

## ORIGINAL ARTICLE

# Prognostic Accuracy of the Revised Trauma Score, Shock Index, and Injury Severity Score for In-Hospital Mortality in Adult Trauma Patients: A Retrospective Single-Center Cohort Study

Denny Prasetyo<sup>1\*</sup>, Arie Zainul Fatoni<sup>1</sup>, Ristiawan Muji Laksono<sup>1</sup>, Buyung Hartiyo Laksono<sup>1</sup><sup>1</sup> Department of Anaesthesiology and Intensive Therapy, Faculty of Medicine, Universitas Brawijaya / Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

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## Corresponding author

Denny Prasetyo  
[dennyjuan28@gmail.com](mailto:dennyjuan28@gmail.com)

## ABSTRACT

**Background.** Early risk stratification is essential in severe trauma, particularly in resource-limited settings. Physiological scores such as the Revised Trauma Score (RTS) and Shock Index (SI) allow rapid bedside triage, whereas the Injury Severity Score (ISS) quantifies anatomical tissue damage.

**Objective.** To compare the discrimination of RTS, SI, and ISS for in-hospital mortality in severely injured adults.

**Methods.** This retrospective single-center cohort study included 100 adults aged 20–60 years admitted to the Emergency Department of Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia, in 2023. An outcome-enriched sample (50 survivors, 50 non-survivors) was analyzed. The outcome was all-cause in-hospital mortality. Each score was entered into a separate binary logistic regression model; discrimination was assessed with receiver operating characteristic curves and calibration with the Hosmer–Lemeshow test.

**Results.** Each score was significantly associated with mortality ( $p < 0.001$ ). ISS had the highest area under the curve (AUC 0.88, 95% CI 0.81–0.95; odds ratio [OR] 1.15, 95% CI 1.09–1.21), followed by RTS (AUC 0.83, 95% CI 0.75–0.91; OR 0.45, 95% CI 0.32–0.63) and SI (AUC 0.76, 95% CI 0.67–0.85; OR 4.12, 95% CI 2.15–7.89). ISS differed significantly from SI (DeLong  $p = 0.014$ ) but not from RTS ( $p = 0.238$ ). Optimal cut-offs were  $ISS \geq 29$ ,  $RTS \leq 6.95$ , and  $SI \geq 1.05$ . Hosmer–Lemeshow tests were not significant ( $p > 0.05$ ).

**Conclusion.** ISS had the highest AUC, while RTS and SI allow rapid bedside assessment. A staged approach combining immediate physiological scores with later anatomical scoring merits prospective testing in unselected trauma patients.

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## 1. Introduction

Traumatic injury is a major global public health problem. It is a leading cause of premature death and permanent disability. Young, economically productive adults bear the greatest share.<sup>1</sup> Surveillance data from the World Health Organization show that physical trauma kills more than 4.4 million people each year. Of this burden, 90% occurs in low- and middle-income countries (LMICs).<sup>2</sup> The geographic gap is more than a statistical artifact. It reflects structural inequities in health systems: pre-hospital retrieval networks, resuscitation facilities, and emergency surgical capacity are most limited in the regions with the highest incidence of mechanical injury.<sup>3</sup> Indonesia, the fourth most populous country in the world, is part of this burden.<sup>4</sup> Rapid industrialization, expanding highway networks, and fast growth in motorized two-wheeled transport have driven a rise in severe road traffic collisions which, together with industrial hazards and domestic trauma, place a heavy and unprecedented clinical workload on provincial tertiary emergency departments.<sup>4</sup>

For the attending emergency physician, the resuscitating anesthesiologist, and the trauma team leader, the first

period after arrival largely decides the patient's fate.<sup>5</sup> The classic "golden hour" concept holds that the course between physiological recovery and irreversible collapse is mostly set by the speed, diagnostic precision, and therapeutic effectiveness of interventions given in the immediate post-injury window.<sup>5</sup> In this chaotic, high-stakes setting, the basic operational process is clinical triage, the rapid and systematic sorting of casualties so that scarce surgical teams, blood components, operative capacity, and intensive care beds go to patients facing the greatest threat to life.<sup>6,7</sup> Accuracy matters here. An erroneous assessment spreads into failure across the whole system.<sup>7</sup> Undertriage is the failure to recognize the physiological severity or lethal occult hemorrhage of a severely injured casualty, and it causes fatal delays in damage-control surgery, hemostatic resuscitation, or emergent neurosurgical decompression.<sup>6,7</sup> Overtriage is the misallocation of high-tier resuscitation resources to patients with minor, self-limiting injuries. It exhausts limited institutional capacity, disrupts acute emergency workflows, and delays medical access for those facing imminent death.<sup>6,7</sup>

Standardized trauma scoring systems have been developed and refined over the past half-century to protect

triage decisions from subjective cognitive heuristic errors, clinical variability, and emotional stress.<sup>8,9</sup> They give clinicians a shared language in acute trauma care. They turn complex combinations of anatomical disruption and physiological derangement into reproducible, quantitative estimates of mortality risk.<sup>10,11</sup> Existing models rest on two paradigms. These are physiological scoring and anatomical scoring.<sup>8,9</sup> Physiological scores capture the instantaneous functional breakdown of vital homeostatic systems. Anatomical scores quantify the cumulative physical disruption of tissues and internal organs.<sup>8,9,12</sup>

The Revised Trauma Score (RTS), developed by Champion and colleagues, is among the oldest and most widely used physiological instruments in contemporary emergency medicine.<sup>8</sup> It is computed with a weighted logistic equation that combines the Glasgow Coma Scale (GCS), systolic blood pressure (SBP), and respiratory rate (RR), and the result is a single value summarizing central nervous system function, cardiovascular perfusion, and ventilatory mechanics.<sup>8</sup> The RTS depends only on vital signs obtained at stretcher arrival. It needs no laboratory or imaging delay, so it remains a core tool for point-of-care pre-hospital triage and for the emergency department primary survey.<sup>8</sup> A rapidly falling RTS warns of impending multi-system collapse and very high mortality risk.<sup>8</sup>

The Shock Index (SI) is an even simpler bedside physiological metric. Allgöwer and Burri introduced it in 1967 as the ratio of heart rate to systolic blood pressure ( $SI = HR / SBP$ ).<sup>12</sup> In acute traumatic hemorrhage, the cardiovascular system first mounts an intense neuroendocrine sympathetic response, and baroreceptor-mediated tachycardia and peripheral vasoconstriction keep systolic blood pressure deceptively normal despite a large loss of circulating intravascular volume.<sup>13,14</sup> The Shock Index can therefore flag compensated occult shock well before frank hypotension appears.<sup>13,15</sup> An elevated value exposes hidden hypoperfusion. It signals an impending cardiovascular crash, prompting urgent hemostatic resuscitation, blood typing, and rapid surgical control of bleeding.<sup>13,16</sup>

In contrast to these dynamic physiological indices, the historical gold standard for quantifying the structural insult of trauma is the Injury Severity Score (ISS), formulated by Baker and colleagues from the Abbreviated Injury Scale (AIS).<sup>9</sup> The ISS sums the squares of the highest AIS severity grades in the three most severely injured body regions. It yields an integer from 1 to 75 that expresses the patient's total anatomical trauma burden.<sup>9</sup> An ISS above 15 denotes major trauma. A score of 25 or greater signifies severe injury with a high probability of in-hospital death.<sup>17</sup> The Trauma and Injury Severity Score (TRISS) method later built on the strengths of both approaches, combining RTS, ISS, patient age, and injury mechanism into a hybrid prognostic algorithm.<sup>10</sup>

The anatomical ISS has strong predictive power for late trauma mortality, multiple organ dysfunction, and intensive care utilization. Its main limitation is operational. The definitive score can be calculated only in retrospect, after comprehensive whole-body computed tomography (CT), specialized radiologic interpretation, or surgical exploration.<sup>9,18</sup> Validation studies in diverse healthcare systems have therefore repeatedly compared the predictive performance of physiological and anatomical systems,<sup>11,19</sup> and recent meta-analyses and multicenter investigations show that the ISS gives good discrimination for overall mortality across broad cohorts, while bedside

physiological metrics such as RTS and SI offer speed of diagnosis during the immediate resuscitation window.<sup>13,20,21</sup> Emerging evidence indicates that the prehospital and early emergency department Shock Index predicts both 24-hour mortality and the need for massive transfusion, even when initial blood pressure appears normotensive.<sup>22,23</sup> Changes in physiological metrics over time, such as the delta Shock Index, also give useful information on responsiveness to resuscitation and on trajectory.<sup>24</sup>

Most existing validation trials of scoring systems were conducted in well-organized, well-funded trauma systems in high-income nations, which feature rapid pre-hospital helicopter transport, dedicated trauma centers, and immediate universal pan-scan CT imaging.<sup>21</sup> Direct extrapolation to low- and middle-income countries such as Indonesia is uncertain.<sup>4,21</sup> In Indonesian provincial settings, trauma management is shaped by long transit intervals. Civilian vehicles carry patients without monitoring, and the availability of immediate advanced imaging and blood products varies.<sup>4,25</sup> These realities could change the temporal course of acute trauma pathology and could also shift the discriminative boundaries and calibration of scoring tools developed in high-income environments.<sup>5,21</sup>

This investigation targets adult trauma patients with extreme injury severity at a major Indonesian tertiary referral center. The study used an outcome-enriched cohort of severely injured patients. Enrichment prevents discrimination estimates from being diluted by minor injuries, but it changes outcome prevalence, so predictive values and calibration cannot be transferred directly to unselected patients.<sup>26</sup> The primary aim was therefore to evaluate and compare the predictive performance, discriminative accuracy, and model calibration of the Revised Trauma Score, Shock Index, and Injury Severity Score for in-hospital mortality in a high-mortality cohort of severely injured adult trauma patients admitted to the Emergency Department of Dr. Saiful Anwar Regional General Hospital in Malang, Indonesia.

