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Early haemodynamic risk stratification in critically ill patients: comparative performance of four shock indices for 7- and 28-day mortality

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Abstract

Background

The standard shock index (SI) heart rate divided by systolic blood pressure relies solely on systolic pressure and may not capture haemodynamic deterioration in vasodilation predominant states. We compared SI with three alternative formulations diastolic (DSI), age-weighted (Age SI), and modified (MSI) shock indices for short-term mortality prediction in a heterogeneous cohort of critically ill adults requiring intensive care unit admission.

Methods

This single-centre retrospective cohort study analysed 598 adult patients admitted from the emergency department to the intensive care unit of Mardin Training and Research Hospital between 1 January and 31 December 2024. The four indices were calculated from admission vital signs. Discrimination for 7-day and 28-day all-cause mortality was assessed by receiver operating characteristic (ROC) analysis with DeLong pairwise comparisons. Independent prognostic value was tested in four separate multivariable logistic regression models, adjusted for age, lactate, albumin, C-reactive protein, procalcitonin, coronary artery disease, and malignancy. Reporting followed the STROBE statement.

Results

The 7-day and 28-day mortality rates were 22.7% and 36.0%, respectively. For 7-day mortality, the four indices showed comparable discrimination (AUC 0.71–0.72; all DeLong $p > 0.05$). For 28-day mortality, SI showed lower discrimination than the three diastolic-inclusive alternatives (SI vs DSI ΔAUC − 0.025, $p = 0.004$; SI vs Age SI ΔAUC − 0.041, $p = 0.007$; SI vs MSI ΔAUC − 0.016, $p = 0.001$). After multivariable adjustment, all four indices remained independent predictors of both endpoints (all $p < 0.001$). For 28-day mortality, the largest standardised effect was observed for DSI (1.92 per 1 standard deviation), followed by MSI (1.81), Age SI (1.74), and SI (1.64).

Conclusions

In this cohort of critically ill adults, all four shock indices were independently associated with 7-day and 28-day mortality. Diastolic-inclusive indices showed greater discrimination than the standard SI for 28-day outcomes. Calculated from routinely available heart rate and blood pressure values, DSI may serve as a complementary haemodynamic marker alongside the standard SI in early risk assessment. Prospective multicentre validation of the proposed thresholds (DSI ≈ 2.10–2.25; Age SI ≈ 51) is required before clinical implementation.

Background

Rapid identification of critically ill patients presenting to the emergency department constitutes one of the most fundamental problems of emergency medicine in terms of preventing unnecessary delays and ensuring appropriate resource utilisation. Vital signs are still the primary point of reference in the early detection of haemodynamic instability; however, no single parameter alone achieves sufficient sensitivity and specificity. The shock index (SI), developed to fill this gap, has been used since the 1960s as the ratio of heart rate to systolic blood pressure and has gained widespread use in emergency practice due to its ease of calculation and immediate applicability [1, 2]. However, SI has been subject to criticism because it considers only systolic pressure and ignores diastolic pressure, which is an important indicator of vascular tone [3].

From a physiological perspective, diastolic arterial pressure in healthy individuals is largely determined by systemic vascular tone and remains relatively constant along peripheral vessels in the absence of aortic valve pathology [4]. In cases where vascular tone is lost, a drop in diastolic pressure can occur before systolic pressure changes significantly; this may result in missed identification of critically ill patients. Moreover, since coronary perfusion continues throughout diastole, diastolic hypotension is associated with increased risk of myocardial ischaemia and with greater severity of acute illness [5]. Based on this pathophysiological background, the diastolic shock index (DSI) — defined as the ratio of heart rate to diastolic blood pressure — has been proposed and has shown promising results, particularly in predicting septic shock and the need for vasopressors [3, 6].

Various modifications to the shock index have also been described in the literature. The age-weighted shock index (Age SI) incorporates patient age by multiplying the standard SI by the patient's age in years; the modified shock index (MSI) is calculated by the ratio of heart rate to mean arterial pressure, indirectly reflecting both systolic and diastolic components [7, 8]. Studies directly comparing these four indices are quite limited, and the vast majority have been conducted on a single diagnostic group (especially sepsis or trauma). Simultaneous comparison of the four indices in emergency department patients with heterogeneous presentation profiles, and moreover, only in the critical subgroup admitted to the ICU, has not yet been sufficiently addressed in the literature.

We therefore conducted a retrospective cohort study in critically ill adult patients admitted directly from the emergency department to the ICU, comparing the prognostic accuracy of SI, DSI, Age SI, and MSI for 7-day and 28-day all-cause mortality, with the primary hypothesis that indices incorporating diastolic blood pressure would outperform standard SI in predicting short-term outcomes.

Methods

This study is a single-centre retrospective cohort study conducted by retrospectively reviewing the records of patients admitted from the adult emergency department to the intensive care unit of Mardin Training and Research Hospital during the calendar year 2024. The study was approved by the Mardin Artuklu University Non-Interventional Clinical Research Ethics Committee on 10 September 2024

(decision number 2024/9-10), and retrospective extraction of patient records from the hospital information system was performed only after ethics committee approval was granted; the period from 1 January to 31 December 2024 refers to the patient admission window covered by the chart review, not to the period of data collection by the investigators. Data were obtained through the online patient record system, and mortality information was confirmed by scanning the Death Notification System. Vital status at 28 days was ascertained for 100% of analysed patients via the national Death Notification System; no patient was lost to follow-up. Due to the retrospective nature of the study and the use of anonymised data, individual patient consent was not obtained.

Of 939 adult patients admitted directly from the ED to the ICU between 1 January and 31 December 2024, 263 were excluded for incomplete or unverifiable admission data (missing vital signs, laboratory values, or follow-up/mortality information) and 78 for predefined exclusion criteria (age < 18 years, n = 25; repeated ICU admissions or ward-to-ICU re-admissions during follow-up, n = 53), yielding 598 patients in the analytic cohort (Figure 1). Inclusion required direct ED-to-ICU admission with complete data for the primary exposures (heart rate, systolic and diastolic blood pressure), the primary outcomes (7-day and 28-day mortality), and all seven prespecified covariates. Missing data were handled by complete-case analysis without imputation; the per-variable pattern of missingness could not be reconstructed at the individual level under local data-protection constraints, and is acknowledged as a limitation. Vital status at 28 days was confirmed in 100% of analysed patients via the national Death Notification System, with no loss to follow-up.

The four shock indices were calculated from initial ED admission vital signs as follows: SI = HR/SBP; DSI = HR/DBP; Age SI = age × SI; and MSI = HR/MAP, where MAP = (SBP + 2 × DBP)/3. Laboratory values were drawn from samples obtained at initial admission. Diagnostic categories and ICU level definitions are reported in Table 1. The primary endpoints were 7-day and 28-day all-cause mortality. All measurements were obtained as part of routine clinical care using identical monitoring protocols and a single central laboratory throughout the study period.

