

Research Article

Shock Index versus Modified Shock Index as Predictors of In-Hospital Mortality in COVID-19: A Retrospective Comparative Analysis

Abhishek S¹, Ramakrishnan T V², Srihari Cattamanchi³, Parthasarathy Vijayan⁴

¹Department of Trauma Care Management, Yenepoya School of Allied Health Sciences, Yenepoya, Deemed to be University, Mangalore, Karnataka, India

^{2,3}Department of Emergency Medicine, Sri Ramachandra Institute of Higher Education and Research, Chennai, Tamil Nadu, India

⁴Emergency Medical Services, School of Rehabilitation and Medical Sciences, College of Health Sciences, University of Nizwa, Sultanate of Oman

DOI: <https://doi.org/10.24321/0019.5138.202657>

I N F O

Corresponding Author:

Abhishek S, Department of Trauma Care Management, Yenepoya School of Allied Health Sciences, Yenepoya, Deemed to be University, Mangalore, Karnataka, India

E-mail Id:

abhisheksalomon98@gmail.com

Orcid Id:

<https://orcid.org/0000-0003-0833-7145>

How to cite this article:

Abhishek S, Ramakrishnan T V, Cattamanchi S, Vijayan P. Shock Index versus Modified Shock Index as Predictors of In-Hospital Mortality in COVID-19: A Retrospective Comparative Analysis. J Commun Dis. 2026;58(3):68-82.

Date of Submission: 2026-04-24

Date of Acceptance: 2026-09-14

A B S T R A C T

Introduction: Rapid identification of haemodynamic instability is important when triaging patients with severe COVID-19. The Shock Index (SI; heart rate/systolic blood pressure) ignores the diastolic component of perfusion pressure, whereas the Modified Shock Index (MSI; heart rate/mean arterial pressure) incorporates it, and MSI has therefore been assumed to be the better prognostic marker. Whether MSI actually discriminates in-hospital mortality better than SI in critically ill patients has not been established. We performed an exploratory head-to-head comparison of the discriminative ability of the two indices.

Methods: We conducted a retrospective observational study of 93 consecutive adults with RT-PCR-confirmed COVID-19 admitted to the intensive care unit of a tertiary care centre between June 2020 and January 2021 (Institutional Ethics Committee for Student Projects, Sri Ramachandra Institute of Higher Education and Research; approval number CSP/21/APR/93/316, dated 30 April 2021). SI and MSI were computed from admission heart rate and blood pressure. The primary outcome was in-hospital mortality. Discrimination was quantified as the area under the receiver-operating-characteristic curve (AUC) with DeLong 95% confidence intervals (CI), and the AUCs of SI and MSI were compared directly using the paired DeLong test with bootstrap confirmation. Optimal cut-offs were derived by the Youden index; sensitivity, specificity, predictive values and likelihood ratios are reported with Wilson CIs. Relative risks (RR) and absolute risk differences were used for categorical associations because mortality was common, and odds ratios are reported only from logistic regression models adjusted for age and sex. No a priori sample-size calculation was possible; the precision attainable in this fixed cohort is reported instead.

Results: In-hospital mortality was 68.8% (64/93). Non-survivors were older than survivors (68.9 ± 12.5 vs 62.2 ± 14.9 years; mean difference 6.7 years, 95% CI 0.8–12.6; $p=0.026$), but admission SI (0.80 ± 0.21 vs 0.77 ± 0.16 ; $p=0.447$) and MSI (1.08 ± 0.27 vs 1.03 ± 0.19 ; $p=0.373$)

did not differ significantly. Discrimination was poor and virtually identical for the two indices: AUC 0.546 (95% CI 0.415–0.676) for SI and 0.546 (95% CI 0.417–0.674) for MSI, a difference of -0.0003 (95% CI -0.036 to +0.035; DeLong $p=0.988$), with both CIs including the no-discrimination value of 0.50. At Youden-optimal cut-offs (SI ≥ 0.80 ; MSI ≥ 1.05), sensitivity and specificity were 54.7%/65.5% and 56.3%/58.6%, respectively, and accuracy (58.1% and 57.0%) did not exceed the 68.8% no-information rate. An abnormal MSI (<0.7 or >1.3 ; 18.3% of patients) was specific (93.1%) with a positive predictive value of 88.2%, but the association was imprecise and not statistically significant (RR 1.37, 95% CI 1.08–1.74; odds ratio 4.13, 95% CI 0.88–19.44; Fisher exact $p=0.081$), and mortality across MSI quartiles was non-monotonic (66.7%, 60.9%, 87.0%, 60.9%). Age was the only independent predictor of death (adjusted odds ratio 1.04 per year, 95% CI 1.01–1.08; $p=0.023$); adding SI or MSI to an age- and sex-based model changed the AUC only marginally (0.642 to 0.657 and 0.662).

