.001
RDW	1.120 (1.091–1.149)	< 0.001
PaO_2	0.998 (0.997–0.999)	< 0.001

HRs are expressed per 1-year increase in age, per 1 kg/m² increase in BMI, per 1-point increase in the APACHE III score, per 1-point increase in the Charlson comorbidity index, per 1% increase in RDW, and per 1 mmHg increase in PaO_2.

Performance and validation of the nomogram

The nomogram demonstrated good discrimination, with a C-index of 0.699 in the training cohort and 0.690 in the validation cohort, and a 90-day AUC of 0.738 and 0.731, respectively (Fig. 5). After 1000 bootstrap resamples on the full cohort, the apparent C-index was 0.696 and the optimism-corrected C-index was 0.694 (optimism 0.002; 95% percentile CI 0.684–0.711), and the apparent 90-day AUC was 0.733 with an optimism-corrected AUC of 0.731 (optimism 0.002; 95% CI 0.707–0.750), indicating minimal overfitting. Calibration was excellent: the apparent calibration slope was 0.973 and the bootstrap-corrected slope 0.959 (ideal value 1), with a calibration-in-the-large (intercept) of 0.001 (ideal value 0); calibration curves showed good agreement between predicted and observed 90-day mortality in both cohorts (Fig. 6). Decision curve analysis demonstrated net clinical benefit across threshold probabilities of 1% to 50% (Fig. 7).

Model assumptions and diagnostics

The proportional hazards assumption was satisfied for all predictors except the APACHE III score (Schoenfeld test $P < 0.001$; all others $P \geq 0.36$). A time-varying coefficient model (APACHE III × ln(time)) confirmed that the APACHE III effect was strongest in the first days of the ICU stay and attenuated over follow-up (interaction $\beta = -0.041$, $P < 0.001$). Because the 90-day endpoint was a fixed window with complete follow-up, a 90-day logistic regression with the same six predictors was additionally fitted; it gave discrimination essentially identical to the Cox model (AUC 0.738/0.729 vs 0.738/0.731) and odds ratios concordant in direction and magnitude with the hazard ratios (e.g., APACHE III OR 1.023 vs HR 1.019), indicating that the non-proportionality did not materially affect the 90-day risk estimates used by the nomogram. Restricted cubic splines showed no evidence of non-linearity for any continuous

predictor (likelihood-ratio tests, all P > 0.05; BMI P = 0.077), and adding a restricted cubic spline term for BMI did not change discrimination (C-index 0.699 vs 0.699), supporting linear modelling. Multicollinearity was negligible (all VIF ≤ 1.36); PaO2 was essentially uncorrelated with APACHE III (Pearson r = -0.03 and Spearman ρ = -0.03 on the n = 1969 pairwise-complete observations; r = 0.02 after median imputation; Supplementary Figure S1), reflecting that raw PaO2 does not account for the inspired oxygen fraction and that the APACHE III oxygenation axis uses the PaO2/FiO2 ratio or alveolar–arterial gradient rather than raw PaO2, so PaO2 contributed prognostic information independent of the severity score. The standardized mean differences of the six predictors between the training and validation cohorts were all ≤ 0.09, and 90-day mortality was identical in the two cohorts (41.3% each), indicating satisfactory balance after stratified randomization.

Incremental value over generic severity scores

When applied alone, the generic severity scores showed substantially lower discrimination than the nomogram: in the training cohort, the C-index was 0.629, 0.602, and 0.632 for APACHE III, SOFA, and SAPS II, respectively, versus 0.699 for the nomogram (Table 3). Relative to APACHE III alone, the nomogram improved the category-free NRI by 0.588 and the IDI by 0.121 in the training cohort, and by 0.540 and 0.102, respectively, in the validation cohort, indicating meaningful incremental discrimination. At fixed risk thresholds, the category-based NRI was 0.039 and 0.179 at 10% and 20% in the training cohort, and 0.052 and 0.203 in the validation cohort, respectively, driven predominantly by correct downward reclassification of non-events.

Table 3
Comparison of discrimination between the nomogram and generic severity scores used alone.

Model	C-index (training)	C-index (validation)	90-day AUC (training)	90-day AUC (validation)
APACHE III score	0.629	0.630	0.642	0.652
SOFA score	0.602	0.593	0.610	0.607
SAPS II score	0.632	0.644	0.648	0.672
Nomogram (6 variables)	0.699	0.690	0.738	0.731

Sensitivity analysis

When the endpoint was redefined as 28-day and 365-day mortality, all six predictors remained independently associated with mortality (all P < 0.001) with stable hazard ratios (Table 4), confirming the robustness of the model across different follow-up windows (C-index 0.723/0.706 at 28 days and 0.683/0.685 at 365 days, training/validation). In a missing-indicator analysis, missingness of serum albumin (HR 0.79, P < 0.001) and lactate dehydrogenase (HR 0.73, P < 0.001) was itself associated with lower 90-day mortality, indicating informative (non-ignorable) missingness. A sensitivity analysis incorporating albumin and LDH by iterative multiple imputation retained albumin as an additional

predictor and modestly increased validation discrimination (C-index 0.697 vs 0.690 for the six-variable model), whereas LDH was not retained. We therefore acknowledge that excluding these high-missingness variables may have discarded some prognostic information, and retained the parsimonious six-variable model to maximize clinical utility.