Statistical analyses were performed using SPSS 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as median (interquartile range, IQR) according to distribution; categorical variables were presented as frequency and percentage. Normality of distribution was assessed using the Shapiro–Wilk test. The Mann–Whitney U test was used for group comparisons of non-normally distributed continuous variables, and the Pearson χ^2 test (or Fisher's exact test where expected cell counts were < 5) was used for categorical variables. The threshold for statistical significance was $p < 0.05$ in all analyses.

The diagnostic accuracy of shock indices in predicting mortality was evaluated using receiver operating characteristic (ROC) curve analysis; areas under the curve (AUC) were reported with 95% confidence intervals (CI). The statistical significance of differences between paired ROC curves was tested using the DeLong method. Optimal cut-off values were determined by the Youden index (sensitivity + specificity − 1). All cut-off values reported in this study were derived post-hoc from the same dataset and have not

been externally validated; their generalisability requires confirmation in independent cohorts. No formal correction for multiple comparisons was applied; the DeLong pairwise comparisons were exploratory in nature, and primary inference was based on AIC and Nagelkerke R^2 ranking of the eight prespecified multivariable models described below.

To determine whether shock indices were independent predictors of mortality, four separate multivariable logistic regression models — one per index — were constructed for each outcome, yielding eight models in total. All models were adjusted for the same seven prespecified covariates: age, lactate, albumin, C-reactive protein, procalcitonin, coronary artery disease, and malignancy, selected on the basis of biological plausibility and prior evidence in critical illness. Each shock index was entered as a continuous variable. Separate models were used because the four indices share heart rate as numerator and overlapping denominators, producing extreme multicollinearity (Spearman r 0.937–0.986; VIF > 500 in a single combined model) that precluded simultaneous inclusion. Discrimination was assessed by Nagelkerke R^2; calibration by the Hosmer–Lemeshow test. No a priori sample size calculation was performed; all eligible patients were included. Two prespecified sensitivity analyses were conducted: lactate-dropped multivariable models, and age-stratified subgroup analyses (≥ 65 vs < 65 years). Reporting followed the STROBE guidelines (Additional File 1).

Results

Of 939 patients initially identified, 598 met the inclusion criteria and were included in the final analysis (Figure 1). The cohort had a median age of 68 years (IQR 50–80), and 53.3% (n = 319) were male. The most frequent comorbidities were hypertension (42.3%), coronary artery disease (33.3%), and diabetes mellitus (31.8%); metabolic and infectious diagnoses (32.6%) and respiratory diseases (26.9%) were the most common reasons for ICU admission. Vasopressor support was required by 43.8% of patients, 12.2% were intubated in the emergency department, and 47.2% were intubated in the ICU. The median total length of stay was 5.0 days (mean 9.2 ± 14.2). Full baseline characteristics, vital signs, and laboratory values are presented in Table 1.

The 7-day and 28-day mortality rates were 22.7% (n = 136) and 36.0% (n = 215), respectively. Compared with survivors, non-survivors at both endpoints had significantly lower systolic and diastolic blood pressure, albumin, and platelet counts, and significantly higher heart rate, lactate, glucose, C-reactive protein, and procalcitonin (all $p < 0.05$). At 28 days, the median lactate was 2.7 mmol/L in non-survivors versus 1.9 mmol/L in survivors ($p < 0.001$). Malignancy was more frequent in non-survivors at both endpoints ($p < 0.001$), and coronary artery disease additionally at 28 days ($p = 0.003$). All four shock indices were significantly higher among non-survivors at both endpoints (all $p < 0.001$), with median DSI 2.34 vs 1.50 at 7 days and 2.16 vs 1.45 at 28 days. Vasopressor use, ED intubation, and ICU intubation rates were all significantly higher in non-survivors (all $p < 0.001$). Detailed comparisons are presented in Table 2.

All four indices showed significant discriminative power for both endpoints (all AUC $p < 0.001$; Table 3, Figures 2 and 3). For 7-day mortality, the four indices performed comparably: DSI achieved the highest AUC (0.720; 95% CI 0.667–0.772), followed closely by MSI (0.719), Age SI (0.715), and SI (0.709). At their Youden cut-offs, DSI achieved the highest specificity (76.4% at 2.10), while Age SI achieved the highest sensitivity (80.1% at 51.0). For 28-day mortality, the ranking shifted: Age SI ranked highest by AUC (0.707; 95% CI 0.663–0.752), followed by DSI (0.691), MSI (0.683), and SI (0.667). At a cut-off of 2.25, DSI yielded the highest specificity (86.2%) and positive predictive value (66.0%; 95% CI 58.3–73.0%) among all four indices for 28-day mortality, with a corresponding negative predictive value of 74.7% (95% CI 70.4–78.5%). All operating characteristics with Wilson 95% confidence intervals are summarised in Table 3A.

In pairwise DeLong comparisons (Table 3B), no significant differences emerged between any pair of indices for 7-day mortality (all $p > 0.05$). For 28-day mortality, however, SI was significantly inferior to all three diastolic-inclusive alternatives: SI vs DSI (ΔAUC −0.025; 95% CI −0.042 to −0.008; $p = 0.004$), SI vs Age SI (ΔAUC −0.041; 95% CI −0.071 to −0.011; $p = 0.007$), and SI vs MSI (ΔAUC −0.016; 95% CI −0.025 to −0.007; $p = 0.001$). DSI was also marginally superior to MSI for 28-day mortality (ΔAUC +0.009; $p = 0.032$), while DSI vs Age SI and Age SI vs MSI did not reach statistical significance.

After adjustment for age, lactate, albumin, C-reactive protein, procalcitonin, coronary artery disease, and malignancy, all four indices remained independent predictors of both 7-day and 28-day mortality (all $p < 0.001$; Table 4). For 28-day mortality, DSI achieved the lowest AIC (645.0), the highest Nagelkerke R^2 (0.312), and the largest standardised effect (1.92 per 1 SD), followed by MSI (AIC 651.4; standardised OR 1.81), SI (AIC 660.2; 1.64), and Age SI (AIC 661.6; 1.74). The AIC gap of approximately 16 units between DSI and the worst-fitting model is conventionally considered substantial evidence favouring DSI. Among the seven covariates, lactate (adjusted OR ≈ 1.20–1.24 per mmol/L), albumin (≈ 0.62–0.66 per g/dL), and malignancy (≈ 2.00–2.10) were consistently independent predictors across all eight models, whereas CRP, procalcitonin, and coronary artery disease did not reach independent significance. All models showed adequate calibration (Hosmer–Lemeshow $p > 0.05$). Full covariate-level estimates for each model are provided in Additional File 2.