Trauma care delivery in Southeast Asia and archipelagic Indonesia is shaped by geographical and logistical challenges that differ sharply from those of integrated high-income trauma networks.<sup>4,25</sup> In urban and peri-urban Indonesian centers, the pre-hospital environment has no unified emergency dispatch system. Transit times are long on congested road corridors. Severely injured casualties are often carried by non-medical civilian bystanders.<sup>4,25</sup> Patients therefore frequently reach provincial tertiary referral emergency departments with advanced, unmitigated physiological decompensation, and the classic “golden hour” has already elapsed before formal medical care begins.<sup>5,25</sup> Under such strained conditions, point-of-care scoring systems must work as more than academic classification tools and need to serve as dependable clinical decision rules that quickly identify occult circulatory failure and direct aggressive resuscitation without waiting for laboratory results.<sup>27,28</sup>

Physiological masking is an under-recognized challenge in contemporary trauma triage. Young, previously healthy adults may sustain massive internal injuries. Their vital signs can nevertheless stay deceptively normal in the early post-impact period.<sup>14</sup> In adults aged 20 to 60 years, good cardiovascular physiological reserve and intense endogenous sympathetic neurohumoral responses can preserve blood pressure despite an acute loss of 20% to

30% of circulating blood volume.<sup>14</sup> When clinicians rely only on conventional vital sign thresholds, such as systolic blood pressure < 90 mmHg, these patients are often undertriaged, which leads to missed resuscitation windows and avoidable mortality.<sup>16,29</sup> Evaluating dynamic bedside indices such as the Shock Index together with composite tools such as the Revised Trauma Score gives an objective safeguard. It helps unmask occult physiological failure before catastrophic decompensation develops.<sup>13,30</sup>

## 2. Methods

### 2.1. Study design and reporting

This was a single-center retrospective observational cohort study of adult trauma patients admitted to the Emergency Department (ED) of Dr. Saiful Anwar Regional General Hospital in Malang, East Java, Indonesia. Reporting followed international epidemiological guidelines for observational prognostic research and the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) statement.<sup>31</sup> The TRIPOD statement<sup>32</sup> is also relevant to a score-comparison study.

### 2.2. Setting

The hospital is a provincial tertiary referral center and the primary teaching hospital of the Faculty of Medicine, Universitas Brawijaya, and it is also the designated Level I-equivalent trauma receiving facility for the southern and eastern regions of East Java and serves a catchment population exceeding fifteen million people. Its emergency resuscitation bay handles a very high volume of complex blunt polytrauma, industrial crush injuries, and severe burn casualties.<sup>4</sup>

### 2.3. Participants and eligibility

Patient data were retrospectively extracted from hospital medical records for trauma admissions during a three-month study interval, from September 1 to November 30, 2023. In this period, 312 trauma patients were evaluated in the emergency resuscitation bay. A purposive sampling strategy was used to build an outcome-enriched cohort of exactly 100 adult trauma patients, comprising 50 in-hospital survivors (50.0%) and 50 non-survivors (50.0%). Outcome-enriched sampling can be used to estimate discrimination at the extreme end of trauma severity, but outcome prevalence is set by the design, so predictive values and calibration do not transfer to unselected populations.<sup>26</sup> Recent trauma prediction literature has developed new point-of-care metrics, such as the Reverse Shock Index multiplied by Glasgow Coma Scale (rSIG) score, to improve early mortality prediction, and this keeps benchmarking of traditional scoring instruments against high-acuity cohorts necessary.<sup>27,28</sup>

Patient records were selected using predefined, standardized inclusion and exclusion criteria. Inclusion criteria were as follows. Patients were adults aged 20 to 60 years inclusive. This age range was chosen to build a physiologically homogenous non-geriatric sample and to limit confounding from pediatric physiology or geriatric homeostenosis. They had a primary diagnosis of acute physical trauma from blunt mechanical impacts, motor vehicle crashes, falls from height, penetrating mechanisms, or thermal burns. They presented to the emergency resuscitation bay within 24 hours of injury. Their records contained complete documentation of initial vital signs, diagnostic imaging reports, operative records, and definitive in-hospital survival outcome. Patients were

excluded if they were declared dead on arrival (DOA) at emergency triage (n = 18). Patients outside the age range of 20 to 60 years (n = 94, as in the study flowchart) were also excluded. Individuals with incomplete or missing physiological vital signs or radiographic records (n = 42) were also excluded, as were patients transferred to external healthcare facilities before the definitive outcome was determined (n = 22), and so were patients with pre-existing chronic systemic comorbidities, defined operationally as a Charlson Comorbidity Index (CCI) score of 3 or greater (n = 36). It was meant to prevent pre-existing end-stage medical pathologies from acting as unmeasured confounders for trauma-related mortality.

### 2.4. Sample size

A sample-size calculation is not reported. The outcome-enriched sampling was intended to give adequate statistical power to analyze determinants of the fatal outcome without the dilution typical of unselected cohorts.

### 2.5. Variables and operational definitions

The primary outcome was all-cause in-hospital mortality. It was recorded as a binary dependent variable: “Survived” (patient discharged alive after successful clinical stabilization) or “Deceased” (patient death at any point during the index trauma hospitalization).

Demographic variables were age, sex, mechanism of injury, and pre-hospital transit duration in minutes. Initial physiological parameters came from the earliest documented set of vital signs taken in the resuscitation bay by attending emergency physicians or triage nurses, including Glasgow Coma Scale (GCS) score, systolic blood pressure (SBP in mmHg), diastolic blood pressure (DBP in mmHg), heart rate (HR in beats per minute), and respiratory rate (RR in breaths per minute).<sup>34</sup> These vital signs reflect the early clinical manifestations of systemic cellular hypoperfusion, endotheliopathy of trauma, and microvascular barrier compromise that characterize severe physical injury.<sup>35,36</sup>

The physiological and anatomical trauma scoring systems were computed according to international methodological standards.<sup>8,9</sup> The Revised Trauma Score (RTS) was calculated with the canonical weighted logistic equation:  $RTS = (0.9368 \times GCS\_code) + (0.7326 \times SBP\_code) + (0.2908 \times RR\_code)$ .<sup>8</sup> Each physiological variable received a coded score from 0 to 4 under predefined clinical boundaries. GCS scores of 13–15 were coded as 4, 9–12 as 3, 6–8 as 2, 4–5 as 1, and 3 as 0; SBP values >89 mmHg were coded as 4, 76–89 as 3, 50–75 as 2, 1–49 as 1, and 0 as 0; and RR values of 10–29 breaths/min were coded as 4, >29 as 3, 6–9 as 2, 1–5 as 1, and 0 as 0.<sup>8</sup> The resulting continuous RTS ranges from 0 (physiological death) to 7.84 (normal physiological baseline).<sup>8</sup> The Shock Index (SI) used the same initial set of vital signs. It was calculated with the standard formula:  $SI = \text{Heart Rate (beats/min)} / \text{Systolic Blood Pressure (mmHg)}$ .<sup>12</sup> A calculated Shock Index value <0.7 was categorized as normal, 0.7 to 0.9 as borderline, and >0.9 as overt hemodynamic shock. These categories reflect increasing risk of tissue hypoperfusion.<sup>13</sup>

The Injury Severity Score (ISS) was calculated retrospectively. The calculation drew on all emergency imaging examinations (plain radiography, ultrasonography / FAST, and multidetector computed tomography), operative surgical protocols, forensic autopsy summaries, and clinical progress notes from the index hospitalization.<sup>9</sup> Each anatomical lesion was

classified and coded with the Abbreviated Injury Scale (AIS 2005, update 2008) across six defined body regions: Head and Neck, Face, Chest, Abdomen and Pelvic Contents, Extremities and Pelvic Girdle, and External / Burns.<sup>9</sup> AIS severity grades run from 1 (minor) to 6 (currently untreatable / fatal).<sup>9</sup> The definitive ISS was derived by summing the squares of the highest AIS severity scores from the three most severely injured anatomical regions, giving a score between 1 and 75,<sup>9</sup> and any patient with an AIS grade of 6 was assigned an automatic ISS of 75.<sup>9</sup> Massive anatomical disruption, quantified by a high ISS, triggers release of damage-associated molecular patterns (DAMPs), systemic inflammation, and cellular organ dysfunction.<sup>37,38</sup> In severe neurotrauma, concurrent loss of cerebral autoregulation further raises mortality risk.<sup>39</sup>

## 2.6. Data sources and measurement

Data were abstracted with a standardized, pre-piloted electronic data collection template developed for this protocol. To limit transcription error, one dedicated clinical investigator trained in trauma registry protocols performed the primary extraction from archived electronic medical records and paper nursing charts. All data abstraction followed predefined standard operating protocols. The emergency electronic medical records and paper casualty charts were reviewed in duplicate for key physiological parameters. Reviewers sought the earliest set of vital signs. These had to be recorded within 15 minutes of emergency department presentation. To remove possible treatment-induced confounding, vital signs documented after endotracheal intubation, intravenous fluid boluses exceeding 1,000 mL, or vasopressor infusions had begun were excluded from the baseline scoring calculations.

Retrospective AIS coding and ISS derivation can vary between observers. A quality control audit was therefore built into the study design.<sup>40</sup> An independent certified trauma specialist, blinded to patient clinical outcomes and to the initial ratings, re-scored a randomly selected 20% validation subset of the cohort ( $n = 20$ ). Inter-rater reliability was evaluated with Cohen's kappa statistic, which gave  $\kappa = 0.84$  (standard error 0.082,  $p < 0.001$ ). This indicates almost perfect agreement between the two coders and supports the integrity of the anatomical score data.<sup>41</sup>

Anatomical injury coding for the Abbreviated Injury Scale and Injury Severity Score followed a defined procedure.<sup>9,40</sup> Certified trauma abstractors reviewed all high-resolution multidetector computed tomography scans, operative exploration logs, orthopedic fixation records, and forensic pathology summaries. Each injury diagnosis was matched verbatim to its six-digit AIS dictionary descriptor (AIS 2005, update 2008).<sup>9</sup> When a patient had several distinct anatomical lesions within one body region, only the single highest AIS score from that region was extracted for the ISS calculation, in strict compliance with the foundational Baker methodology.<sup>9</sup> Following international trauma registry standards, an AIS grade of 6 was assigned to unsurvivable catastrophic anatomical disruptions. Examples include massive brainstem transection, complete aortic avulsion, or extensive thoracic crush injury. Such injuries generated an automatic ISS of 75.<sup>9</sup>

## 2.7. Statistical analysis

Statistical analyses were run in IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA) and verified with custom statistical pipelines in Python (Python

Software Foundation, Beaverton, OR, USA). Normality of continuous variables was first assessed with the Shapiro–Wilk and Kolmogorov–Smirnov tests. Normally distributed continuous variables were summarized as mean  $\pm$  standard deviation (SD) and compared between survival groups with independent-samples Student's *t*-tests; non-normally distributed continuous variables were expressed as median [interquartile range, IQR] and analyzed with the non-parametric Mann–Whitney *U* test. Categorical variables were tabulated as frequencies and percentages (summing to 100.0%) and compared with Pearson chi-square tests or Fisher's exact tests as appropriate.