Table 4
Sensitivity analysis of the six predictors at 28-, 90-, and 365-day endpoints.

Variable	28-day HR (95% CI)	90-day HR (95% CI)	365-day HR (95% CI)
Age	1.018 (1.010–1.025)	1.015 (1.009–1.021)	1.016 (1.011–1.021)
BMI	0.972 (0.959–0.986)	0.972 (0.962–0.982)	0.978 (0.970–0.986)
APACHE III	1.023 (1.019–1.027)	1.019 (1.016–1.022)	1.015 (1.012–1.018)
Charlson comorbidity index	1.102 (1.063–1.142)	1.102 (1.072–1.133)	1.103 (1.077–1.130)
RDW	1.130 (1.095–1.166)	1.120 (1.091–1.149)	1.109 (1.084–1.134)
PaO_2	0.998 (0.996–0.999)	0.998 (0.997–0.999)	0.999 (0.998–0.999)

HR units are as defined in Table 2.

Discussion

In this retrospective cohort study of 2880 ICU patients with pressure injury, we developed and internally validated a nomogram for predicting 90-day mortality that integrates six readily available variables: age, BMI, APACHE III score, Charlson comorbidity index, RDW, and PaO2. The model showed good discrimination (C-index 0.699/0.690 and AUC 0.738/0.731 in the training and validation cohorts, respectively), satisfactory calibration, and net clinical benefit, with minimal overfitting on bootstrap validation. To our knowledge, this is among the few prognostic nomograms specifically targeting ICU patients who already have pressure injury.

The independent associations identified are biologically and clinically plausible. Age, acute physiology derangement, and comorbidity burden are established determinants of prognosis in critically ill patients [14, 18], and the strong performance of the APACHE III score and Charlson comorbidity index in our model is consistent with their documented prognostic value [14, 18, 27], reflecting the central role of physiological derangement and organ dysfunction captured by severity-of-illness and organ-failure scores [28, 29]. The protective effect of higher BMI mirrors the widely described "obesity paradox" in the ICU, whereby greater nutritional and metabolic reserve is associated with better survival [30], an observation recently corroborated in a MIMIC-IV analysis of critically ill patients with community-acquired pneumonia [31]. RDW, a routine and inexpensive index of anisocytosis that reflects ineffective erythropoiesis and systemic inflammation, emerged as one of the strongest predictors (HR 1.120), in line with accumulating evidence that elevated RDW independently predicts mortality across diverse ICU populations, including those with kidney failure and ARDS [32, 33]. Higher PaO2 was independently associated with lower 90-day mortality, an observational association consistent with recent data

suggesting that adequate oxygenation within the physiological range may be favourable in selected critically ill patients [34]; because FiO2 and ventilatory settings were not available, PaO2 could not be expressed as a P/F ratio, so this association should be interpreted cautiously given the confounding of oxygenation by illness severity and respiratory support.

Importantly, these markers are especially pertinent to patients with pressure injury. Malnutrition and systemic inflammation are both central to PI pathogenesis and are strong drivers of poor outcome in this population. The prognostic relevance of nutritional status—indexed by the prognostic nutritional index—has been demonstrated in ICU patients generally [35], in those receiving renal replacement therapy [36], and, notably, in ICU patients with pressure ulcers specifically [8]. Our finding that BMI, RDW, and oxygenation jointly contribute to prognosis is therefore concordant with the pathophysiology of this high-risk group.

Compared with most previous PI studies, which have focused on incidence, staging, and prevention [3, 4, 6], our study addresses the unmet need for prognosis prediction in patients who already have PI. Recent machine-learning efforts have predicted 90-day mortality in ICU patients with pressure ulcers using the Braden Scale [7] or have examined single nutritional biomarkers [8]; our nomogram, by contrast, offers a transparent, point-of-care tool based on six variables that are routinely captured at ICU admission, facilitating individualized risk stratification and early, targeted intervention.