Conclusion: In this single-centre, high-mortality ICU cohort, neither the Shock Index nor the Modified Shock Index discriminated survivors from non-survivors, and MSI was not superior to SI ($\Delta\text{AUC} \approx 0.000$; $p=0.988$). Admission SI and MSI should therefore not be used on their own to prognosticate critically ill patients with COVID-19, in whom age carried more prognostic information than either index. Because this cohort could only reliably detect an AUC ≥ 0.675 , these findings are exploratory; adequately powered studies using serial rather than single admission measurements are required before either index is adopted or discarded for triage.

Keywords: COVID-19, Haemodynamic Monitoring, Shock Index, Modified Shock Index, Mortality Prediction, Roc Analysis, Prognostic Accuracy, And Critical Care Triage

Introduction

The emergence of the novel coronavirus (SARS-CoV-2) in late 2019 precipitated a global health crisis, with COVID-19 manifesting as a complex clinical entity ranging from mild respiratory symptoms to severe viral sepsis and multi-organ failure. A defining characteristic of severe COVID-19 is rapid clinical deterioration, often driven by a combination of hypoxaemia, systemic inflammation, and haemodynamic instability. In overwhelmed emergency departments and intensive care units, the ability to promptly identify patients teetering on the edge of decompensation is critical for triage and resource allocation.^{1,2}

Traditionally, clinicians have relied on standard vital signs—heart rate and blood pressure—to assess circulatory status. However, physiological compensation mechanisms can mask early signs of shock. Young or relatively healthy patients may maintain a normal systolic blood pressure

despite significant hypoperfusion, a phenomenon often termed “occult shock”. Relying solely on hypotension to trigger aggressive management can result in delayed intervention and increased mortality.^{3,4} Consequently, integrated haemodynamic ratios have gained prominence as more sensitive markers of early instability.⁵

The Shock Index (SI), calculated as the ratio of heart rate to systolic blood pressure, was introduced as a simple bedside tool to gauge the severity of hypovolemia and sepsis.^{6,7} An SI value exceeding 0.7—and particularly values approaching 1.0—has been widely correlated with poor outcomes in trauma, haemorrhage, and bacterial sepsis. Yet, the SI has a limitation: it relies exclusively on systolic pressure, potentially overlooking changes in vascular tone that are reflected in the diastolic pressure.^{8,9}

To address this, the Modified Shock Index (MSI) was developed, incorporating diastolic pressure by using Mean Arterial Pressure (MAP) in the denominator. Since diastolic pressure is a key determinant of coronary perfusion and reflects systemic vascular resistance, the MSI theoretically offers a more comprehensive picture of the cardiovascular state.^{10,11} While MSI has shown superior predictive value in trauma and general emergency populations, its specific utility in COVID-19 patients—who often present with unique hemodynamic profiles due to viral myocarditis or cytokine-induced vasodilation—remains under-explored.^{12,13}

This study therefore compares the Shock Index and the Modified Shock Index head-to-head in a cohort of critically ill patients with COVID-19. The primary objective was to quantify and directly compare the discriminative ability of the two indices for in-hospital mortality using receiver-operating-characteristic analysis with formal comparison of the areas under the curve. The secondary objectives were to characterize the performance of each index at conventional and data-derived cut-offs and to determine whether either index adds prognostic information to age and sex. Because the cohort was fixed in size and no a priori power calculation was feasible, the analysis is explicitly exploratory and hypothesis-generating.