To evaluate predictive performance while avoiding severe multicollinearity and mathematical instability, the three scoring metrics were entered into separate univariable binary logistic regression models, with in-hospital mortality as the dependent outcome. Effect sizes were reported as unstandardized regression coefficients (B), standard errors (SE), Wald chi-square statistics, and odds ratios (OR) with associated 95% confidence intervals (95% CI). Discriminative ability was quantified as the area under the receiver operating characteristic curve (AUC-ROC), with non-parametric 95% confidence intervals and standard errors.<sup>42</sup> Optimal prognostic cutoff thresholds for predicting mortality were identified with the maximum Youden Index ( $J = \text{Sensitivity} + \text{Specificity} - 1$ ), alongside sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and overall diagnostic accuracy.<sup>43</sup> Calibration was assessed across risk deciles with the Hosmer–Lemeshow goodness-of-fit test. A non-significant *p* value ( $p > 0.05$ ) indicates no detectable disagreement between predicted and observed mortality probabilities.<sup>44</sup> All statistical tests were two-tailed. An alpha threshold of  $p < 0.05$  was set a priori as the criterion for statistical significance.

Secondary diagnostic performance characteristics were computed across predefined severity strata. The purpose was to evaluate the clinical discrimination of the three scoring models in different clinical settings. Beyond sensitivity and specificity, the positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (+LR), and negative likelihood ratio (-LR) were calculated for each optimal cutoff threshold. Calibration curves were drawn by plotting the mean predicted mortality probabilities across ten risk deciles against the observed in-hospital mortality rates. Goodness of fit was evaluated with the Hosmer–Lemeshow chi-square test.<sup>44,45</sup> The three correlated ROC curves (same patients) were also compared with the non-parametric method of DeLong and colleagues. The comparison was meant to establish formal statistical differences between the respective AUC-ROC values.<sup>46</sup>

## 2.8. Ethics

The Ethics Committee CMHC Research Center Indonesia reviewed the study protocol and granted full ethical approval. The Approval Certificate is No. 2023/400. The committee formally waived individual written informed consent. This followed the Declaration of Helsinki and international guidelines for retrospective research involving de-identified health data.

### 3. Results

#### 3.1. Participant flow and baseline characteristics

Over the three-month study interval, 312 trauma patients were evaluated in the resuscitation unit of Dr. Saiful Anwar Regional General Hospital. Strict eligibility criteria and purposive selection were applied to build an outcome-balanced high-acuity cohort. The final analytical dataset held 100 adult trauma patients, exactly 50 survivors (50.0%) and 50 non-survivors (50.0%). Figure 1A illustrates the enrollment and stratification workflow, and

Table 1 details baseline demographic and clinical variables by survival status. Men predominated, numbering 64 (64.0%) against 36 women (36.0%). The overall male-to-female ratio was 1.78:1.<sup>47</sup> Sex distribution did not differ significantly between survivors (31 males, 62.0%) and non-survivors (33 males, 66.0%;  $\chi^2 = 0.174$ ,  $df = 1$ ,  $p = 0.677$ ). The overall mean age was  $43.8 \pm 11.2$  years (range 20 to 60 years). Non-survivors were slightly older than survivors, although the difference was not significant ( $45.5 \pm 11.4$  years vs.  $42.1 \pm 10.8$  years; Student's  $t = -1.53$ ,  $df = 98$ ,  $p = 0.129$ ).

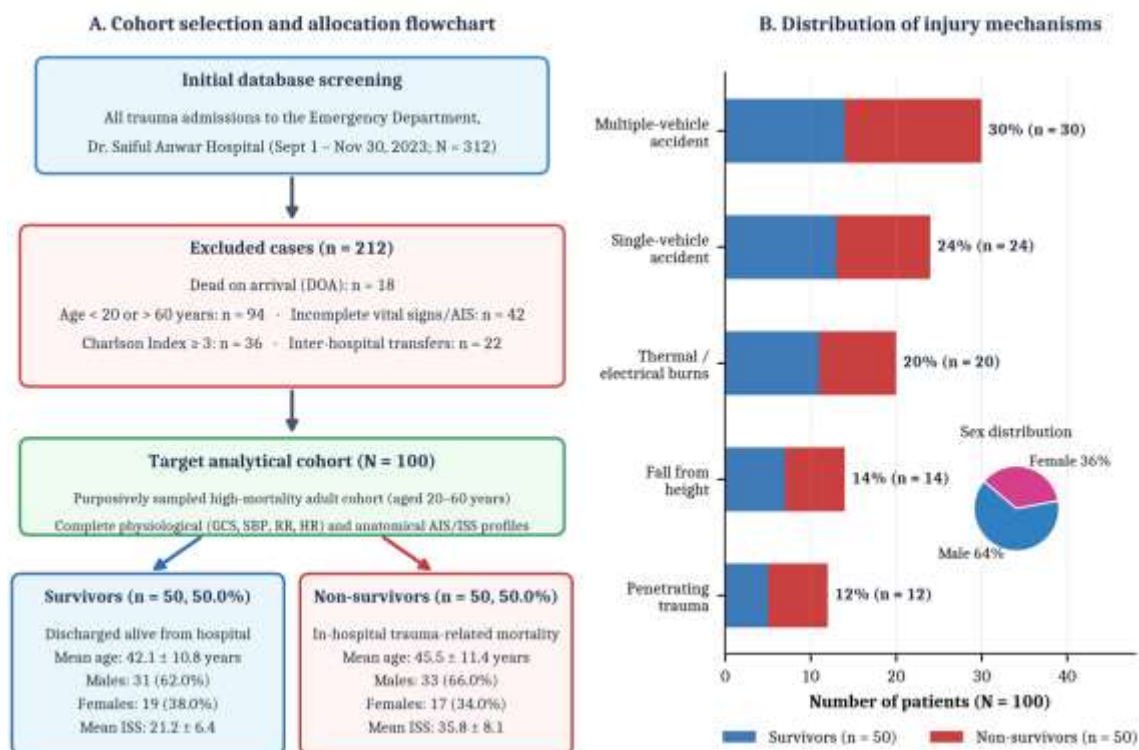


**Figure 1.** Study population selection and distribution of injury mechanisms (N = 100). (A) Flowchart of participant screening and outcome stratification (50 survivors and 50 non-survivors). (B) Frequency distribution of injury mechanisms by survival status.

**Table 1.** Baseline demographics, clinical characteristics, and injury mechanisms by in-hospital survival status (N = 100).

| Characteristic / Variable                       | Total Cohort (N = 100) | Survivors (n = 50) | Non-Survivors (n = 50) | p-value* |
|-------------------------------------------------|------------------------|--------------------|------------------------|----------|
| Age (years), Mean $\pm$ SD                      | $43.8 \pm 11.2$        | $42.1 \pm 10.8$    | $45.5 \pm 11.4$        | 0.129†   |
| Age Range (min – max)                           | 20 – 60                | 20 – 59            | 21 – 60                | —        |
| <b>Sex, n (%)</b>                               |                        |                    |                        |          |
| Male                                            | 64 (64.0%)             | 31 (62.0%)         | 33 (66.0%)             | 0.677‡   |
| Female                                          | 36 (36.0%)             | 19 (38.0%)         | 17 (34.0%)             | —        |
| <b>Mechanism of Injury, n (%)</b>               |                        |                    |                        | 0.934§   |
| Multiple-Vehicle Accident                       | 30 (30.0%)             | 14 (28.0%)         | 16 (32.0%)             | —        |
| Single-Vehicle Accident                         | 24 (24.0%)             | 13 (26.0%)         | 11 (22.0%)             | —        |
| Thermal / Electrical Burns                      | 20 (20.0%)             | 11 (22.0%)         | 9 (18.0%)              | —        |
| Fall from Height                                | 14 (14.0%)             | 7 (14.0%)          | 7 (14.0%)              | —        |
| Penetrating Injury                              | 12 (12.0%)             | 5 (10.0%)          | 7 (14.0%)              | —        |
| Pre-hospital Transport Time (min), Median [IQR] | 75.0 [50.0–110.0]      | 70.0 [45.0–100.0]  | 80.0 [55.0–120.0]      | 0.184    |
| Emergency Department GCS, Median [IQR]          | 11.0 [6.0–14.0]        | 14.0 [12.0–15.0]   | 7.0 [4.0–10.0]         | <0.001   |
| Systolic Blood Pressure (mmHg), Mean $\pm$ SD   | $102.4 \pm 24.6$       | $118.2 \pm 16.4$   | $86.6 \pm 21.8$        | <0.001†  |
| Heart Rate (beats/min), Mean $\pm$ SD           | $106.8 \pm 19.5$       | $96.4 \pm 14.2$    | $117.2 \pm 18.9$       | <0.001†  |
| Respiratory Rate (breaths/min), Mean $\pm$ SD   | $23.6 \pm 5.8$         | $20.8 \pm 3.4$     | $26.4 \pm 6.3$         | <0.001†  |

**Notes:** Values are presented as mean  $\pm$  standard deviation (SD), median [interquartile range, IQR], or frequency (percentage, %). Categorical distributions sum to exactly 100.0%.  
†Independent-samples Student's  $t$ -test. ‡Pearson chi-square test ( $\chi^2 = 0.174$ ,  $df = 1$ ). §Pearson chi-square test across 5 injury mechanisms ( $\chi^2 = 0.83$ ,  $df = 4$ ). ||Mann-Whitney U test. GCS: Glasgow Coma Scale; IQR: Interquartile range; SD: Standard deviation. Student's  $t$  statistics in the text are survivors minus non-survivors.