A key clinical question is whether this nomogram offers value beyond the generic severity scores already available at the bedside. In our cohort, APACHE III, SOFA, and SAPS II used alone achieved C-indexes of only 0.629, 0.602, and 0.632 in the training cohort, respectively—considerably lower than the 0.699 of the nomogram—and the nomogram increased the category-free NRI by 0.588 and the IDI by 0.121 relative to APACHE III alone in the training cohort (0.540 and 0.102, respectively, in the validation cohort). These findings indicate that the additional variables—BMI, comorbidity burden, RDW, and oxygenation—capture prognostic information beyond acute physiological derangement alone, supporting the use of this PI-specific tool over a generic severity score in this population.

Several limitations should be acknowledged. First, the study is retrospective and single-center, and although internal validation indicated minimal overfitting, external validation in independent, multicenter cohorts is required before clinical application [37]. Second, some laboratory variables had missing values that required median imputation; in particular, several pressure-injury-relevant biomarkers (serum albumin, C-reactive protein, and leukocyte differentials) were missing in 57–95% of patients and therefore could not be included, which may have omitted additional prognostic information (Supplementary Table S1). A multiple-imputation sensitivity analysis indicated that this exclusion likely discarded some prognostic information (albumin was retained as an additional predictor with a modest improvement in validation discrimination); we prioritized a parsimonious model to maximize clinical utility. Third, the stage, site, and severity of pressure injury, as well as the Braden Scale, were not available in MIMIC-IV and could not be incorporated [3, 5]. Fourth, because pressure injury was identified from coded diagnoses (ICD-9 707.0x / ICD-10 L89.x) without a present-on-admission flag or a diagnosis

timestamp in our extract, we could not distinguish admission-present from ICU-acquired pressure injuries, nor perform the recommended sensitivity analysis restricted to pressure injury coded within 48 hours or a landmark analysis at the time of pressure injury diagnosis. If ICU-acquired cases were included, the use of day-1 physiology to predict subsequent mortality could introduce a degree of immortal-time bias. Follow-up was anchored at ICU admission (time zero)—the earliest point at which all covariates were measured—and the restriction to first ICU admissions minimizes inclusion of long-stay survivors, but fully resolving this issue would require re-querying the diagnoses_icd table with its poa field and diagnosis date. Fifth, BMI in critically ill patients may be confounded by fluid overload and edema, so attributing low BMI solely to malnutrition warrants caution. Sixth, FiO2 and ventilatory settings were not available in our extract, so PaO2 could not be expressed as the P/F ratio; the oxygenation association should therefore be interpreted with caution, although PaO2 was essentially uncorrelated with APACHE III (Pearson r = − 0.03) and contributed independently to the model. Finally, treatments and interventions received during the ICU stay were not fully accounted for.

Conclusions

We developed and internally validated a six-variable nomogram—incorporating age, BMI, APACHE III score, Charlson comorbidity index, RDW, and PaO2—that reliably predicts 90-day mortality in ICU patients with pressure injury. The model demonstrated good discrimination, calibration, and clinical utility, and may aid individualized risk stratification and early intervention in this high-risk population. An accompanying free online calculator is provided to facilitate clinical translation. External validation in prospective, multicenter cohorts is warranted.

Declarations

Clinical trial number: not applicable.

Ethics approval and consent to participate:

The study used de-identified, publicly available data from the MIMIC-IV database, for which institutional review board approval and informed consent were waived.

Competing interests:

The authors declare no competing interests.

Funding:

None.

Author Contribution

T.X. and L.T. conceived and designed the study. T.X., Z.L., and C.W. collected and curated the data and performed the statistical analysis. T.X. wrote the main manuscript text and prepared the figures and tables. X.H. and L.T. contributed to the interpretation of the results and critically revised the manuscript. All authors reviewed and approved the final manuscript.

Data Availability

The datasets used and/or analysed during the current study are available in the MIMIC-IV database (https://mimic.mit.edu/), a freely accessible electronic health record dataset. The analysis code is available from the corresponding author on reasonable request.