Materials and Methods

Study Design and Setting

This retrospective observational study was conducted at the Department of Emergency Medicine and Intensive Care Unit (ICU) of a tertiary care center. The analysis focused on clinical data collected over an eight-month period from June 1, 2020, to January 31, 2021. The study protocol was reviewed and approved by the Institutional Ethics Committee for Student Projects (UG and Non-Medical PG Students), Sri Ramachandra Institute of Higher Education and Research (IEC approval number: CSP/21/APR/93/316; date of approval: 30 April 2021), and a

copy of the approval certificate is provided as a separate submission file (Supplementary File 1). Because the study was a retrospective review of existing hospital records, approval of the Committee was obtained before the records were accessed for research purposes, and the clinical period analyzed therefore precedes the date of approval. Because the study involved only retrospective review of de-identified hospital records, with no patient contact and no deviation from routine care, the committee waived the requirement for individual informed consent. The study was conducted in accordance with the Declaration of Helsinki and the Indian Council of Medical Research National Ethical Guidelines for Biomedical and Health Research Involving Human Participants.

Study Population and Sampling

We employed a complete enumeration sampling strategy, screening all patients admitted to the ICU during the study window. A total of 93 patients met the eligibility requirements and were included in the final analysis. No a priori power calculation was performed, because the study was a retrospective analysis of a fixed, completely enumerated cohort rather than a prospectively sized study. Instead of a post hoc power calculation on the observed effect, which is uninformative, we quantified the precision and the detectable effect size that this sample can support and interpreted all findings within those limits, as detailed below.

Sample Size and Precision Considerations

With 64 deaths and 29 survivors, the two-sided 95% confidence interval for a single AUC has a half-width of approximately ± 0.13 , so any AUC estimate in this cohort spans a range of about 0.26 AUC units. At $\alpha=0.05$ (two-sided) and 80% power, the smallest AUC that can be distinguished from chance (0.50) is 0.675; an index whose true AUC is 0.55 would be detected with only about 10% power. For the head-to-head comparison, the standard error of the paired difference in AUCs (DeLong) was 0.018, so the study could detect a true between-index difference of ≥ 0.051 AUC units with 80% power. Precision for threshold-based measures is similarly limited: the 95% CI half-width is approximately ± 11 percentage points for sensitivity (64 events) and ± 17 percentage points for specificity (29 non-events). Finally, the smaller outcome group (29 survivors) restricts regression modelling to about three covariates at the conventional ten events per variable. This cohort is therefore adequate to exclude a large discriminative advantage of one index over the other and to detect strong prognostic performance ($\text{AUC} \geq 0.675$) had it been present, but it is underpowered to establish modest predictive utility for either index. All results are accordingly reported as exploratory, with emphasis on effect sizes and confidence intervals rather than on statistical significance.

Eligibility Criteria

Patients were included if they were 18 years of age or older and presented with clinical symptoms suggestive of COVID-19 (including but not limited to fever, dyspnea, cough, or fatigue). Confirmatory diagnosis was defined as a positive Reverse Transcription-Polymerase Chain Reaction (RT-PCR) test for SARS-CoV-2.

Exclusion criteria were strictly applied to ensure data integrity. Patients with negative RT-PCR results, those lacking definitive outcome data (e.g., patients who left against medical advice or were transferred to other facilities), and individuals with incomplete medical records preventing the calculation of hemodynamic indices were excluded from the study.

Data Collection and Variables

Demographic, clinical, and physiological data were extracted from the Medical Records Department (MRD) using a structured case report form. The primary baseline variables included age, gender, and the presence of comorbidities such as diabetes mellitus, hypertension, coronary artery disease (CAD), and chronic kidney disease (CKD).

Hemodynamic parameters recorded at the time of admission included heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP). From these primary vitals, two derived indices were calculated:

- **Shock Index (SI):** Defined as the ratio of Heart Rate to Systolic Blood Pressure (HR / SBP).
- **Modified Shock Index (MSI):** Defined as the ratio of Heart Rate to Mean Arterial Pressure (HR / MAP), where MAP was calculated as $[(2 \times \text{DBP}) + \text{SBP}] / 3$.

The primary outcome measure was in-hospital mortality, categorized simply as “Survivors” versus “Non-Survivors.”