Both sensitivity analyses supported the robustness of the primary findings (Table 5). When lactate was removed from the multivariable models, all four indices remained independent predictors of both endpoints (all $p < 0.001$), but the magnitude of the OR shift varied: SI showed the largest dependence on lactate (OR change +35–43%), MSI an intermediate dependence (+21–27%), and DSI a smaller one (+14–18%); Age SI showed negligible change (+0.4–0.5%, reflecting its near-unity OR scale). In age-stratified subgroup analyses (≥ 65 vs < 65 years), all indices retained significance in both strata (all $p < 0.05$). DSI showed the most stable discrimination across age groups for 28-day mortality (AUC 0.688 vs 0.699; $\Delta = -0.011$), whereas Age SI showed the largest age-dependent variation (AUC 0.656 vs 0.762; $\Delta = -0.106$), consistent with age being embedded in its calculation.

Discussion

In this retrospective cohort of 598 critically ill adults admitted directly from the emergency department to the intensive care unit, all four shock indices were independent predictors of both 7-day and 28-day mortality. Their relative discriminative performance was time-dependent: at seven days the four indices performed equivalently, whereas at 28 days standard SI showed significantly lower discrimination than its three diastolic-inclusive counterparts. This temporal pattern is consistent with diastolic-inclusive indices providing additional prognostic information over longer follow-up, although the difference did not eliminate SI as an independent predictor. Prior work has highlighted SI's limited sensitivity in isolation [9, 10]; our findings reinforce that no single haemodynamic ratio should be interpreted independently of biochemical markers and clinical context.

The physiological rationale for the discriminative advantage observed with DSI relates to the behaviour of diastolic pressure under conditions of vascular tone loss. Because diastolic arterial pressure is primarily determined by systemic vascular resistance, it tends to decline earlier and more steeply than systolic pressure in vasodilation-predominant states; consequently, heart rate-to-diastolic ratios may detect deterioration not yet evident in systolic-based indices [2, 3]. This mechanism is supported by direct clinical evidence: Ospina-Tascón et al. demonstrated in two independent septic shock cohorts (a preliminary cohort of 337 patients and 424 patients enrolled in the ANDROMEDA-SHOCK trial) that mortality risk increased proportionally with rising DSI [3]. Our findings are consistent with this observation in a broader population. Although only 32.6% of our cohort presented with infectious or metabolic diagnoses — a profile distinct from pure septic shock — DSI showed greater 28-day discrimination than SI, the lowest AIC, and the largest standardised effect size among the four indices. Together, these patterns are consistent with vascular tone loss being a prognostically relevant phenomenon across a wider spectrum of critical illness, not limited to sepsis.

The independent prognostic weight of diastolic pressure was recognised before DSI was formally defined. Benchekroune et al. demonstrated that a DBP threshold of > 50 mmHg measured on day three of septic shock predicted in-hospital survival more strongly than both SAPS II and LODS scores, whereas systolic and mean arterial pressure carried no significant predictive value at the same time point [11]. Our reliance on a single admission measurement is a genuine limitation; nonetheless, the persistence of DSI's independent significance for 28-day mortality (adjusted OR 2.02; $p < 0.001$) suggests that even a single early, static diastolic measurement is associated with sustained prognostic information — likely a conservative estimate of what serial monitoring might yield.

A threshold is only clinically useful if it is both accurate and memorable. Kim et al. constructed a bedside screening rule combining DSI ≥ 2.0 with lactate ≥ 2.5 mmol/L in 1,917 hypotensive ED patients with suspected infection, achieving an AUC of 0.741 for vasopressor requirement prediction [6]. Our independently derived cut-offs — 2.10 for 7-day mortality and 2.25 for 28-day mortality — converge on this range without having been calibrated to an infection-specific population. At the proposed thresholds, both DSI and Age SI achieved clinically meaningful separation between high- and low-risk strata for 28-day mortality: a DSI ≥ 2.25 corresponded to a positive predictive value of 66.0% (95% CI 58.3–73.0%) compared with a negative predictive value of 74.7% (95% CI 70.4–78.5%), translating the relative risk

estimates reported here into actionable triage thresholds for emergency department clinicians. The convergence across independent cohorts with markedly different composition is consistent with the "DSI ≈ 2.0–2.3" range representing a generalisable physiological threshold rather than a population-specific artefact; whether it is sufficiently robust for guideline incorporation remains a question for prospective multicentre validation.

Whether age adds prognostic information beyond what haemodynamics already capture has been a recurring question. Lee et al. answered affirmatively in the intubation setting, where Age SI (AUC 0.676) outperformed both SI and MSI in predicting post-intubation hypotension [12]; Zarzaur et al. reached the same conclusion in 16,077 blunt trauma patients [7]. Our data extend this consensus to a non-traumatic, heterogeneous ICU population and add a temporal dimension neither prior study examined: Age SI's discriminative advantage emerged specifically at 28 days, not at 7 days, a pattern consistent with age-related physiological reserve becoming more prognostically informative over longer follow-up — a distinction with direct clinical relevance in a cohort with a mean age of 63.9 years.

MSI occupied an intermediate position in our analyses: it outperformed standard SI for 28-day mortality discrimination (ΔAUC + 0.016; p = 0.001) and remained an independent predictor after multivariable adjustment (adjusted OR 2.45; 95% CI 1.75–3.43; p < 0.001), yet did not match the model-fit performance of DSI (AIC 651.4 vs 645.0; Nagelkerke R^2 0.300 vs 0.312). This intermediate behaviour is biologically plausible: because mean arterial pressure incorporates diastolic pressure as a weighted component (MAP = [SBP + 2 × DBP] / 3), MSI captures part of the vascular-tone information that DSI captures more directly, but dilutes this signal by averaging it with systolic pressure. This pattern was further supported by sensitivity analyses (Table 5A): when lactate was removed from the multivariable models, MSI's adjusted OR increased substantially (+ 21–27%), whereas DSI's increased only modestly (+ 14–18%), consistent with MSI's prognostic signal being more biochemically mediated than that of DSI. Liu et al. and Singh et al. previously established MSI's superiority over SI in general ED and trauma populations [8, 13]; our findings extend this consensus to a heterogeneous critically ill cohort while clarifying that the MSI advantage, although real, is partial — when diastolic information is the prognostic driver, the unmediated DSI is more informative than its averaged surrogate.

Beyond static measurements, several lines of evidence suggest that dynamic indexing may add prognostic value: cumulative SI exposure across hours has been associated with mortality in critically ill patients [14], delta-SI between triage and ICU admission has carried information beyond either endpoint alone [15], and failure of vital sign normalisation has predicted outcome better than single readings [16]. Our single-timepoint design cannot address these dynamic dimensions; nonetheless, the persistence of DSI and Age SI as independent predictors from a single ED measurement supports their practical utility precisely when triage decisions must be made within minutes. Whether delta-DSI between pre-hospital, ED, and ICU contact yields additional discriminative power represents a clear direction for future work.