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Figures

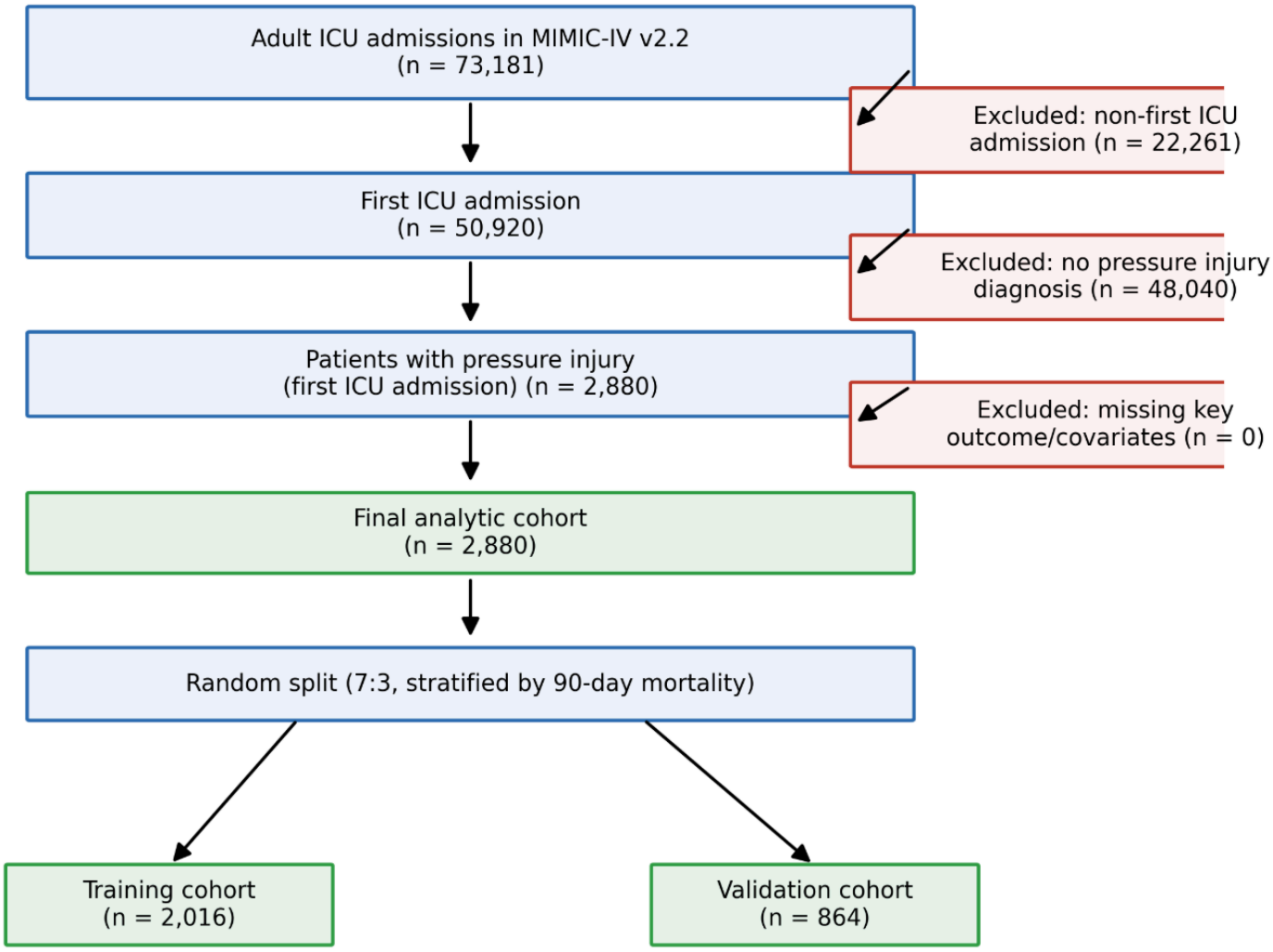


Figure 1

Flow diagram of study cohort selection and assignment.