Statistical Analysis

All analyses were performed on the complete dataset (93 patients, no missing values) using SPSS version 26.0 (IBM Corp., Armonk, NY), with ROC curves, DeLong confidence intervals, bootstrap resampling, and profile-likelihood estimates computed in Python 3.13 (NumPy, pandas); the analysis code and the derived dataset are available from the corresponding author. SI and MSI were computed for every patient from the recorded heart rate and systolic and diastolic blood pressure and agreed exactly with the values held in the study database (maximum absolute difference 0.000), confirming the internal consistency of the dataset. A two-sided p-value below 0.05 was considered statistically significant; because the analysis is exploratory, no adjustment for multiple comparisons was applied, and emphasis is placed on interval estimates.

Continuous variables are summarized as mean $\pm$ standard deviation together with median and interquartile range

and were compared between survivors and non-survivors using the independent-samples t-test; because heart rate and both indices were right-skewed, every comparison was repeated with the Mann–Whitney U test as a sensitivity analysis, and both p-values are reported. For each variable we report the between-group mean difference with its 95% confidence interval and the standardized mean difference (Cohen's d). The mean difference is retained because it expresses the group-level separation in the native physiological units, which is clinically interpretable, while Cohen's d expresses that separation relative to the within-group spread; neither quantity measures how well an individual patient can be classified, which is what the area under the ROC curve measures. This distinction is made explicitly because a statistically detectable difference in mean index values between outcome groups does not by itself establish that the index is clinically useful for predicting the outcome of an individual patient.

Categorical variables are presented as counts and percentages and compared with Pearson's χ^2 test; Fisher's exact test was used as the primary test whenever any expected cell count was below five, and the continuity-corrected χ^2 is additionally reported for sparse tables. Because in-hospital mortality was a common outcome (68.8%), the odds ratio substantially overestimates the risk ratio in this cohort; the relative risk, together with the absolute risk difference in percentage points, was therefore selected as the primary effect measure for all univariable categorical associations, and odds ratios are reported only where they are the native parameter of the analysis, that is, within logistic regression models. Confidence intervals for proportions, sensitivity, specificity, predictive values, and accuracy were calculated by the Wilson score method, and for likelihood ratios by the log method. Where any cell count was below five, odds-ratio confidence intervals were also computed by profile likelihood, because Wald (Woelf) intervals are unreliable with sparse data and can be discordant with the corresponding χ^2 p-value; in such tables the exact test and the profile-likelihood interval take precedence.

The primary analysis of predictive utility was discrimination. Receiver-operating-characteristic curves were constructed for SI and MSI and for age and the individual vital signs as context, and the area under the curve was estimated with 95% confidence intervals and p-values (versus AUC 0.50) by the DeLong method. Because both indices were measured in the same patients, they were compared directly using the paired DeLong test for correlated AUCs, reporting the difference in AUCs, its 95% confidence interval, and the correlation between the indices; the comparison was

repeated with 4,000 bootstrap resamples of the paired difference as a sensitivity analysis. Optimal cut-offs were identified by maximizing the Youden index ($J = \text{sensitivity} + \text{specificity} - 1$), and performance at these data-derived cut-offs is reported alongside performance at the conventional thresholds ($SI > 0.7$, $MSI > 1.3$, and the composite abnormal MSI of < 0.7 or > 1.3) so that the two types of thresholds are not conflated. Because a cut-off derived from the same data is optimistically biased, such values are reported as apparent performance only. Accuracy was interpreted against the no-information rate, that is, the accuracy obtained by predicting the majority outcome for every patient (68.8%).

The association of each index with mortality was additionally modelled by binary logistic regression, scaled per 0.1-unit increase in the index, first unadjusted and then adjusted for age and sex; the number of covariates was capped at three in accordance with the events-per-variable constraint described above, and both indices were also entered together to assess collinearity. Model performance is described by the likelihood-ratio test, Nagelkerke R^2 , the model AUC, and the Brier score, and the incremental value of each index was assessed by comparing an age- and sex-based model with and without the index. Finally, mortality was examined across quartiles of SI and MSI to test whether risk increased monotonically with the index, as would be expected if the index carried graded prognostic information.