Motor vehicle collisions were the leading cause of injury (Figure 1B and Table 1). They accounted for more than half of admissions (54.0%,  $n = 54$ ). Multiple-vehicle collisions made up 30.0% ( $n = 30$ ) of the cohort and single-vehicle crashes 24.0% ( $n = 24$ ). The remaining major etiologies were thermal and electrical burns (20.0%,  $n = 20$ ), falls from height (14.0%,  $n = 14$ ), and penetrating injuries from sharp weapons or projectiles (12.0%,  $n = 12$ ). Injury mechanisms were distributed similarly in survivors and non-survivors (Pearson  $\chi^2 = 0.83$ ,  $df = 4$ ,  $p = 0.934$ ). Median pre-hospital transport time was 75.0 minutes [IQR 50.0–110.0 minutes], reflecting systemic transit delays typical of developing healthcare networks. Non-survivors had numerically longer transit times than survivors (80.0 [55.0–120.0] vs. 70.0 [45.0–100.0] minutes; Mann-Whitney  $U$ ,  $p = 0.184$ ). This is in line with regional transport realities described across Southeast Asian trauma systems.<sup>25</sup>

In the emergency department, initial physiological parameters were clearly deranged in non-survivors compared with survivors (Table 1). Initial GCS scores were significantly lower in non-survivors (median 7.0 [IQR 4.0–10.0] vs. 14.0 [IQR 12.0–15.0]; Mann-Whitney  $U$ ,  $p < 0.001$ ), a pattern that may reflect severe traumatic brain injury or cerebral hypoperfusion (neither was measured directly).<sup>48</sup> Their initial systolic blood pressure was lower ( $86.6 \pm 21.8$  vs.  $118.2 \pm 16.4$  mmHg; Student's  $t = 8.16$ ,  $p < 0.001$ ) and their heart rate was significantly higher ( $117.2 \pm 18.9$  vs.  $96.4 \pm 14.2$  beats/min; Student's  $t = -6.23$ ,  $p <$

0.001), consistent with decompensated circulatory shock (lactate was not measured).<sup>13</sup> Respiratory rate was also significantly higher among non-survivors ( $26.4 \pm 6.3$  vs.  $20.8 \pm 3.4$  breaths/min; Student's  $t = -5.51$ ,  $p < 0.001$ ). This may reflect respiratory compensation, although blood gases and lactate were not measured.

### 3.2. Primary outcome

Comparison of the initial trauma scores showed clear differences between survival groups on all three instruments (Table 2 and Figure 2, with RTS distributions in Figure 2A, SI distributions in Figure 2B, and ISS distributions in Figure 2C). The overall mean Revised Trauma Score was  $6.8 \pm 2.1$  (median 7.84 [IQR 6.38–7.84], range 1.16 to 7.84). Survivors had significantly higher RTS values than non-survivors (mean  $7.64 \pm 0.38$ , median 7.84 [IQR 7.55–7.84] vs. mean  $5.96 \pm 2.68$ , median 6.38 [IQR 3.80–7.84]; Student's  $t = 4.38$ ,  $df = 98$ ,  $p < 0.001$ ; Mann-Whitney  $U = 422.5$ ,  $Z = -5.48$ ,  $p < 0.001$ ; Figure 2A). The Shock Index averaged  $1.1 \pm 0.4$  across the entire cohort (median 1.02 [IQR 0.81–1.35], range 0.50 to 2.10). Mean SI was significantly higher in non-survivors, at  $1.36 \pm 0.38$  (median 1.33 [IQR 1.08–1.62]), than in survivors, at  $0.84 \pm 0.19$  (median 0.81 [IQR 0.69–0.94]; Student's  $t = -8.67$ ,  $df = 98$ ,  $p < 0.001$ ; Mann-Whitney  $U = 458.0$ ,  $Z = -5.19$ ,  $p < 0.001$ ; Figure 2B). This supports severe occult or overt hemodynamic instability in the fatal cohort. It also agrees with recent large-scale registry observations comparing SI against isolated blood pressure.<sup>49,50</sup>

**Table 2.** Initial trauma scores (RTS, SI, ISS) at emergency department admission by in-hospital survival status.

| Trauma Scoring System               | Total Cohort (N = 100) | Survivors (n = 50) | Non-Survivors (n = 50) | Test Statistic   | p-value*          |
|-------------------------------------|------------------------|--------------------|------------------------|------------------|-------------------|
| <b>Revised Trauma Score (RTS)</b>   |                        |                    |                        |                  |                   |
| Mean $\pm$ SD                       | $6.8 \pm 2.1$          | $7.64 \pm 0.38$    | $5.96 \pm 2.68$        | $t = 4.38$       | $<0.001^\dagger$  |
| Median [IQR]                        | 7.84 [6.38–7.84]       | 7.84 [7.55–7.84]   | 6.38 [3.80–7.84]       | $Z = -5.48$      | $<0.001^\ddagger$ |
| Observed Range (min – max)          | 1.16 – 7.84            | 6.38 – 7.84        | 1.16 – 7.84            | —                | —                 |
| <b>Shock Index (SI = HR / SBP)</b>  |                        |                    |                        |                  |                   |
| Mean $\pm$ SD                       | $1.1 \pm 0.4$          | $0.84 \pm 0.19$    | $1.36 \pm 0.38$        | $t = -8.67$      | $<0.001^\dagger$  |
| Median [IQR]                        | 1.02 [0.81–1.35]       | 0.81 [0.69–0.94]   | 1.33 [1.08–1.62]       | $Z = -5.19$      | $<0.001^\ddagger$ |
| Observed Range (min – max)          | 0.50 – 2.10            | 0.50 – 1.28        | 0.72 – 2.10            | —                | —                 |
| <b>Injury Severity Score (ISS)</b>  |                        |                    |                        |                  |                   |
| Mean $\pm$ SD                       | $28.5 \pm 10.3$        | $21.2 \pm 6.4$     | $35.8 \pm 8.1$         | $t = -9.98$      | $<0.001^\dagger$  |
| Median [IQR]                        | 27.5 [19.0–36.0]       | 20.5 [16.0–26.0]   | 36.0 [29.0–41.0]       | $Z = -6.74$      | $<0.001^\ddagger$ |
| Observed Range (min – max)          | 9 – 50                 | 9 – 34             | 18 – 50                | —                | —                 |
| <b>Major Trauma Category, n (%)</b> |                        |                    |                        |                  |                   |
| ISS < 16 (Moderate)                 | 12 (12.0%)             | 12 (24.0%)         | 0 (0.0%)               | $\chi^2 = 38.98$ | $<0.001^\S$       |
| ISS 16 – 24 (Severe)                | 26 (26.0%)             | 22 (44.0%)         | 4 (8.0%)               | —                | —                 |
| ISS $\geq 25$ (Critical / Profound) | 62 (62.0%)             | 16 (32.0%)         | 46 (92.0%)             | —                | —                 |

**Notes:** Statistically significant at  $\alpha = 0.05$ .  $^\dagger$ Independent-samples Student's  $t$ -test ( $df = 98$ ).  $^\ddagger$ Mann-Whitney  $U$  non-parametric test.  $^\S$ Pearson chi-square test across severity categories ( $df = 2$ ). RTS: Revised Trauma Score (weighted equation); SI: Shock Index (Heart Rate / Systolic Blood Pressure); ISS: Injury Severity Score (sum of squares of highest AIS in three body regions).

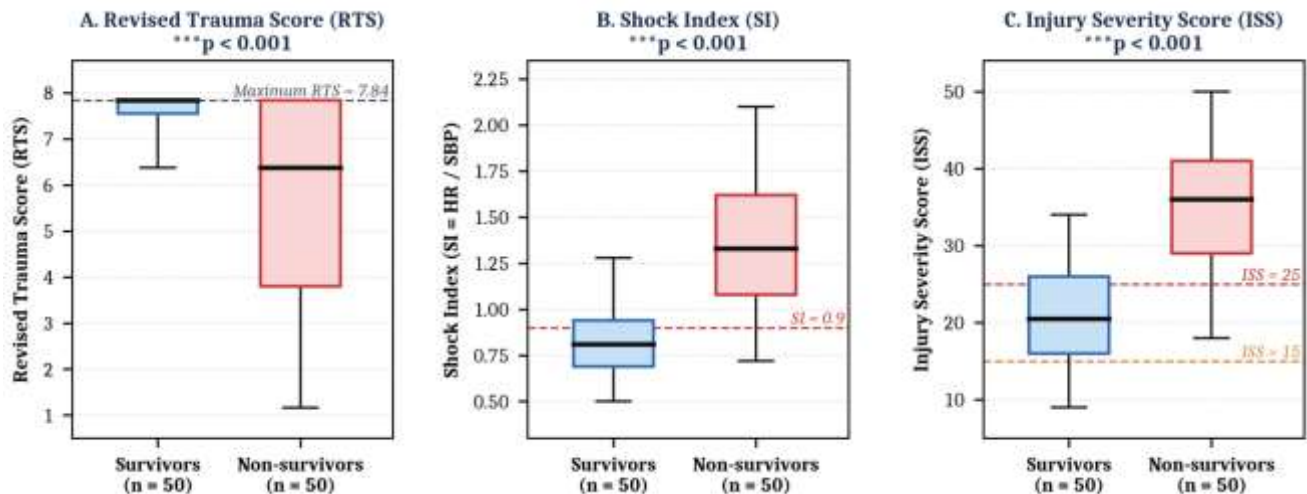


**Figure 2.** Initial trauma scores by in-hospital survival: (A) Revised Trauma Score (RTS), (B) Shock Index (SI), and (C) Injury Severity Score (ISS). Boxes show medians and interquartile ranges; whiskers show the observed range (Table 2). Dashed lines mark reference thresholds. \*\*\*p < 0.001.