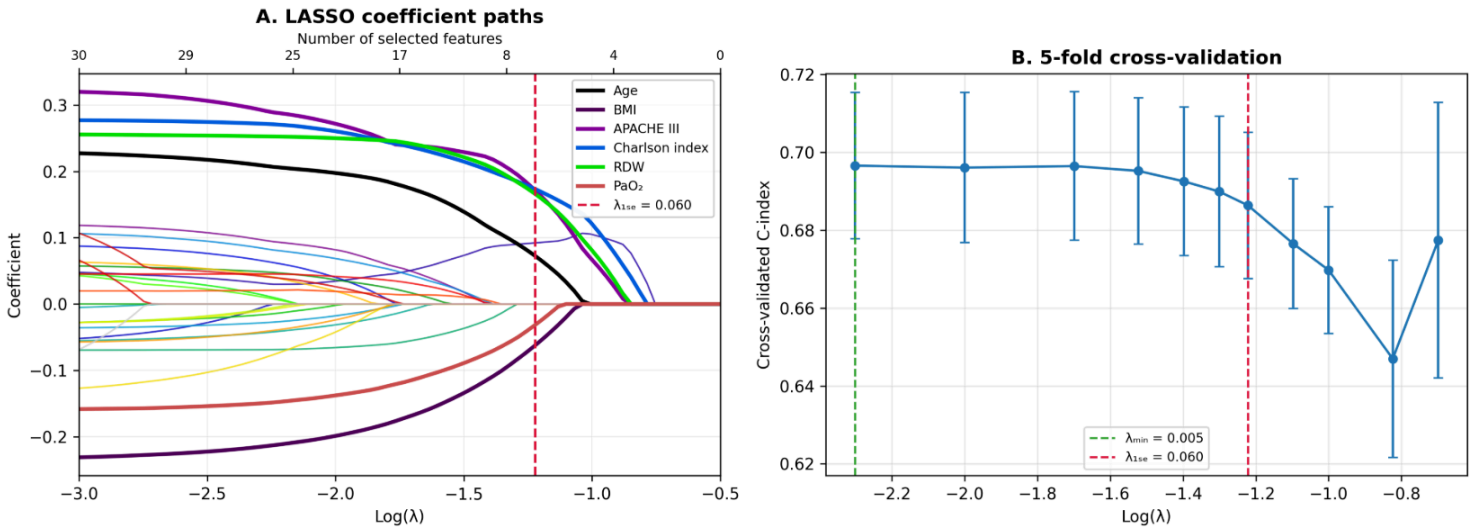


Figure 2

LASSO coefficient paths and 5-fold cross-validation for variable selection.

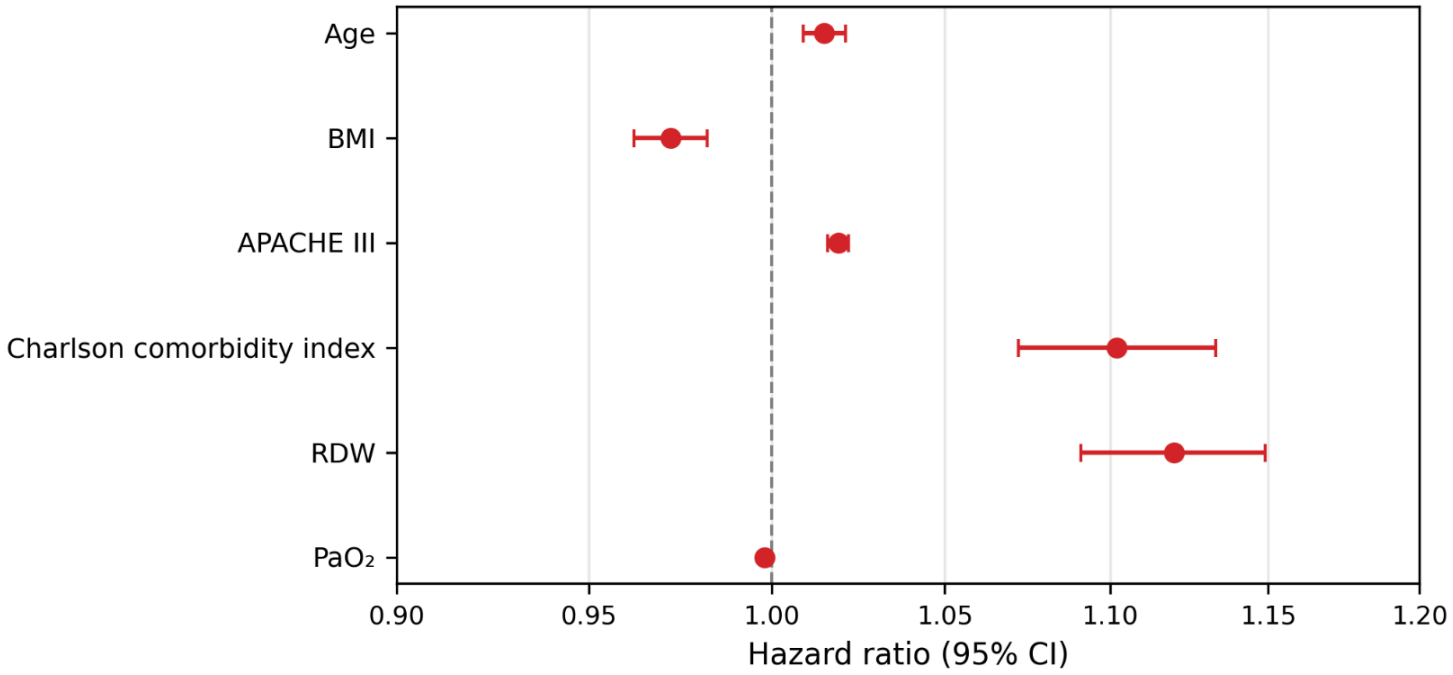


Figure 3

Forest plot of the six predictors in the final model.