Results

Baseline Characteristics and Physiological Profile

A total of 93 critically ill patients with confirmed COVID-19 were included in the final analysis, and in-hospital mortality was 68.8% (64/93). Non-survivors were significantly older than survivors (68.9 ± 12.5 vs 62.2 ± 14.9 years; mean difference 6.7 years, 95% CI 0.8 to 12.6; Cohen's d 0.51; t-test $p=0.026$, Mann–Whitney $p=0.036$). None of the admission vital signs or derived indices differed significantly between the outcome groups: heart rate 95.4 ± 13.1 vs 97.5 ± 15.3 bpm ($p=0.511$), systolic blood pressure 122.8 ± 20.7 vs 128.6 ± 16.8 mmHg ($p=0.188$), diastolic blood pressure 75.2 ± 12.2 vs 79.0 ± 9.0 mmHg ($p=0.136$), mean arterial pressure 91.0 ± 14.2 vs 95.5 ± 10.6 mmHg ($p=0.133$), SI 0.80 ± 0.21 vs 0.77 ± 0.16 ($p=0.447$), and MSI 1.08 ± 0.27 vs 1.03 ± 0.19 ($p=0.373$). Both indices were numerically higher in non-survivors, but the confidence intervals of both mean differences included zero (SI 0.03, 95% CI -0.05 to 0.12; MSI 0.05, 95% CI -0.06 to 0.16), and the standardized effect sizes were small (Cohen's d 0.17 and 0.20). Details are given in Table 1 and Figure 2.

Table 1. Admission characteristics and haemodynamic profile of survivors and non-survivors (n=93)

Characteristic	Survivors (n=29)	Non-survivors (n=64)	Mean difference (95% CI)	Cohen's d	p-value
Age, years	62.2 ± 14.9 [63.0 (51.0–73.0)]	68.9 ± 12.5 [69.5 (60.0–75.5)]	6.70 (0.80 to 12.60)	0.51	0.026 (0.036)
Heart rate, bpm	97.5 ± 15.3 [99.0 (82.0–112.0)]	95.4 ± 13.1 [95.5 (90.0–102.5)]	-2.04 (-8.19 to 4.11)	-0.15	0.511 (0.621)
Systolic BP, mmHg	128.6 ± 16.8 [130.0 (120.0–140.0)]	122.8 ± 20.7 [120.0 (110.0–132.5)]	-5.81 (-14.51 to 2.89)	-0.30	0.188 (0.164)
Diastolic BP, mmHg	79.0 ± 9.0 [80.0 (70.0–80.0)]	75.2 ± 12.2 [75.0 (70.0–82.5)]	-3.81 (-8.84 to 1.23)	-0.34	0.136 (0.169)
Mean arterial pressure, mmHg	95.5 ± 10.6 [100.0 (86.7–103.3)]	91.0 ± 14.2 [90.0 (83.3–103.3)]	-4.48 (-10.35 to 1.40)	-0.34	0.133 (0.167)
Shock Index (HR/SBP)	0.77 ± 0.16 [0.75 (0.67–0.90)]	0.80 ± 0.21 [0.81 (0.68–0.88)]	0.03 (-0.05 to 0.12)	0.17	0.447 (0.481)
Modified Shock Index (HR/MAP)	1.03 ± 0.19 [1.01 (0.88–1.17)]	1.08 ± 0.27 [1.07 (0.90–1.15)]	0.05 (-0.06 to 0.16)	0.20	0.373 (0.483)

Data are mean ± SD [median (interquartile range)]. The mean difference is non-survivors minus survivors with its 95% confidence interval; p-values are from the independent-samples t-test with the Mann–Whitney U test p-value in parentheses. Cohen's d is the standardised mean difference (0.2 small, 0.5 moderate, 0.8 large). The mean difference and Cohen's d quantify group-level separation in interpretable units; they do not measure the ability to classify an individual patient, which is given by the area under the ROC curve in Table 4 (0.546 for both indices). Categorical variables are reported in Table 2.