The anatomical injury burden, measured by the Injury Severity Score, was considerably higher in non-survivors (Table 2, Figure 2C). The overall cohort mean ISS was  $28.5 \pm 10.3$  (median 27.5 [IQR 19.0–36.0], range 9 to 50), which confirms the severe, high-acuity nature of the purposively selected study population. Among non-survivors the mean ISS was  $35.8 \pm 8.1$  (median 36.0 [IQR 29.0–41.0], range 18 to 50), reflecting a high anatomical injury burden. Survivors had a significantly lower mean ISS,  $21.2 \pm 6.4$  (median 20.5 [IQR 16.0–26.0], range 9 to 34; Student's  $t = -9.98$ ,  $df = 98$ ,  $p < 0.001$ ; Mann-Whitney  $U = 265.0$ ,  $Z = -6.74$ ,  $p < 0.001$ ). Using conventional trauma severity thresholds, 92.0% of non-survivors ( $n = 46$ ) had severe trauma (ISS  $\geq 25$ ), compared with only 32.0% of survivors ( $n = 16$ ; Pearson  $\chi^2 = 38.98$ ,  $df = 2$ ,  $p < 0.001$ ; Table 2). This closely matches intensive care admission profiles in severe polytrauma cohorts.<sup>51</sup>

Receiver operating characteristic curve analyses ordered the AUC point estimates of the three scoring systems for

predicting in-hospital mortality as ISS, RTS, and SI (Table 3 and Figure 3); the 95% confidence intervals overlapped. The Injury Severity Score had the highest AUC, 0.88 (95% CI: 0.81–0.95, SE 0.036,  $p < 0.001$ ). The optimal ISS cutoff was  $\geq 29$ , based on the maximum Youden Index ( $J = 0.66$ ). It gave a sensitivity of 82.0%, specificity of 84.0%, positive predictive value of 83.7%, negative predictive value of 82.4%, and overall diagnostic accuracy of 83.0%. The Revised Trauma Score discriminated well, with an AUC of 0.83 (95% CI: 0.75–0.91, SE 0.041,  $p < 0.001$ ). Its optimal threshold of  $\leq 6.95$  (Youden Index  $J = 0.54$ ) yielded a sensitivity of 76.0%, specificity of 78.0%, PPV of 77.6%, NPV of 76.5%, and diagnostic accuracy of 77.0%. The Shock Index showed acceptable discrimination. Its AUC was 0.76 (95% CI: 0.67–0.85, SE 0.046,  $p < 0.001$ ). Its optimal cutoff of  $\geq 1.05$  (Youden Index  $J = 0.46$ ) produced a sensitivity of 72.0%, specificity of 74.0%, PPV of 73.5%, NPV of 72.5%, and diagnostic accuracy of 73.0%.

**Table 3.** Discrimination and diagnostic accuracy of the three scores for in-hospital mortality (receiver operating characteristic analysis).

| Scoring System              | AUC-ROC | 95% CI      | SE    | Cutoff*     | Sens  | Spec  | PPV   | NPV   |
|-----------------------------|---------|-------------|-------|-------------|-------|-------|-------|-------|
| Injury Severity Score (ISS) | 0.88    | 0.81 – 0.95 | 0.036 | $\geq 29$   | 82.0% | 84.0% | 83.7% | 82.4% |
| Revised Trauma Score (RTS)  | 0.83    | 0.75 – 0.91 | 0.041 | $\leq 6.95$ | 76.0% | 78.0% | 77.6% | 76.5% |
| Shock Index (SI)            | 0.76    | 0.67 – 0.85 | 0.046 | $\geq 1.05$ | 72.0% | 74.0% | 73.5% | 72.5% |

**Notes:** Optimal cutoff thresholds were derived using the maximum Youden Index ( $J = \text{Sensitivity} + \text{Specificity} - 1$ ):  $J_{ISS} = 0.66$ ,  $J_{RTS} = 0.54$ ,  $J_{SI} = 0.46$ . All AUC values differed significantly from the null hypothesis area of 0.50 ( $p < 0.001$ ). AUC: Area Under the Receiver Operating Characteristic Curve; CI: Confidence Interval; NPV: Negative Predictive Value; PPV: Positive Predictive Value; SE: Standard Error; Sens: Sensitivity; Spec: Specificity. Sens, Spec, PPV, and NPV are percentages; PPV and NPV apply to a sample with 50% mortality by design.

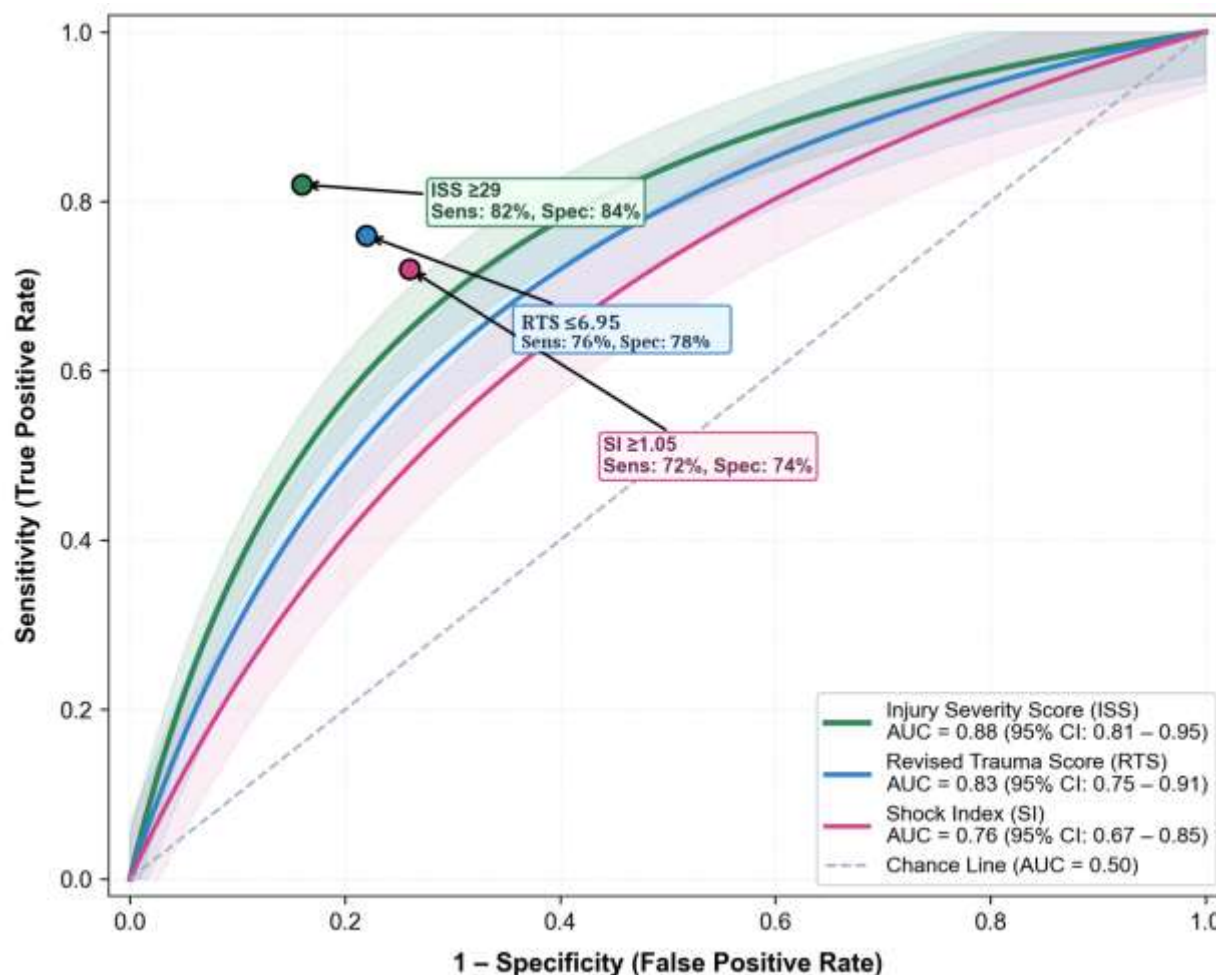


**Figure 3.** Receiver operating characteristic (ROC) curves for in-hospital mortality: Injury Severity Score (ISS, AUC 0.88, 95% CI 0.81–0.95), Revised Trauma Score (RTS, AUC 0.83, 95% CI 0.75–0.91), and Shock Index (SI, AUC 0.76, 95% CI 0.67–0.85). Shaded regions show 95% confidence intervals; dots mark the optimal Youden index cut-offs.

A direct DeLong comparison of the receiver operating characteristic curves showed that the Injury Severity Score (AUC 0.88, 95% CI: 0.81–0.95) discriminated significantly better than the Shock Index (AUC 0.76, 95% CI: 0.67–0.85; DeLong  $Z = 2.45$ ,  $p = 0.014$ ). Against the Revised Trauma Score (AUC 0.83, 95% CI: 0.75–0.91; DeLong  $Z = 1.18$ ,  $p = 0.238$ ), the difference was not statistically significant. The composite physiological score (RTS) reached numerically higher discrimination than the simple hemodynamic ratio (SI), although the difference was not statistically significant (DeLong  $Z = 1.62$ ,  $p = 0.105$ ). These tests show that the ISS had the highest AUC and differed significantly only from the Shock Index; the RTS, which draws on bedside measurements, did not differ significantly from the ISS.

In individual binary logistic regression models, all three scoring systems were significant predictors of in-hospital mortality (Table 4 and Figure 4, with odds ratio forestplots in Figure 4A and decile calibration curves in Figure 4B). In the ISS model, each one-point increase in anatomical injury

severity was associated with a 15% increase in the odds of in-hospital death (Beta = +0.140, SE 0.027, Wald  $\chi^2 = 26.89$ ,  $p < 0.001$ ; Odds Ratio [OR] 1.15, 95% CI: 1.09–1.21). In the RTS model, higher physiological stability was protective, and each one-point increase in RTS more than halved the odds of mortality (Beta = -0.799, SE 0.173, Wald  $\chi^2 = 21.32$ ,  $p < 0.001$ ; OR 0.45, 95% CI: 0.32–0.63). In the SI model, hemodynamic decompensation raised mortality risk considerably: for every 1.0-unit increase in Shock Index, the odds of death quadrupled (Beta = +1.416, SE 0.332, Wald  $\chi^2 = 18.20$ ,  $p < 0.001$ ; OR 4.12, 95% CI: 2.15–7.89). The Hosmer–Lemeshow test showed no significant disagreement between predicted and observed mortality probabilities in any of the three models (Table 4, Figure 4B):  $\chi^2$  was 8.91 for the ISS model ( $df = 8$ ,  $p = 0.350$ ), 10.24 for the RTS model ( $df = 8$ ,  $p = 0.248$ ), and 7.66 for the SI model ( $df = 8$ ,  $p = 0.467$ ). Calibration estimated in 100 patients with an enriched outcome prevalence is limited evidence of calibration in unselected patients.<sup>44,45</sup>

**Table 4.** Univariable logistic regression models and Hosmer–Lemeshow calibration for in-hospital mortality (N = 100).

| Model Predictor                  | B      | SE    | Wald $\chi^2$ | p-value | Odds Ratio | 95% CI      | Hosmer-Lemeshow*         |
|----------------------------------|--------|-------|---------------|---------|------------|-------------|--------------------------|
| ISS Model (per 1-point increase) | +0.140 | 0.027 | 26.89         | <0.001  | 1.15       | 1.09 – 1.21 | $\chi^2=8.91$ (p=0.350)  |
| RTS Model (per 1-point increase) | -0.799 | 0.173 | 21.32         | <0.001  | 0.45       | 0.32 – 0.63 | $\chi^2=10.24$ (p=0.248) |
| SI Model (per 1.0-unit increase) | +1.416 | 0.332 | 18.20         | <0.001  | 4.12       | 2.15 – 7.89 | $\chi^2=7.66$ (p=0.467)  |

**Notes:** Hosmer–Lemeshow goodness-of-fit test evaluated across 10 deciles of predicted risk (degrees of freedom = 8). A non-significant p-value ( $p > 0.05$ ) indicates acceptable model calibration without statistically significant discrepancy between observed and expected mortality events. B: Regression unstandardized coefficient; CI: Confidence Interval; SE: Standard error; Wald  $\chi^2$ : Wald chi-square test statistic.