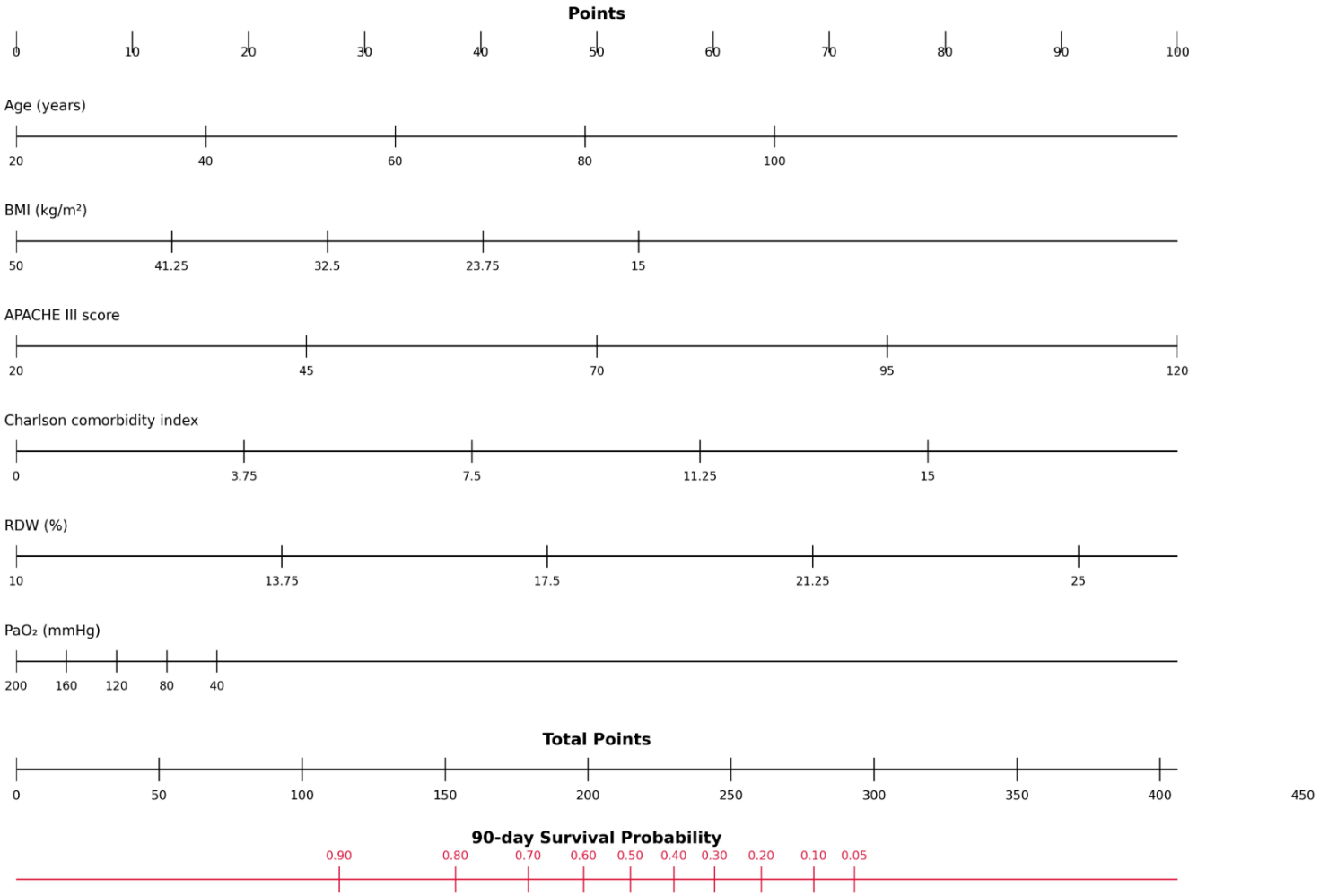


Figure 4

Nomogram for predicting 90-day mortality in ICU patients with pressure injury.