In the trauma literature, SI ≥ 1.0 has long served as a binary danger threshold. A 2022 meta-analysis by Vang et al. pooling 348,687 patients reported approximately fourfold higher in-hospital mortality in

patients exceeding this threshold [17], and Lai et al. demonstrated that patients whose SI normalised between prehospital and ED assessment still faced 5.8-fold higher mortality than those who remained stable [18]. Our cohort is non-traumatic, precluding direct comparison; yet the convergence of our optimal SI cut-off for 7-day mortality (1.00) with the canonical trauma threshold raises the possibility that SI ≥ 1.0 may correspond to a haemodynamic decompensation point shared across aetiologies. The consistency between our cohort (adjusted OR 3.41 for SI at day 7) and the trauma meta-analysis (pooled RR 4.15) is consistent with this interpretation but does not establish causality.

The ANDROMEDA-SHOCK trial, comparing peripheral perfusion-targeted with lactate-targeted resuscitation in septic shock, raised a broader question relevant here: which haemodynamic parameter most faithfully tracks the perfusion deficit that ultimately determines outcome [19]. Diastolic-inclusive indices, by capturing vascular tone, may align more closely with this perfusion-centred framework — a hypothesis that prospective studies could feasibly test.

The operational appeal of DSI lies in its calculability at the moment of first clinical contact — by paramedics during pre-hospital transport, by triage nurses at ED arrival, or by attending physicians at the bedside — using only heart rate and a manual or automated diastolic pressure reading. Unlike biomarker-based risk stratifiers requiring laboratory turnaround, no additional equipment or training infrastructure is needed beyond the calculation itself. In trauma systems where pre-hospital decision-making determines transport destination and trauma team activation, a haemodynamic index sensitive to vascular tone loss may have particular operational relevance. Although our cohort was non-traumatic, the underlying physiological premise (diastolic pressure as an early marker of vascular tone failure) is mechanism-based rather than aetiology-specific, supporting cautious extrapolation pending dedicated validation studies.

Strengths and limitations

The principal strengths of this study include the use of all eligible patients during a continuous 12-month accrual window from a routine emergency department-to-ICU pathway; a prespecified covariate set selected on biological grounds rather than by data-driven inclusion; head-to-head comparison of all four shock indices in the same cohort using paired (DeLong) statistical inference; and prespecified sensitivity analyses (lactate-dropped models and age-stratified subgroup analyses) demonstrating robustness of the primary findings.

Several limitations should nonetheless be acknowledged. First, the retrospective design, although based on prospectively collected clinical records, cannot fully eliminate the risk of selection bias; the predefined exclusion criteria were applied uniformly, but the possibility of unmeasured confounding remains. Second, the study was conducted in a single centre, and the regional case-mix and ICU configuration of Mardin Training and Research Hospital may limit external generalisability; the heterogeneous diagnostic distribution of the cohort partially mitigates this concern by reflecting routine ED practice rather than a single disease-defined population. Third, shock indices were calculated from a

single admission measurement, precluding assessment of dynamic indices that may yield additional discriminative information; this limitation is particularly relevant for pre-hospital applications, where temporal trajectories during transport may carry independent prognostic value. Fourth, missing data were handled by complete-case analysis without imputation, and the baseline characteristics of the 263 excluded patients could not be reconstructed at the individual level under local data-protection constraints, raising the possibility of systematic differences. Fifth, established critical illness severity scores (SOFA, APACHE II) were not available in the dataset; their absence does not invalidate the primary findings but warrants direct comparison in future prospective studies.

Conclusions

In this retrospective cohort of 598 critically ill adults admitted from the emergency department to the intensive care unit, all four shock indices were independent predictors of both 7-day and 28-day mortality after multivariable adjustment. For 28-day outcomes, DSI provided the strongest discrimination, the largest standardised effect, and the lowest AIC, while standard SI was significantly inferior in pairwise comparisons. Interpretation alongside biochemical markers such as lactate and albumin further strengthens prognostic estimation. DSI may complement, rather than replace, the standard SI at the point of first clinical contact. Prospective multicentre validation of the proposed thresholds (DSI ≈ 2.10–2.25; Age SI ≈ 51) is warranted before incorporation into clinical pathways.

Abbreviations

AIC
Akaike Information Criterion
AUC
area under the curve
Age SI
age-weighted shock index (age × SI)
CAD
coronary artery disease
CI
confidence interval
CRP
C-reactive protein
DBP
diastolic blood pressure
DSI
diastolic shock index (heart rate / diastolic blood pressure)
ED
emergency department
ICU

intensive care unit
MAP
mean arterial pressure
MSI
modified shock index (heart rate / mean arterial pressure)
NPV
negative predictive value
OR
odds ratio
PPV
positive predictive value
ROC
receiver operating characteristic
SBP
systolic blood pressure
SD
standard deviation
SI
shock index (heart rate / systolic blood pressure)
STROBE
Strengthening the Reporting of Observational Studies in Epidemiology.

Declarations

Ethics approval and consent to participate

This study was approved by the Non-Interventional Clinical Research Ethics Committee of Mardin Artuklu University (decision number: 2024/9–10; approval date: 10 September 2024). Data extraction from the hospital information system commenced only after ethics committee approval was granted; the calendar year 2024 refers to the patient admission window covered by the chart review, not to the period during which the investigators accessed patient records. The study was conducted in accordance with the Declaration of Helsinki and local regulations. Written informed consent was waived by the ethics committee due to the retrospective nature of the study and the use of anonymised data.

Consent for publication

Not applicable. This manuscript does not contain any individual person's identifiable data in any form.

Competing interests

The authors declare no competing interests.

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Author Contribution

MA conceived the study, designed the methodology, and performed the statistical analyses. MFC and FY contributed to study coordination, data curation, and patient record acquisition. MA drafted the original manuscript. All authors contributed to interpretation of results, critically revised the manuscript for important intellectual content, and approved the final version prior to submission.

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Not applicable.

Data Availability

The dataset analysed during the current study is not publicly available because local ethics approval and data protection regulations do not authorise public release of individual-level patient data, but is available from the corresponding author upon reasonable request.