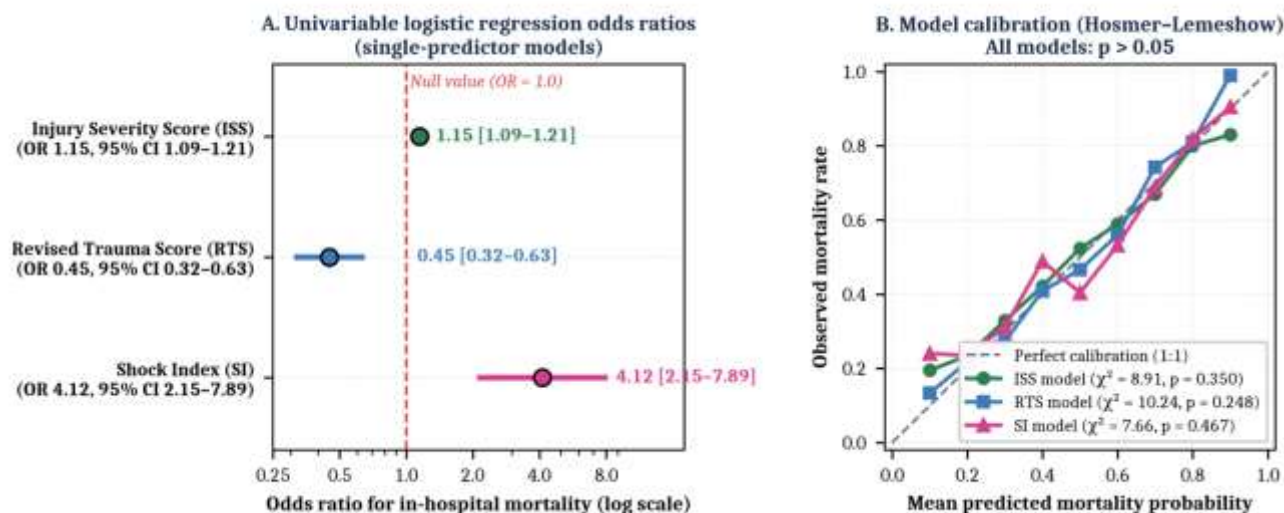


**Figure 4.** Logistic regression effect sizes and calibration: (A) odds ratios with 95% confidence intervals from the univariable models; (B) calibration across risk deciles for ISS ( $p = 0.350$ ), RTS ( $p = 0.248$ ), and SI ( $p = 0.467$ ).

### 3.3. Secondary and subgroup analyses

In exploratory subgroup analyses by injury mechanism ( $n = 12$  to  $54$ ), ISS had the highest AUC in each subgroup, for

**Table 5.** Exploratory subgroup AUCs by injury mechanism and inter-rater reliability audit ( $N = 100$ ).

| Etiology / Audit Subgroup            | Subgroup Size (n) | ISS AUC (95% CI)           | RTS AUC (95% CI)   | SI AUC (95% CI)    | Dominant Insult Axis     |
|--------------------------------------|-------------------|----------------------------|--------------------|--------------------|--------------------------|
| Road Traffic Accidents (Combined)    | 54 (54.0%)        | 0.89 (0.80 – 0.98)         | 0.84 (0.73 – 0.95) | 0.78 (0.66 – 0.90) | Blunt Multi-System       |
| Multiple-Vehicle Collisions          | 30 (30.0%)        | 0.90 (0.78 – 1.00)         | 0.85 (0.71 – 0.99) | 0.79 (0.63 – 0.95) | High-Velocity Impact     |
| Single-Vehicle Incidents             | 24 (24.0%)        | 0.88 (0.74 – 1.00)         | 0.83 (0.66 – 1.00) | 0.77 (0.58 – 0.96) | Deceleration / Roll-over |
| Thermal / Electrical Burns           | 20 (20.0%)        | 0.85 (0.68 – 1.00)         | 0.81 (0.62 – 1.00) | 0.74 (0.52 – 0.96) | Systemic SIRS / Edema    |
| Falls from Height                    | 14 (14.0%)        | 0.88 (0.70 – 1.00)         | 0.82 (0.59 – 1.00) | 0.75 (0.49 – 1.00) | Axial / Musculoskeletal  |
| Penetrating Trauma                   | 12 (12.0%)        | 0.91 (0.74 – 1.00)         | 0.86 (0.65 – 1.00) | 0.80 (0.55 – 1.00) | Vascular Exsanguination  |
| Inter-Rater Reliability (ISS Audit)* | 20 (20.0%)        | $\kappa = 0.84$ (SE 0.082) | Agreement = 90.0%  | $p < 0.001$        | Dual-Coder Validation    |

**Notes:** Inter-rater agreement for retrospective Abbreviated Injury Scale (AIS) and ISS scoring was independently verified on a randomly selected 20% validation sample ( $n = 20$ ) by two certified trauma reviewers, yielding a Cohen's kappa coefficient of  $\kappa = 0.84$  (95% CI: 0.68 – 1.00), demonstrating almost perfect inter-coder reliability. AUC: Area under the receiver operating characteristic curve; CI: Confidence interval; SE: Standard error;  $\kappa$ : Cohen's kappa statistic.

To characterize further the physiological trajectory of this high-mortality cohort, detailed non-parametric and quartile stratifications were evaluated across the initial trauma scores (Table 2, Table 5). Among in-hospital survivors, 76.0% ( $n = 38$ ) had a Revised Trauma Score of 7.84 (the theoretical normal ceiling), whereas only 20.0% of non-survivors ( $n = 10$ ) had an RTS of 7.84, and most fatal cases showed marked physiological derangement (RTS  $< 6.0$  in 48.0%,  $n = 24$ ; Mann-Whitney  $U = 422.5$ ,  $p < 0.001$ ). For the Shock Index, 84.0% of survivors ( $n = 42$ ) presented with an SI  $< 1.0$ , whereas 76.0% of non-survivors ( $n = 38$ ) presented with an SI  $\geq 1.0$  (mean  $1.36 \pm 0.38$ ;  $t = -8.67$ ,  $p < 0.001$ ). This corroborates a high prevalence of uncompensated shock among fatal cases.

## 4. Discussion

### 4.1. Principal findings

We set out to compare the predictive accuracy, discriminative power, and calibration of three

road traffic crashes (AUC 0.89), thermal burns (AUC 0.85), falls (AUC 0.88), and penetrating trauma (AUC 0.91; Table 5).<sup>52</sup>

foundational trauma scoring systems, the Revised Trauma Score, Shock Index, and Injury Severity Score, for in-hospital mortality in an enriched cohort of severely injured adult trauma patients in Indonesia. Three findings emerged. Each score was a statistically significant predictor of in-hospital mortality in separate models. The anatomically based ISS had the highest AUC (0.88), the composite physiological RTS came next (AUC 0.83), and the simple hemodynamic SI was lowest (AUC 0.76); only the difference between ISS and SI was statistically significant. The Hosmer-Lemeshow test was not significant in any of the three models ( $p > 0.05$ ). This ordering may reflect the biological sequence that runs from initial physical tissue trauma through acute physiological shock to downstream systemic organ failure, as schematized in Figure 5; none of these biological processes was measured in this study.<sup>37</sup>

### Integrated pathophysiological architecture of severe trauma and scoring metrics

*Mechanistic interplay linking initial structural/physiological insults to cellular cascades, endotheliopathy, and lethal outcome*