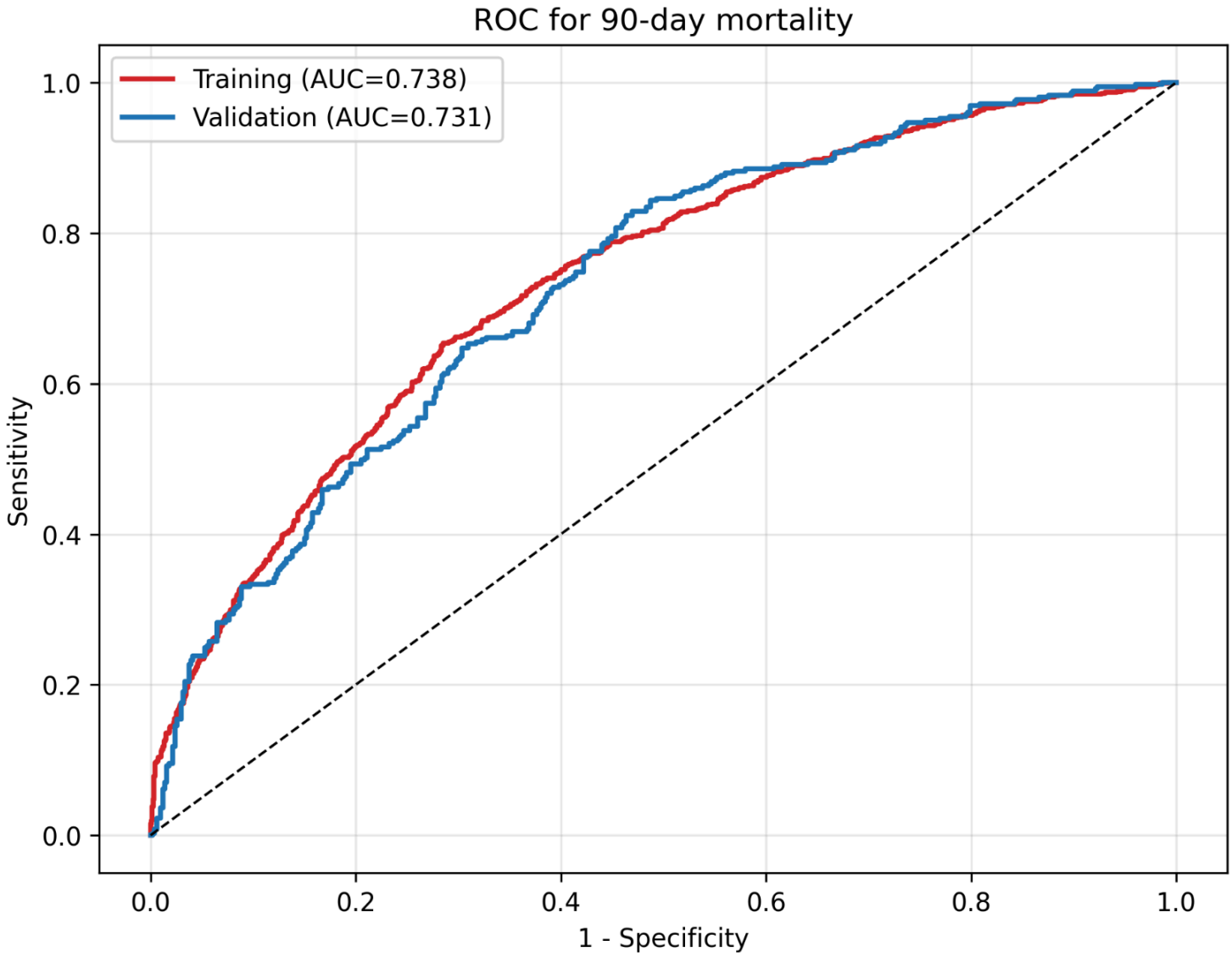


Figure 5

Receiver operating characteristic curves for the training and validation cohorts.