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Tables

Table 1. Baseline characteristics of the study cohort (n = 598)

Variable	Median (IQR) or n (%)
Demographics	
Age, years	68 (50–80)
Female sex	279 (46.7%)
Comorbidities (one patient may belong to >1 category)	
Hypertension	253 (42.3%)
Diabetes mellitus	190 (31.8%)
Coronary artery disease	199 (33.3%)
Renal failure	99 (16.6%)
Chronic obstructive pulmonary disease	100 (16.7%)
Malignancy	65 (10.9%)
Liver disease	26 (4.3%)
Primary diagnosis	
Neurological	108 (18.1%)
Respiratory	161 (26.9%)
Cardiac	42 (7.0%)
Metabolic / infectious	195 (32.6%)
Electrolyte disorders	6 (1.0%)
Traumatic	63 (10.5%)
Intentional overdose	13 (2.2%)
Emergency surgical	10 (1.7%)
Vital signs	
Systolic blood pressure, mmHg	110 (90–133)
Diastolic blood pressure, mmHg	60 (50–78)
Heart rate, beats/min	105 (85–120)
Pulse pressure, mmHg	50 (40–60)
Temperature, °C	36.3 (36.1–36.6)
Laboratory values	
Lactate, mmol/L	2.1 (1.4–3.6)

Glucose, mg/dL	143 (108–217)
Potassium, mEq/L	4.2 (3.8–4.8)
Albumin, g/dL	3.4 (3.0–3.9)
C-reactive protein, mg/L	26.7 (7.2–100.3)
Procalcitonin, ng/mL	0.35 (0.07–2.33)
White blood cell count, ×10³/μL	12.0 (8.8–16.0)
Platelets, ×10³/μL	230 (172–297)
Neutrophils, ×10³/μL	8.9 (6.0–12.8)
Lymphocytes, ×10³/μL	1.4 (0.8–2.4)
Shock indices	
Shock index	0.87 (0.68–1.24)
Diastolic shock index	1.63 (1.17–2.30)
Age-weighted shock index	54.1 (36.0–81.9)
Modified shock index	1.25 (0.94–1.76)
Clinical interventions and outcomes	
Vasopressor support	262 (43.8%)
ICU — Level 2	206 (34.4%)
ICU — Level 3 anaesthesia	173 (28.9%)
ICU — Level 3 general	219 (36.6%)
Length of stay, days	5 (2–10)
Intubated in ED	73 (12.2%)
Intubated in ICU	282 (47.2%)
7-day mortality	136 (22.7%)
28-day mortality	215 (36.0%)

All continuous variables were non-normally distributed (Shapiro–Wilk $p < 0.05$) and are therefore presented as median (interquartile range). Categorical variables are presented as n (%). One patient may belong to more than one comorbidity category; therefore, comorbidity percentages do not sum to 100%.

Table 2. Univariate comparison of clinical and laboratory characteristics by mortality status

(Full table provided as separate document due to size. Continuous variables compared using the Mann–Whitney U test; categorical variables using Pearson χ^2 or Fisher's exact test as appropriate.)

Variable	7-day surv. (n=462)	7-day non-surv. (n=136)	p	28-day surv. (n=383)	28-day non-surv. (n=215)	p
Age, years	68 (52–79)	70 (56–82)	0.047	66 (46–79)	72 (59–83)	<0.001
Female sex	218 (47.2%)	61 (44.9%)	0.632	183 (47.8%)	96 (44.7%)	0.462
CAD	149 (32.3%)	50 (36.8%)	0.326	111 (29.0%)	88 (40.9%)	0.003
Malignancy	37 (8.0%)	28 (20.6%)	<0.001	27 (7.0%)	38 (17.7%)	<0.001
SBP, mmHg	120 (96–140)	90 (74–120)	<0.001	120 (100–143)	100 (78–130)	<0.001
DBP, mmHg	65 (53–80)	50 (40–65)	<0.001	66 (55–80)	50 (40–68)	<0.001
Heart rate, /min	100 (84–117)	115 (96–130)	<0.001	99 (83–115)	110 (93–127)	<0.001
Lactate, mmol/L	1.9 (1.2–3.3)	3.7 (1.9–7.0)	<0.001	1.9 (1.2–3.1)	2.7 (1.5–5.8)	<0.001
Albumin, g/dL	3.5 (3.1–3.9)	3.2 (2.7–3.7)	<0.001	3.5 (3.1–3.9)	3.2 (2.8–3.7)	<0.001
CRP, mg/L	23.1 (6.0–92.1)	34.5 (12.6–117.3)	0.013	18.6 (5.7–81.8)	44.9 (11.2–129.8)	<0.001
Procalcitonin, ng/mL	0.23 (0.05–1.50)	0.93 (0.19–5.90)	<0.001	0.20 (0.05–1.30)	0.71 (0.15–4.90)	<0.001
SI	0.82 (0.66–1.02)	1.18 (0.91–1.55)	<0.001	0.81 (0.64–0.99)	1.12 (0.88–1.48)	<0.001
DSI	1.50 (1.19–1.93)	2.34 (1.68–3.06)	<0.001	1.45 (1.15–1.84)	2.16 (1.62–2.99)	<0.001
Age SI	49.6 (35.2–73.4)	77.9 (57.3–109.5)	<0.001	47.3 (32.9–69.5)	70.5 (52.1–103.3)	<0.001
MSI	1.16 (0.94–1.50)	1.77 (1.32–2.30)	<0.001	1.13 (0.91–1.45)	1.64 (1.26–2.22)	<0.001
Vasopressor	130 (28.1%)	132 (97.1%)	<0.001	60 (15.7%)	202 (94.0%)	<0.001
Intubated in ICU	149 (32.3%)	133 (97.8%)	<0.001	74 (19.3%)	208 (96.7%)	<0.001

Abbreviated table; full version with all 27 variables available on request. CAD: coronary artery disease; SBP: systolic blood pressure; DBP: diastolic blood pressure; CRP: C-reactive protein; SI: shock index; DSI: diastolic shock index; Age SI: age-weighted shock index; MSI: modified shock index.

Table 3. Diagnostic performance of shock indices and pairwise AUC comparison

3A. Diagnostic operating characteristics

Index	AUC (95% CI)	p	Cut-off	Sens % (95% CI)	Spec % (95% CI)	PPV % (95% CI)	NPV % (95% CI)
7-day mortality							
SI	0.709 (0.656–0.762)	<0.001	1.00	67.6 (59.4–74.9)	68.4 (64.0–72.5)	38.7 (32.7–45.0)	87.8 (84.0–90.8)
DSI	0.720 (0.667–0.772)	<0.001	2.10	58.8 (50.4–66.7)	76.4 (72.3–80.1)	42.3 (35.5–49.5)	86.3 (82.6–89.3)
Age SI	0.715 (0.662–0.768)	<0.001	51.0	80.1 (72.7–86.0)	52.4 (47.8–56.9)	33.1 (28.3–38.4)	90.0 (85.8–93.0)
MSI	0.719 (0.666–0.771)	<0.001	1.44	66.2 (57.9–73.6)	69.0 (64.7–73.1)	38.6 (32.6–45.0)	87.4 (83.6–90.4)
28-day mortality							
SI	0.667 (0.620–0.713)	<0.001	1.00	57.7 (51.0–64.1)	70.2 (65.5–74.6)	52.1 (45.8–58.4)	74.7 (70.0–78.9)
DSI	0.691 (0.646–0.737)	<0.001	2.25	47.9 (41.3–54.6)	86.2 (82.3–89.3)	66.0 (58.3–73.0)	74.7 (70.4–78.5)
Age SI	0.707 (0.663–0.752)	<0.001	51.0	75.3 (69.2–80.6)	56.4 (51.4–61.3)	49.2 (43.9–54.6)	80.3 (75.1–84.6)
MSI	0.683 (0.637–0.728)	<0.001	1.63	51.6 (45.0–58.2)	78.6 (74.2–82.4)	57.5 (50.5–64.3)	74.3 (69.9–78.3)