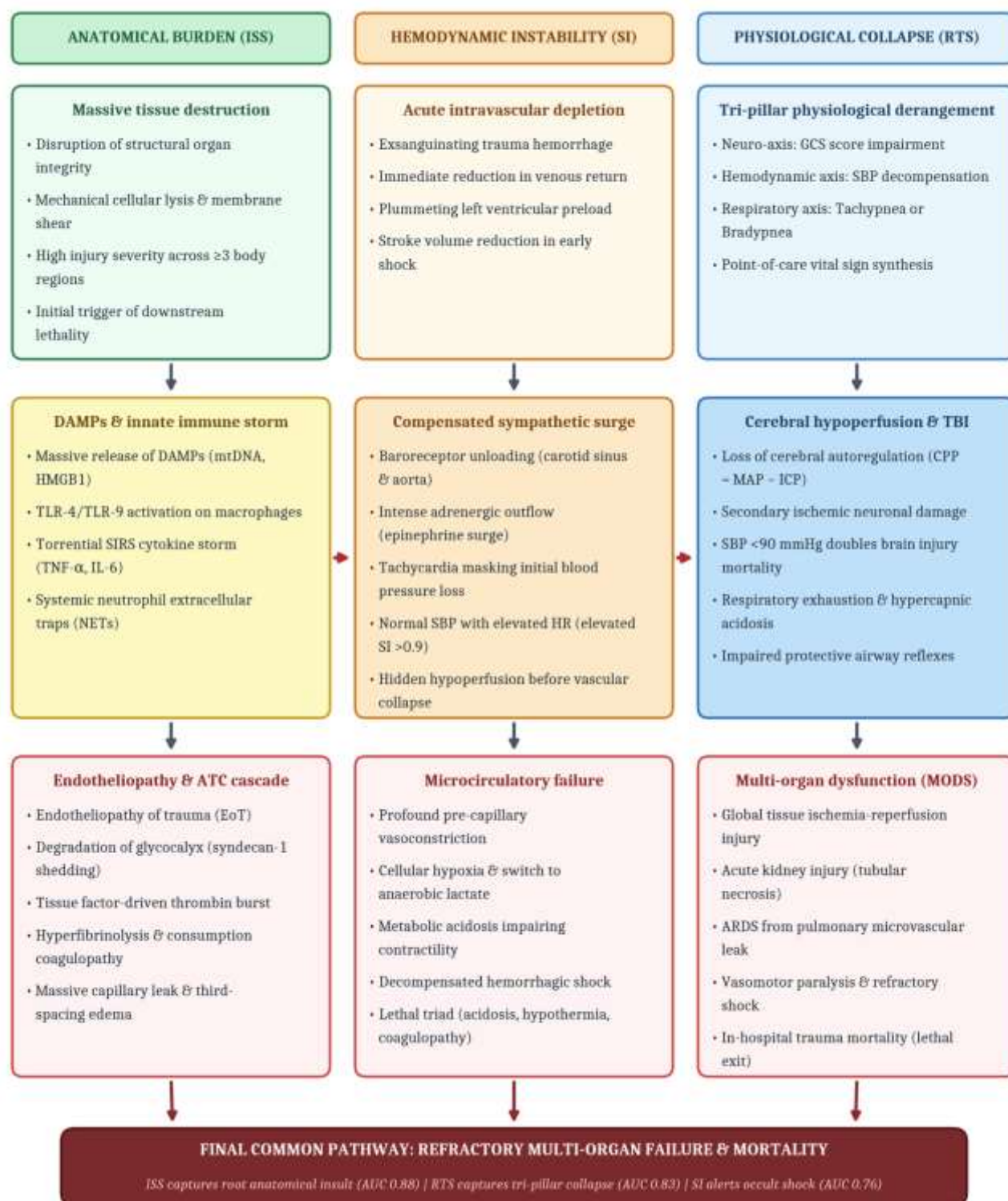


**Figure 5.** Conceptual scheme linking anatomical tissue disruption and physiological failure to cellular inflammatory cascades, endotheliopathy, acute traumatic coagulopathy, and mortality in severe trauma.

#### 4.2. Comparison with the published literature

Our findings agree with and extend contemporary international trauma literature.<sup>11,53</sup> In a meta-analysis of trauma scoring systems, Liu and colleagues reported pooled AUC values for ISS of 0.84 to 0.89 across diverse adult populations, close to our observed AUC of 0.88. Previous study evaluated RTS and ISS in 350 polytrauma

patients and found better discrimination for ISS (AUC 0.86) than for RTS (AUC 0.81).<sup>11</sup> In a prospective investigation of major adult trauma, Samuthtai and colleagues documented that combining physiological parameters with anatomical severity gave optimal predictive performance, while individual physiological scores had AUCs between 0.75 and 0.82.<sup>53</sup> In a Korean single-institution cohort of 7,120 trauma patients, the AUCs for mortality were 0.866 for ISS

and 0.894 for RTS.<sup>54</sup> In a registry of 2,007 adults in Pakistan, RTS had the highest AUC in neurotrauma (0.874), whereas ISS (0.722) and TRISS (0.729) performed less well in polytrauma.<sup>19</sup> Our results are broadly in line with these reports from other centers, although the relative order of RTS and ISS differs between studies, and they were obtained under severe emergency crowding.<sup>55</sup>

We compared our findings with major international trauma registries, namely the German TraumaRegister DGU, the United States National Trauma Data Bank (NTDB), and the Japan Trauma Data Bank. Our discrimination estimates are of a similar magnitude to values reported from these registries. Regional epidemiological differences exist.<sup>27,49,56</sup> In high-income registries, median pre-hospital transport times are frequently under 30 minutes, and advanced helicopter emergency medical services (HEMS) provide physician-led field resuscitation, and prehospital stabilization there can alter arrival vital signs and dampen the predictive power of initial physiological scores.<sup>57</sup> In our Indonesian cohort, pre-hospital transit times averaged 75.0 minutes (IQR 50.0–110.0 minutes), so the initial emergency department vital signs reflect the raw, cumulative impact of protracted tissue hypoperfusion, unresuscitated hemorrhagic shock, and secondary hypoxic-ischemic insults.<sup>4</sup> This may explain why the physiological scores in our study, particularly the RTS (AUC 0.83) and Shock Index (AUC 0.76), discriminated well. Their performance is comparable to figures reported from specialized warzone registries where evacuation delays are similarly prevalent.<sup>58–60</sup>

#### 4.3. Interpretation and possible mechanisms

The mechanisms discussed in this section were not measured in this study and are offered as possible explanations. The Injury Severity Score had the highest AUC (0.88), which is compatible with a role of cumulative anatomical destruction in determining survival after major trauma.<sup>9</sup> Physiological tools capture the immediate, dynamic response to injury. The ISS instead measures the physical insult itself. It does so across the three most severely damaged body regions.<sup>9</sup> Non-survivors in our high-mortality cohort carried a heavy anatomical burden, with a mean ISS of  $35.8 \pm 8.1$  against  $21.2 \pm 6.4$  in survivors. An ISS above 25 denotes severe trauma. Death is likely once physiological reserve is exhausted.<sup>17</sup> Pathophysiologically, extensive tissue destruction triggers the innate immune cascade, in which sheared and necrotic cells release endogenous damage-associated molecular patterns (DAMPs), including high-mobility group box 1 (HMGB1), mitochondrial DNA, histone proteins, and heat shock proteins, as schematized in Figure 5.<sup>37,38</sup> These circulating DAMPs bind toll-like receptors (TLR-4 and TLR-9) on resident tissue macrophages and circulating dendritic cells and set off a severe systemic inflammatory response syndrome (SIRS).<sup>37</sup> The resulting cytokine storm brings surges in tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin-1 beta (IL-1 $\beta$ ), and interleukin-6 (IL-6), and it precipitates widespread endothelial activation and breakdown of the endothelial glycocalyx, which predisposes to multiple organ failure and secondary surgical hits.<sup>36,37</sup>

Severe anatomical trauma also precipitates the endotheliopathy of trauma (EoT) and acute traumatic coagulopathy (ATC; Figure 5).<sup>35,61</sup> Massive tissue disruption exposes subendothelial tissue factor, which activates the extrinsic coagulation cascade and generates uncontrolled thrombin bursts, while hypoperfusion and

cellular hypoxia induce profound endothelial shedding of syndecan-1 and heparan sulfate.<sup>35,62</sup> The shedding degrades the endothelial barrier. Generalized microvascular hyperpermeability, widespread capillary leak, and third-space fluid sequestration follow and worsen the loss of effective circulating volume.<sup>35,36</sup> Damaged endothelial cells also release large quantities of tissue plasminogen activator (tPA). Systemic hyperfibrinolysis results. Hemostatic fibrin clots are lysed as quickly as they form, so targeted viscoelastic monitoring and hemostatic protocols are needed.<sup>62–64</sup> Combined with hypothermia, acidosis, and consumption coagulopathy, the classic trauma triad of death, these cascades lead to refractory multi-organ dysfunction syndrome (MODS) and secondary cardiovascular collapse.<sup>62,63</sup> The ISS captures the cumulative physical volume of cellular damage, so it may serve as an anatomical surrogate for the biological intensity of this downstream inflammatory and coagulopathic cascade. This may partly explain its higher AUC for in-hospital mortality.<sup>9</sup>

The Revised Trauma Score discriminated strongly in our cohort (AUC 0.83), second only to the ISS. Each one-point increase in RTS reduced the odds of in-hospital mortality by 55% (OR 0.45, 95% CI: 0.32–0.63).<sup>8</sup> Its diagnostic strength rests on a three-part physiological design. It evaluates central nervous system integrity (GCS), cardiovascular perfusion (SBP), and respiratory mechanics (RR; Figure 5) together.<sup>8</sup> The GCS component carries the heaviest weight (weight = 0.9368). This reflects the vulnerability of the central nervous system as the body's primary metabolic end-organ.<sup>8</sup> In severe trauma, a suppressed GCS indicates either direct focal mechanical brain trauma (such as traumatic axonal injury, epidural hematoma, or subdural hematoma) or profound global cerebral hypoperfusion secondary to systemic shock.<sup>8,39,65</sup> When mean arterial pressure falls below the autoregulatory limit in the setting of elevated intracranial pressure, cerebral perfusion pressure (CPP = MAP – ICP) drops sharply, and rapid cerebral ischemia, loss of consciousness, and respiratory depression follow.<sup>66</sup>

The cardiovascular (weight = 0.7326) and respiratory (weight = 0.2908) components of the RTS capture the hemodynamic and ventilatory decompensation of shock.<sup>8</sup> An initial SBP < 90 mmHg denotes decompensated circulatory shock. It signals that endogenous adrenergic vasoconstriction and tachycardia have failed to preserve adequate organ perfusion.<sup>13</sup> Non-survivors in our cohort had a mean SBP of  $86.6 \pm 21.8$  mmHg, against  $118.2 \pm 16.4$  mmHg in survivors. Tachypnea (mean RR  $26.4 \pm 6.3$  breaths/min in non-survivors) may reflect respiratory compensation for metabolic acidosis, which was not measured, or direct parenchymal thoracic trauma such as flail chest, pulmonary contusion, or tension pneumothorax.<sup>8</sup> The RTS merges these three vital organ axes into a single continuous metric. It gives an instant snapshot of physiological collapse. The snapshot correlates closely with imminent mortality across both blunt and penetrating injury mechanisms, which supports its continued use as a bedside triage instrument.<sup>8,11</sup>

The Shock Index had the lowest AUC in our comparison (0.76) but remained a statistically significant predictor of mortality ( $p < 0.001$ ), and each 1.0-unit elevation quadrupled the odds of death (OR 4.12, 95% CI: 2.15–7.89). Its clinical value lies in bedside simplicity. It also lies in sensitivity for detecting occult, compensated hemorrhagic shock, more than in discriminative precision.<sup>12,13</sup> In acute trauma hemorrhage, baroreceptor unloading triggers an

immediate sympathetic surge with intense release of epinephrine and norepinephrine.<sup>13</sup> This neurohumoral reflex raises heart rate and peripheral vascular resistance and keeps systolic blood pressure deceptively “normal” despite significant circulating volume loss, as schematized in Figure 5.<sup>13</sup> An SI above 0.9, and particularly one above our identified optimal threshold of 1.05, is an urgent warning that the patient’s cardiovascular system is under extreme compensatory strain.<sup>13</sup> In thermal burns, falls from height, and polytrauma, the initial presentation may mask severe internal injury, and calculating the SI there may help reveal occult hypoperfusion and prompt immediate fluid resuscitation, blood typing, and emergency surgical consultation.<sup>14,16</sup>