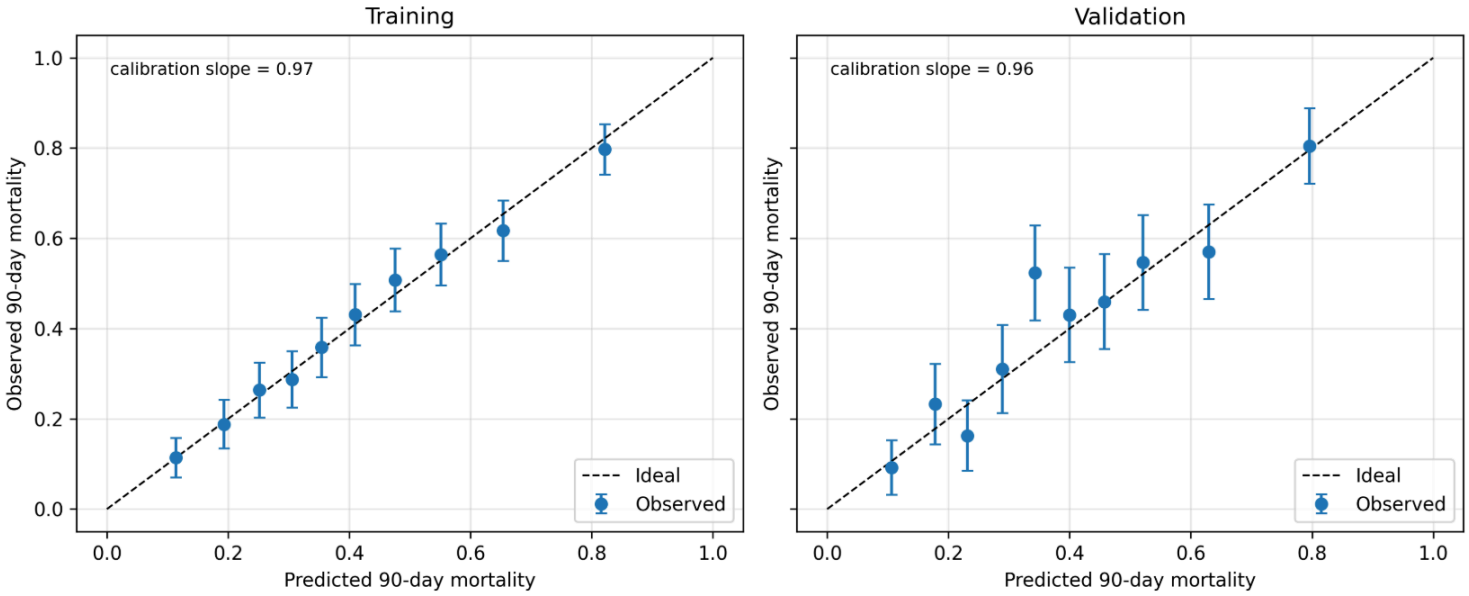


Figure 6

Calibration curves for the training and validation cohorts.

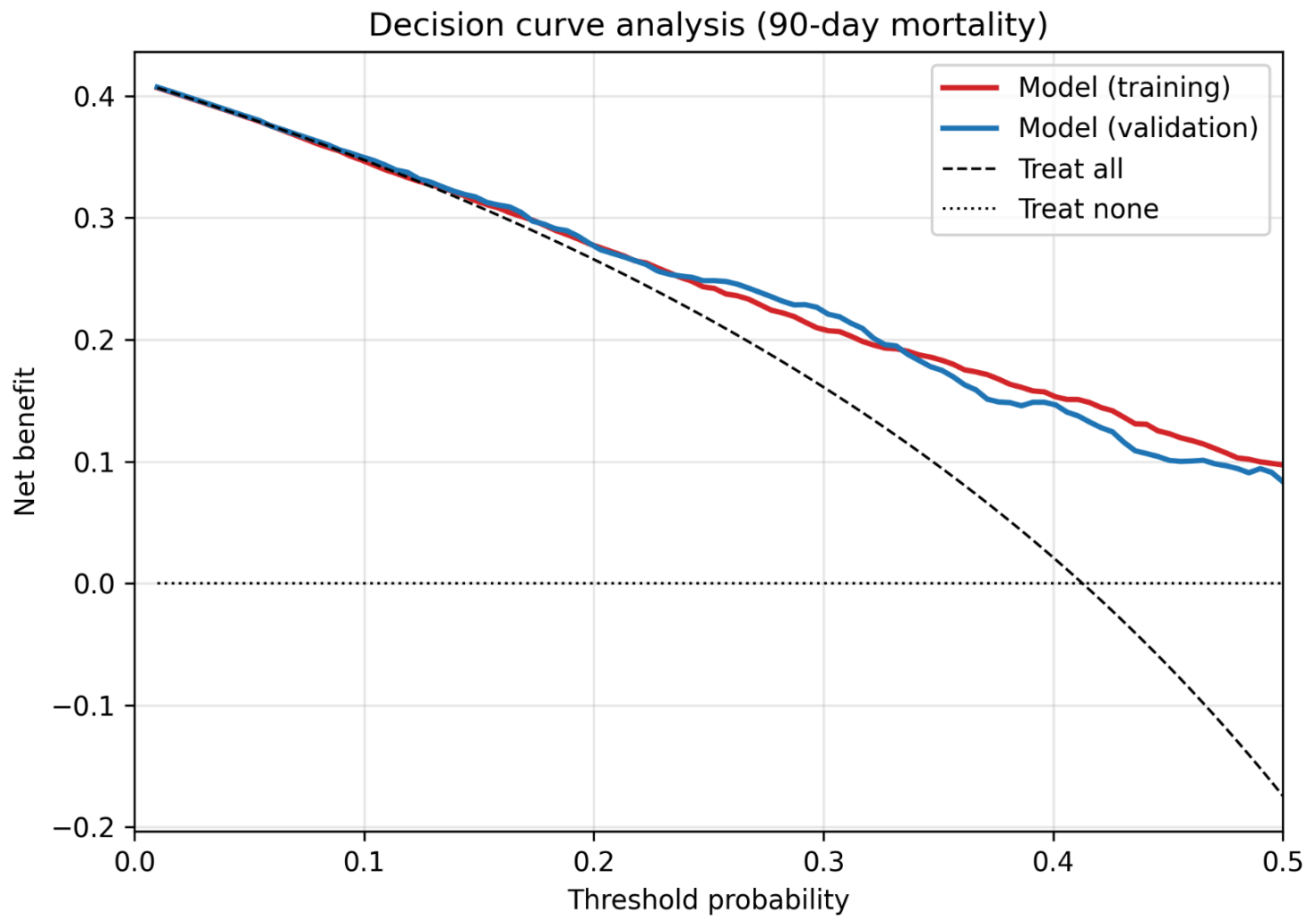


Figure 7

Decision curve analysis for the 90-day mortality nomogram.

Supplementary Files

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- SupplementaryFigureTable.docx