3B. Pairwise AUC comparison (DeLong method)

Comparison	AUC_1	AUC_2	ΔAUC (95% CI)	Z	p value
7-day mortality					
SI vs DSI	0.709	0.720	−0.011 (−0.030 to +0.008)	−1.13	0.257
SI vs Age SI	0.709	0.715	−0.006 (−0.039 to +0.027)	−0.37	0.714
SI vs MSI	0.709	0.719	−0.010 (−0.020 to +0.001)	−1.83	0.067
DSI vs Age SI	0.720	0.715	+0.005 (−0.029 to +0.038)	+0.27	0.783
DSI vs MSI	0.720	0.719	+0.001 (−0.008 to +0.010)	+0.27	0.787
Age SI vs MSI	0.715	0.719	−0.003 (−0.036 to +0.029)	−0.21	0.833
28-day mortality					
SI vs DSI	0.667	0.691	−0.025 (−0.042 to −0.008)	−2.88	0.004
SI vs Age SI	0.667	0.707	−0.041 (−0.071 to −0.011)	−2.69	0.007
SI vs MSI	0.667	0.683	−0.016 (−0.025 to −0.007)	−3.40	0.001
DSI vs Age SI	0.691	0.707	−0.016 (−0.046 to +0.014)	−1.06	0.289
DSI vs MSI	0.691	0.683	+0.009 (+0.001 to +0.017)	+2.14	0.032
Age SI vs MSI	0.707	0.683	+0.025 (−0.004 to +0.054)	+1.69	0.091

AUC: area under the ROC curve; CI: confidence interval; Sens: sensitivity; Spec: specificity; PPV: positive predictive value; NPV: negative predictive value. Cut-off values determined by the Youden index (sensitivity + specificity − 1) and were derived post-hoc from the same dataset; their generalisability requires confirmation in independent cohorts. Confidence intervals for sensitivity, specificity, PPV, and NPV were computed using the Wilson score method. Confidence intervals for AUC differences (ΔAUC) were obtained using the DeLong et al. paired method (DeLong ER, DeLong DM, Clarke-Pearson DL. Biometrics 1988;44(3):837–845). $\Delta AUC = AUC_1 - AUC_2$. No multiple-comparison correction applied; results should be interpreted with appropriate caution. SI: shock index; DSI: diastolic shock index; Age SI: age-weighted shock index; MSI: modified shock index.

Table 4. Univariate and multivariable logistic regression for 7-day and 28-day mortality

Outcome	Index	Crude OR (95% CI)	Adj OR (95% CI)[a]	Std OR per 1 SD[b]	p	AIC	Nagelkerke R^2	H–L p
7-day mortality	SI	5.75 (3.61–9.16)	3.41 (1.99–5.84)	1.72	<0.001	532.6	0.290	0.136
	DSI	2.43 (1.96–3.02)	1.91 (1.49–2.45)	1.82	<0.001	525.9	0.304	0.408
	Age SI	1.021 (1.016–1.027)	1.018 (1.010–1.026)	1.90	<0.001	531.5	0.292	0.088
	MSI	3.45 (2.53–4.70)	2.44 (1.71–3.48)	1.80	<0.001	528.1	0.299	0.285
28-day mortality	SI	5.01 (3.24–7.73)	3.09 (1.87–5.08)	1.64	<0.001	660.2	0.284	0.506
	DSI	2.52 (2.03–3.12)	2.02 (1.59–2.57)	1.92	<0.001	645.0	0.312	0.667
	Age SI	1.023 (1.017–1.028)	1.016 (1.008–1.023)	1.74	<0.001	661.6	0.282	0.434
	MSI	3.36 (2.50–4.52)	2.45 (1.75–3.43)	1.81	<0.001	651.4	0.300	0.667

[a]*Each shock index was entered as a continuous variable in a separate multivariable model, adjusted for the same seven covariates: age, lactate, albumin, C-reactive protein, procalcitonin, coronary artery disease, and malignancy. Separate models were used because the four indices share heart rate as numerator and overlapping denominators, producing extreme multicollinearity that precluded simultaneous inclusion in a single model (see Methods). n = 598 for all models.*

[b]*Standardised OR represents the change in odds for a one standard deviation increase in the index, allowing scale-independent comparison across indices with different numerical ranges (SI: SD = 0.44; DSI: SD = 0.93; Age SI: SD = 35.53; MSI: SD = 0.66). Full covariate-level estimates for all eight models are provided in Additional File 2. AIC, Akaike Information Criterion (lower values indicate better model fit); H–L, Hosmer–Lemeshow goodness-of-fit test.*

Table 5. Sensitivity analyses (lactate-dropped models and age-stratified subgroups)

5A. Lactate-dropped multivariable models

Outcome	Index	Full model Adj OR (95% CI)	Lactate-dropped Adj OR (95% CI)	OR change	ΔAIC
7-day mortality	SI	3.41 (1.99–5.84)	4.88 (2.98–8.00)	+43.1%	+41.7
	DSI	1.91 (1.49–2.45)	2.25 (1.79–2.83)	+17.5%	+39.6
	Age SI	1.018 (1.010–1.026)	1.023 (1.016–1.031)	+0.5%	+43.6
	MSI	2.44 (1.71–3.48)	3.09 (2.23–4.28)	+26.5%	+39.6
28-day mortality	SI	3.09 (1.87–5.08)	4.17 (2.61–6.65)	+35.2%	+31.8
	DSI	2.02 (1.59–2.57)	2.30 (1.83–2.89)	+14.0%	+27.8
	Age SI	1.016 (1.008–1.023)	1.020 (1.013–1.027)	+0.4%	+34.4
	MSI	2.45 (1.75–3.43)	2.97 (2.16–4.07)	+21.1%	+28.9

ΔAIC = lactate-dropped AIC − full model AIC; positive values indicate worse model fit when lactate is removed. All p values < 0.001 in both full and reduced models.

5B. Age-stratified subgroup analysis

Outcome	Index	Older Adj OR (95% CI)	Younger Adj OR (95% CI)	Older AUC	Younger AUC
7-day mortality	SI	3.41 (1.76–6.61)	3.45 (1.35–8.81)	0.690	0.748
	DSI	1.96 (1.43–2.68)	1.93 (1.28–2.91)	0.701	0.749
	Age SI	1.015 (1.007–1.023)	1.037 (1.020–1.055)	0.679	0.778
	MSI	2.47 (1.59–3.85)	2.50 (1.36–4.58)	0.698	0.754
28-day mortality	SI	3.01 (1.63–5.58)	3.00 (1.24–7.26)	0.661	0.691
	DSI	2.07 (1.53–2.81)	1.90 (1.26–2.85)	0.688	0.699
	Age SI	1.013 (1.006–1.020)	1.030 (1.014–1.047)	0.656	0.762
	MSI	2.47 (1.62–3.76)	2.34 (1.31–4.18)	0.677	0.699

Subgroup models adjusted for the same covariates as the main analysis except age (excluded due to age-stratification). Older subgroup (≥ 65 years; n = 336): 7-day mortality 24.7% (n = 83), 28-day mortality 41.4%

(n = 139). Younger subgroup (< 65 years; n = 262): 7-day mortality 20.2% (n = 53), 28-day mortality 29.0% (n = 76). All p values < 0.05 in both subgroups for all indices.