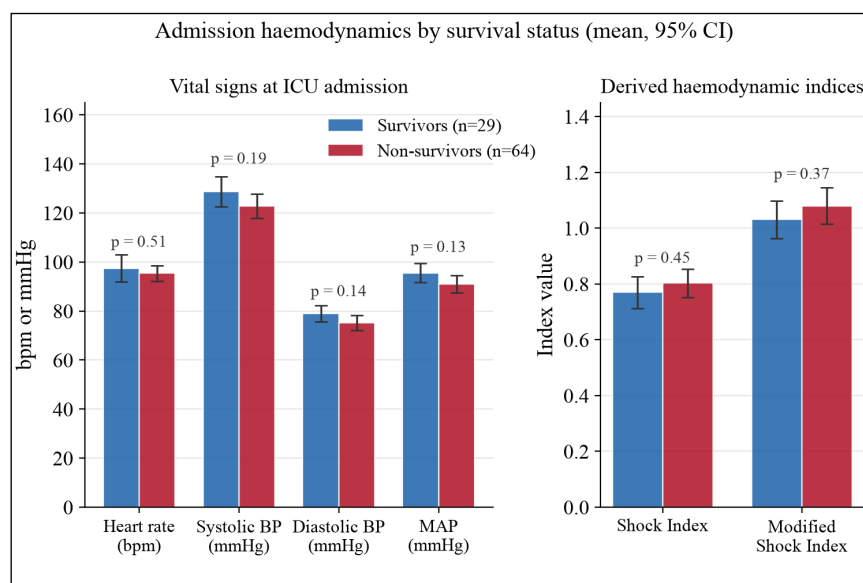


Figure 1. Admission haemodynamics in survivors and non-survivors.

Grouped bar chart of admission vital signs and derived indices (mean with 95% confidence interval) in survivors (blue, n=29) and non-survivors (red, n=64). Non-survivors had numerically lower diastolic and mean arterial pressures and numerically higher SI and MSI, the pattern that motivates use of the MSI, but none of these differences reached statistical significance (all $p \geq 0.13$), and the overlapping

confidence intervals illustrate the substantial physiological overlap between the two outcome groups.

Univariable Associations with In-Hospital Mortality

Univariable associations between demographic factors, comorbidities, and in-hospital mortality are summarized in Table 2, with the relative risk (RR) and the absolute risk difference as the primary effect measures. Age ≥ 70 years

was the only factor significantly associated with mortality (80.0% vs 60.4%; RR 1.33, 95% CI 1.01–1.73; risk difference +19.6 percentage points, 95% CI +1.5 to +37.7; χ^2 $p=0.043$), consistent with age being the strongest predictor in the regression models below. Coronary artery disease carried the highest crude mortality among comorbidities (83.3% vs 66.7%; RR 1.25, 95% CI 0.93–1.68), but with only 12 affected patients, the estimate is imprecise and compatible with no effect (Fisher $p=0.329$). Neither male sex (RR 0.93, 95% CI 0.70–1.23), hypertension (RR 1.10, 95% CI 0.83–1.46), diabetes mellitus (RR 0.97, 95% CI 0.74–1.28), nor chronic kidney disease (RR 1.04, 95% CI 0.64–1.70) was associated with mortality. Because mortality was common in this cohort, odds ratios for these same associations would be considerably further from unity than the corresponding

relative risks—for the abnormal-MSI category, for example, the odds ratio is 4.13, whereas the relative risk is only 1.37—and odds ratios are therefore reported only from the regression models.

Relative risks with 95% confidence intervals for baseline factors and for each Shock Index and Modified Shock Index threshold are plotted on a logarithmic scale; the dashed line marks a relative risk of 1.0 (no association), and intervals excluding 1.0 are highlighted in red. Relative risks rather than odds ratios are displayed because mortality was common in this cohort. Only age ≥ 70 years (RR 1.33) and the composite abnormal-MSI category (RR 1.37) had confidence intervals that excluded unity, and both were borderline on exact testing; all remaining factors, including every SI threshold, were compatible with no association.