Our study did not record the time of death, so the following account of the mechanotransduction of cellular injury and the biological timeline of trauma-induced mortality is hypothetical.<sup>37,67</sup> In severe mechanical trauma, death follows a bimodal or trimodal distribution: immediate deaths occur within minutes from massive exsanguination or devastating neurotrauma, early deaths occur within hours from acute hemorrhagic shock and traumatic brain injury, and late deaths occur within days to weeks from multiple organ dysfunction syndrome (MODS) and sepsis.<sup>67</sup> The physiological scoring tools (SI and RTS) would be expected to be sensitive for the first two phases. They detect the immediate autonomic, respiratory, and neurological manifestations of acute hypoperfusion.<sup>8,12</sup> Late trauma mortality, however, is thought to be driven by the magnitude of the biological derangement that tissue trauma induces: cellular DAMP release, systemic complement activation, neutrophil extracellular trap (NET) formation, and endotheliopathy of trauma, as schematized in Figure 5.<sup>35,37,38</sup> The ISS directly quantifies the aggregate volume of damaged, ischemic, and devitalized tissue across multiple organ beds, so it serves as a proxy for the total biological “second-hit” inflammatory potential that drives late organ failure.<sup>37,68</sup> This mechanistic distinction may explain why the anatomical ISS had the highest AUC (0.88) for in-hospital mortality across the complete hospital stay.<sup>9</sup>

#### 4.4. Implications for practice

Our findings do not support an adversarial “either/or” choice between physiological and anatomical scores. They are compatible with a temporally staged triage approach, which was not tested here. In such an approach the two kinds of score would work together. In resource-constrained emergency environments, whole-body CT imaging may be inaccessible or delayed by operational backlogs, and trauma evaluation could then proceed in distinct temporal phases.<sup>25</sup> The first phase is the Immediate Resuscitation Phase (minutes 0 to 5). The Shock Index could be calculated at once from the initial vital signs when the patient arrives on a stretcher.<sup>13</sup> A patient with an SI  $\geq 1.05$  could trigger an immediate high-tier trauma resuscitation response. Blood components and point-of-care ultrasound (extended focused assessment with sonography for trauma, eFAST) could be prepared.<sup>22,69</sup> For patients with profound shock or peri-arrest physiology, early risk stratification is vital to guide resuscitative efforts and avoid futile interventions.<sup>50</sup> The second phase is the Primary Survey Phase (minutes 5 to 15). The RTS could be computed once the ABCDE (airway, breathing, circulation, disability, exposure) survey is complete.<sup>8</sup> An RTS  $\leq 6.95$  indicates severe multi-system physiological derangement. It could prompt urgent airway control, tube thoracostomy,

and emergent surgical mobilization.<sup>8</sup> The third phase is the Definitive Prognostication Phase (hours 1 to 24). The ISS could be calculated after diagnostic imaging and surgical exploration.<sup>9</sup> An ISS  $\geq 29$  identified patients at extreme mortality risk in this cohort, who may need dedicated intensive care unit (ICU) placement, damage control surgical strategies, and vigilant monitoring for multi-organ failure.<sup>63,68</sup> Emerging machine learning algorithms also show that integrating sequential physiological and anatomical variables gives better prognostic accuracy than single static scores.<sup>70</sup>

From an operational and health systems perspective, our findings have direct practical implications for trauma care delivery in resource-constrained low- and middle-income countries.<sup>1,21</sup> Many district and provincial hospitals across Southeast Asia lack on-site 24-hour trauma surgery teams, immediate pan-scan CT facilities, and massive transfusion protocols supported by viscoelastic testing (thromboelastography or rotational thromboelastometry, ROTEM).<sup>4,63</sup> Under these constraints, a three-tiered, temporally staged triage protocol, which would need prospective testing, could offer an objective clinical algorithm that makes better use of resources:

First, during the Immediate Resuscitation Phase (Minute 0 to 5 upon patient arrival), the Shock Index is calculated at once from the initial pulse and non-invasive blood pressure reading.<sup>13</sup> A threshold of SI  $\geq 1.05$  could identify patients in compensated or decompensated hemorrhagic shock and trigger early activation of the hospital’s emergency blood transfusion protocol, cross-matching of uncrossmatched packed red blood cells, and immediate mobilization of bedside ultrasound (eFAST) and surgical personnel.<sup>22,69</sup> This could avoid the delay of waiting for the patient to develop frank hypotension (SBP  $< 90$  mmHg). It is often a pre-terminal sign in young adults.<sup>14</sup>

Second, during the Primary Survey Phase (Minute 5 to 15), the Revised Trauma Score is formally documented as the trauma team completes the structured ABCDE evaluation.<sup>8</sup> An RTS  $\leq 6.95$  signifies marked multi-axis physiological derangement. It could direct immediate airway securing by rapid sequence induction, placement of large-bore chest tubes for hemothorax or pneumothorax, and prioritization for emergent damage-control laparotomy or craniotomy.<sup>8</sup> In facilities with limited operating room capacity, this objective physiological cutoff helps triage officers decide which patient must enter the operating suite first when multiple casualties present at the same time.<sup>7,55</sup>

Third, during the Definitive Prognostication Phase (hours 1 to 24), the Injury Severity Score is definitively tabulated once diagnostic imaging and surgical interventions are complete.<sup>9</sup> An ISS  $\geq 29$  categorized patients in this cohort with a high anatomical trauma burden. Such patients face a high risk of secondary systemic inflammatory complications, acute respiratory distress syndrome (ARDS), and multi-organ failure.<sup>63,68</sup> In resource-constrained tertiary centers where intensive care unit (ICU) beds and mechanical ventilators are scarce, this anatomical score could support intensive care allocation, prolonged invasive hemodynamic monitoring, and prognostic communication with patient families.<sup>51</sup>

#### 4.5. Strengths

This study has several methodological strengths. The cohort was enriched for outcome, with a 50% mortality rate and a high mean ISS ( $28.5 \pm 10.3$ ), which allowed evaluation of score discrimination at the extreme end of

trauma severity and avoided the dilution seen in low-severity cohorts. All physiological data came from the first set of vital signs taken at emergency department arrival. This limits confounding from post-resuscitation interventions. We also applied a quality control protocol to AIS coding (a 20-patient audit). It produced almost perfect inter-rater reliability (Cohen's kappa  $\kappa = 0.84$ ) and supports the internal validity of our anatomical scoring.<sup>41</sup> Our findings also corroborate recent European studies. These examine the distinct prognostic dynamics of physiological derangement versus anatomical trauma in adult cohorts.<sup>49</sup>

#### 4.6. Limitations

Several limitations must be stated explicitly. First, purposive sampling created an enriched high-mortality cohort, which limits how directly our absolute predictive values and cutoffs apply to unselected trauma populations with lower injury severity. In a typical unselected emergency department trauma registry, where mortality is 2% to 5%, the negative predictive values of these scores would be considerably higher and the positive predictive values would fall.<sup>26</sup> Second, the single-center retrospective design carries inherent susceptibility to information bias and reflects the specific trauma infrastructure, transport delays, and clinical protocols of Dr. Saiful Anwar Hospital, which may not fully represent rural community hospitals with less specialized surgical capacity. Third, our analysis focused strictly on RTS, SI, and ISS. It did not incorporate point-of-care laboratory biomarkers such as serum lactate, base deficit, thromboelastography, or syndecan-1 levels, and it did not examine the effect of whole blood versus component resuscitation.<sup>35,70</sup> Fourth, the ISS was derived with operative records and forensic autopsy summaries, which may carry outcome-related information (incorporation bias). Fifth, the cut-offs were derived and evaluated in the same 100 patients, so they are likely to be optimistic, and the confidence intervals are wide. Sixth, no sample-size calculation was reported, no combined model or TRISS comparison was made, and the time of death was not recorded.

#### 4.7. Recommendations for future research

Future prospective multicenter investigations across Indonesia are needed to validate these findings and to test whether hybrid models that combine point-of-care biomarkers with physiological scores can bridge the gap between rapid bedside triage and definitive anatomical prognostication.<sup>71</sup> Future studies should validate the cut-offs in an independent, consecutively enrolled cohort, report predictive values at the true outcome prevalence, compare the three scores with a combined model, and record the time of death.

### 5. Conclusion

In this single-center study of severely injured adult trauma patients admitted to a tertiary referral hospital in Indonesia, the Injury Severity Score, Revised Trauma Score, and Shock Index were each significantly associated with in-hospital mortality in separate models. The ISS had the highest AUC (0.88). Its AUC was significantly higher than that of the Shock Index, but not of the Revised Trauma Score.<sup>9</sup> The physiological tools had lower AUCs (RTS 0.83, SI 0.76). They remain useful at the bedside: they are fast, simple, and available without diagnostic delay.<sup>8,12</sup> The ISS, by contrast, can be calculated only after imaging and surgical assessment, so it cannot guide hyperacute triage,

whereas the Shock Index and the Revised Trauma Score give an immediate, objective estimate of risk.<sup>8,9,12</sup> These findings suggest a temporally staged approach to risk stratification. Bedside physiological metrics would guide immediate triage, blood component mobilization, and emergent resuscitation; anatomical scoring would then direct definitive surgical planning, intensive care resource allocation, and prognostication. Prospective testing in unselected trauma populations is needed, including its effect on triage precision, time to life-saving interventions, and survival.

### 6. Declarations

**Ethical approval and consent to participate.** The study protocol was approved by the Ethics Committee CMHC Research Center, Indonesia (Approval Certificate No. 2023/400). In accordance with the Declaration of Helsinki and international guidelines for retrospective research involving de-identified health data, the requirement for individual written informed consent was waived by the ethics committee.

**Consent for publication.** Not applicable; no individually identifiable data are presented.

**Availability of data and materials.** The complete de-identified primary dataset generated and analyzed during the current study is available from the corresponding author upon reasonable academic request, subject to institutional data governance agreements.

**Artificial intelligence (AI) use statement.** The authors declare that no generative artificial intelligence tool was used in the preparation of this article.

**Author contribution statement.** D.P.: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – original draft. A.Z.F. and R.M.L.: Supervision, Validation, Writing – review & editing. B.H.L.: Supervision, Project administration, Validation, Writing – review & editing. All authors read and approved the final manuscript.

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