Figures

Figure 1. Participant flow through the study

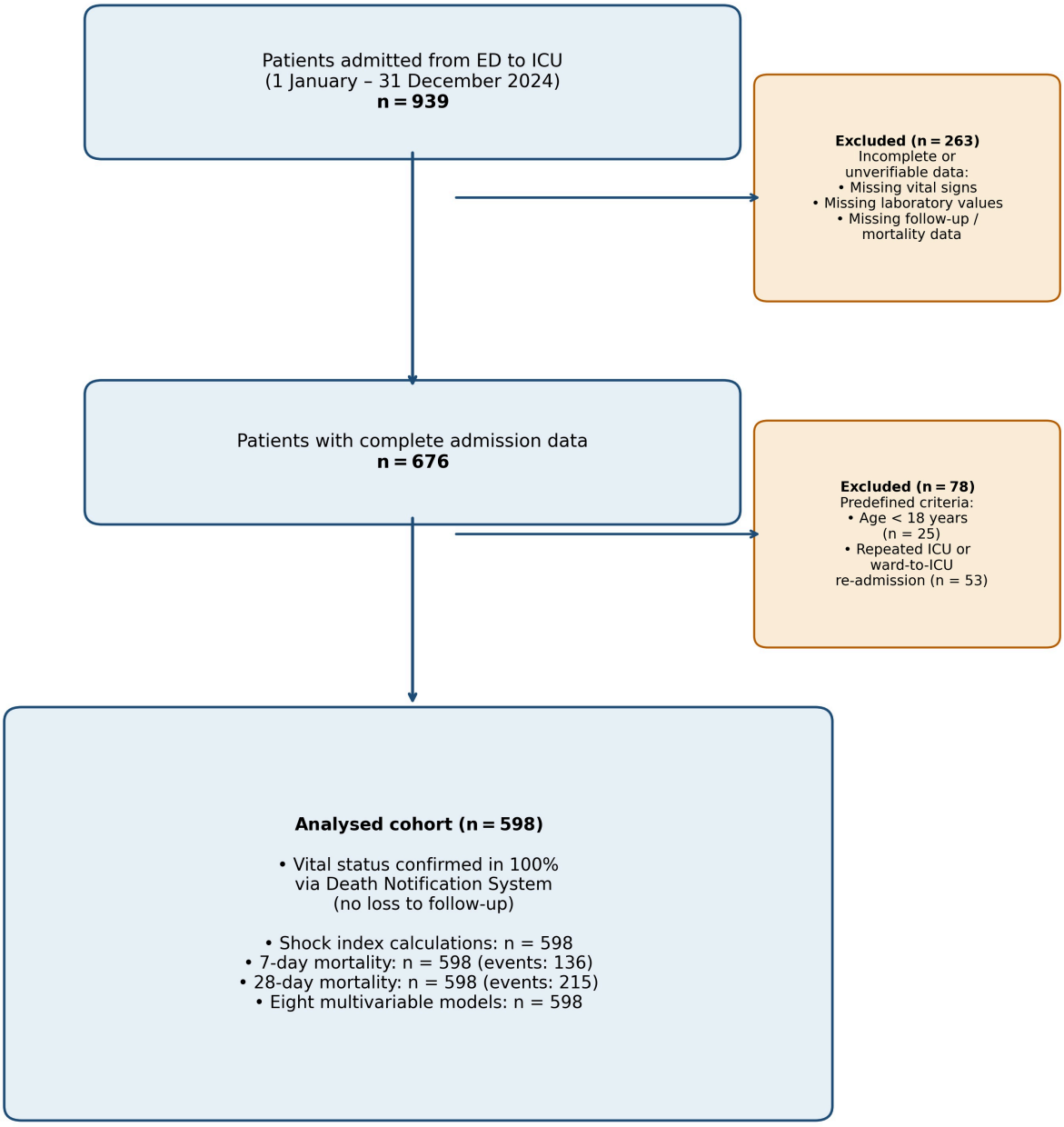


Figure 1

Participant flow through the study. Of 939 adult patients admitted directly from the emergency department to the intensive care unit at Mardin Training and Research Hospital between 1 January and 31 December 2024, 263 were excluded for incomplete or unverifiable admission data and 78 for pre-defined exclusion criteria (age < 18 years, n = 25; repeated/ward-to-ICU re-admissions during follow-up, n = 53), yielding 598 patients in the final analytic cohort. Vital status at 28 days was confirmed in 100% of analysed patients via the national Death Notification System; no patient was lost to follow-up. The same cohort was used for all primary analyses, including shock index calculations, ROC analysis (7-day and 28-day mortality), and eight multivariable logistic regression models.