Table 2. Univariable association of demographic factors and comorbidities with in-hospital mortality

Risk factor	Patients, n (%)	Mortality with vs without factor (%)	Relative risk (95% CI)	Risk difference, pp (95% CI)	p-value
Age ≥ 70 years	40 (43.0)	80.0 vs 60.4	1.33 (1.01–1.73)	+19.6 (+1.5 to +37.7)	0.043
Male sex	64 (68.8)	67.2 vs 72.4	0.93 (0.70–1.23)	-5.2 (-25.1 to +14.7)	0.614
Hypertension	53 (57.0)	71.7 vs 65.0	1.10 (0.83–1.46)	+6.7 (-12.4 to +25.8)	0.490
Diabetes mellitus	53 (57.0)	67.9 vs 70.0	0.97 (0.74–1.28)	-2.1 (-21.0 to +16.9)	0.831
Coronary artery disease	12 (12.9)	83.3 vs 66.7	1.25 (0.93–1.68)	+16.7 (-6.8 to +40.1)	0.329*
Chronic kidney disease	7 (7.5)	71.4 vs 68.6	1.04 (0.64–1.70)	+2.8 (-32.1 to +37.7)	1.000*

pp, percentage points. The relative risk and risk difference are reported as the primary effect measures because in-hospital mortality was common (68.8%), a setting in which the odds ratio systematically overestimates the relative risk and is easily misread as a risk multiplier; odds ratios for the indices are reported only within the logistic regression models (Table 4). Mortality is shown for patients with versus without the factor. p-values are from Pearson's χ^2 test, except for coronary artery disease and chronic kidney disease, where expected cell counts were below five and Fisher's exact test was used. For age ≥ 70 years, Fisher's exact test gives $p=0.070$ against χ^2 $p=0.043$; both are reported for transparency and the result is interpreted as borderline.

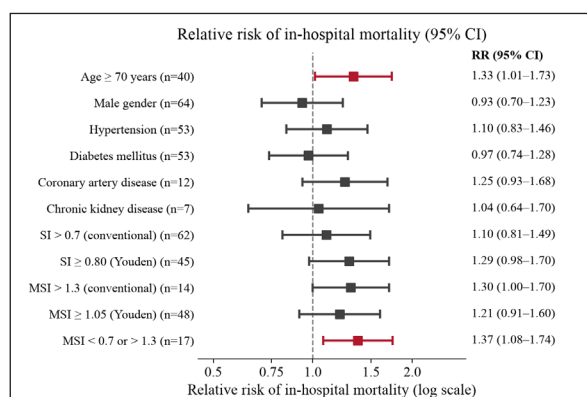


Figure 2. Forest plot of relative risks of in-hospital mortality

Distribution of Shock Index Categories and Crude Mortality

The distribution of both indices across conventional and data-derived categories, together with the corresponding crude mortality and relative risks, is presented in a single table (Table 3).

An SI > 0.7 was present in 66.7% (62/93) of patients, among whom mortality was 71.0% compared with 64.5% in patients with an SI ≤ 0.7 (RR 1.10, 95% CI 0.81–1.49; Fisher p = 0.636). The Youden-optimal cut-off derived from these data, SI ≥ 0.80, was present in 48.4% (45/93) and separated the groups slightly better (mortality 77.8% vs 60.4%; RR 1.29, 95% CI 0.98–1.70; Fisher p=0.079), although this threshold is optimistically biased because it was selected within the same sample.

An MSI >1.3 was present in 15.1% (14/93) of patients (mortality 85.7% vs 65.8%; RR 1.30, 95% CI 1.00–1.70; Fisher

p=0.212), and the composite abnormal MSI category (<0.7 or >1.3) in 18.3% (17/93), among whom mortality was 88.2% compared with 64.5% in patients with a normal MSI (RR 1.37, 95% CI 1.08–1.74; risk difference +23.8 percentage points, 95% CI +5.0 to +42.5). Two features of this composite category require emphasis. First, only three patients had an MSI < 0.7 (all three died), so the category is driven almost entirely by the high-MSI group, and a U-shaped association cannot be supported by three observations. Second, mortality did not increase monotonically across MSI quartiles (66.7%, 60.9%, 87.0%, and 60.9% from the lowest to the highest quartile; the corresponding figures for SI were 66.7%, 56.5%, 87.0%, and 65.2%), the peak occurring in the third quartile rather than at the extremes. The absence of a graded dose–response relationship indicates that the apparent risk attached to abnormal values reflects the position of the cut-points in this small sample rather than a monotonic association between the index and mortality.