Figure 2. ROC curves of shock indices for 7-day mortality prediction

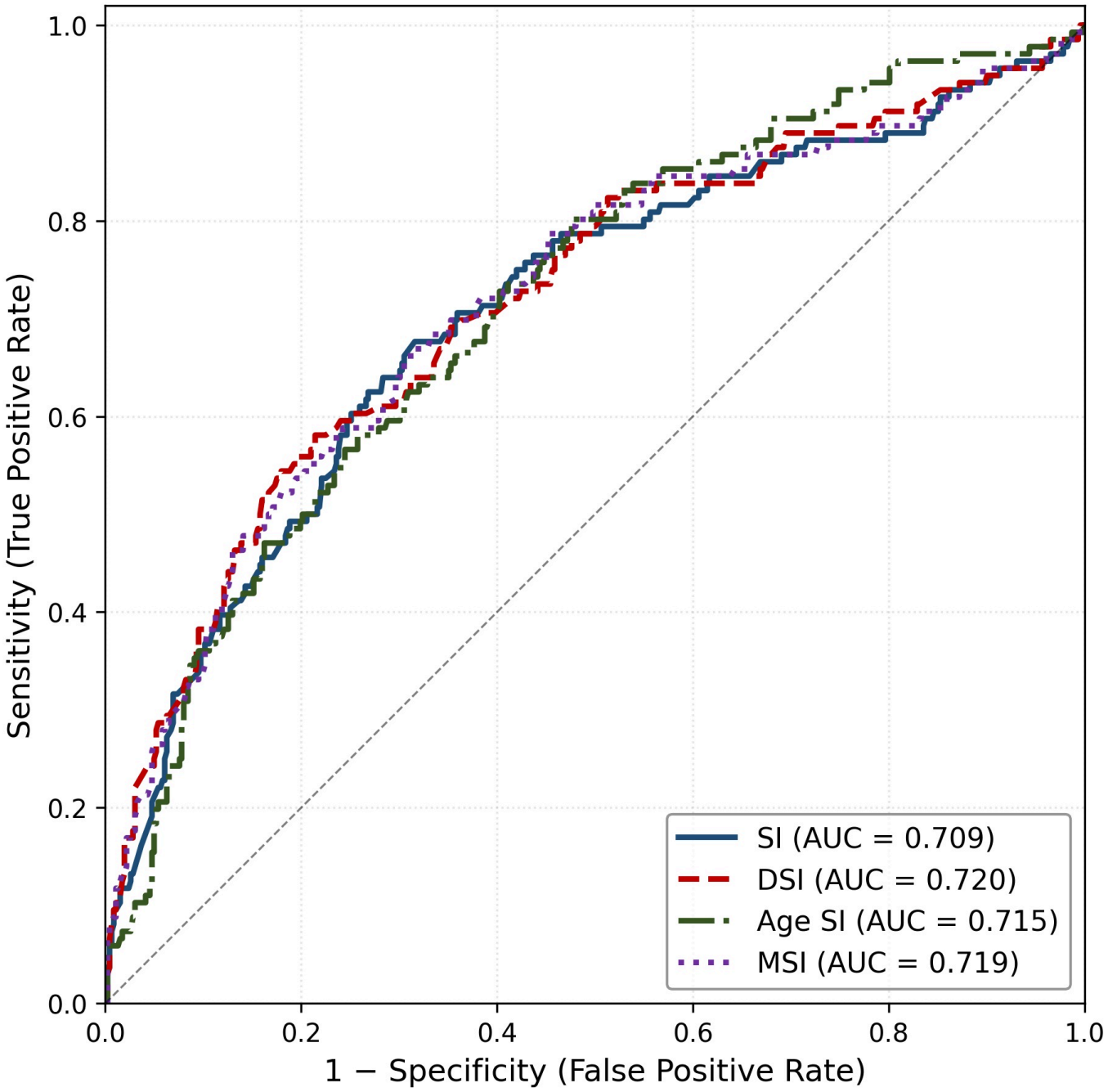


Figure 2

ROC curves of shock indices for 7-day mortality prediction. SI: shock index (heart rate / systolic blood pressure); DSI: diastolic shock index (heart rate / diastolic blood pressure); Age SI: age-weighted shock index (age × SI); MSI: modified shock index (heart rate / mean arterial pressure). The dashed diagonal line represents the reference line of no discrimination (AUC = 0.500).

Figure 3. ROC curves of shock indices for 28-day mortality prediction

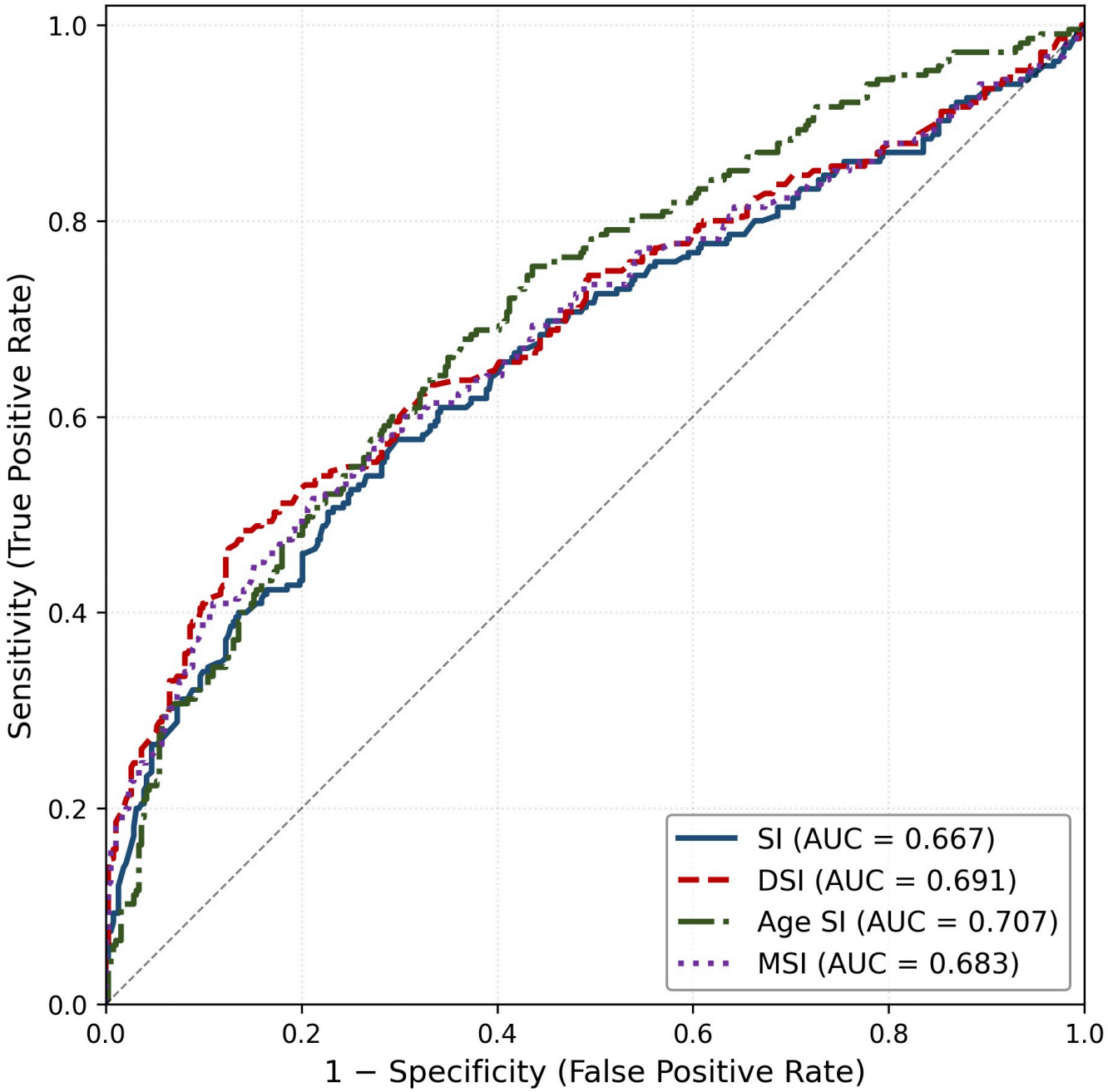


Figure 3

ROC curves of shock indices for 28-day mortality prediction. Abbreviations as in Figure 2. The dashed diagonal line represents the reference line of no discrimination (AUC = 0.500).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- AdditionalFile1STROBE.docx
- AdditionalFile2FullModels.docx