Table 3. Distribution of Shock Index and Modified Shock Index categories and crude in-hospital mortality

Index category	Patients, n (%)	Non-survivors, n (%)	Survivors, n (%)	Relative risk (95% CI)	p-value
Shock Index					
SI ≤ 0.7 (reference)	31 (33.3)	20 (64.5)	11 (35.5)	1.00 (reference)	–
SI > 0.7 (conventional cut-off)	62 (66.7)	44 (71.0)	18 (29.0)	1.10 (0.81–1.49)	0.636
SI < 0.80 (reference)	48 (51.6)	29 (60.4)	19 (39.6)	1.00 (reference)	–
SI ≥ 0.80 (Youden-optimal)	45 (48.4)	35 (77.8)	10 (22.2)	1.29 (0.98–1.70)	0.079
Modified Shock Index					
MSI ≤ 1.3 (reference)	79 (84.9)	52 (65.8)	27 (34.2)	1.00 (reference)	–
MSI > 1.3 (conventional cut-off)	14 (15.1)	12 (85.7)	2 (14.3)	1.30 (1.00–1.70)	0.212
MSI < 1.045 (reference)	45 (48.4)	28 (62.2)	17 (37.8)	1.00 (reference)	–
MSI ≥ 1.045 (Youden-optimal)	48 (51.6)	36 (75.0)	12 (25.0)	1.21 (0.91–1.60)	0.263
MSI 0.7–1.3 (normal, reference)	76 (81.7)	49 (64.5)	27 (35.5)	1.00 (reference)	–
MSI < 0.7 or > 1.3 (abnormal)	17 (18.3)	15 (88.2)	2 (11.8)	1.37 (1.08–1.74)	0.081

Percentages in the second column are of the whole cohort; percentages for non-survivors and survivors are within-category (row) percentages. Each relative risk compares the category with the reference category immediately above it. p-values are from Fisher's exact test. Conventional and Youden-optimal cut-offs for both indices are shown together so that pre-existing and data-derived thresholds can be compared directly; the corresponding χ^2 , continuity-corrected χ^2 and odds-ratio estimates for the abnormal-MSI category are reported in the text and in Table 4.

Discrimination and Head-to-Head Comparison of the Two Indices

Receiver-operating-characteristic analysis (Figure 3) showed that neither index discriminated survivors from non-survivors. The area under the curve was 0.546 (95% CI 0.415–0.676; p=0.492) for SI and 0.546 (95% CI 0.417–0.674;

p=0.489) for MSI, and both confidence intervals comfortably included the no-discrimination value of 0.50. The two indices were almost perfectly correlated ($r=0.962$), and their discriminative ability was statistically indistinguishable: the paired DeLong difference in AUC (MSI minus SI) was -0.0003 (95% CI -0.036 to +0.035; p=0.988), a result confirmed by bootstrap resampling (95% CI -0.039 to +0.038). Given the

precision of this cohort, a true between-index difference of 0.051 AUC units or more would have been detected, so a clinically meaningful advantage of MSI over SI can be excluded, whereas a small advantage cannot. For context, age discriminated better than either index (AUC 0.636, 95% CI 0.507–0.765; $p=0.039$) and was the only variable with an AUC significantly above 0.50; the AUCs for heart rate (0.468), systolic (0.589), and diastolic blood pressure (0.586) were all non-significant.

The effect measures for the abnormal-MSI category should be read in this context and are interpreted here rather than merely tabulated. The relative risk of 1.37 (95% CI 1.08–1.74) means that patients with an abnormal MSI were, on average, 37% more likely to die than patients with a normal MSI, which on the absolute scale corresponds to mortality rising from 64.5% to 88.2%, a risk difference of 23.8 percentage points (95% CI 5.0 to 42.5) or approximately one additional death for every four patients in this category. The odds ratio of 4.13 describes the same association on the odds scale and is far larger than the relative risk only because mortality was common (68.8%); it must not be read as a four-fold increase in the risk of death. Because one cell of this table contained only two patients, the estimate is imprecise, and the choice of test materially affects the inference. The Pearson χ^2 for this table is 3.66 ($p=0.056$), the continuity-corrected χ^2 is 2.63 ($p=0.105$), and Fisher's exact test gives $p=0.081$, while the odds ratio is 4.13 with a Wald 95% CI of 0.88–19.44 and a profile-likelihood 95% CI of 1.06–27.47. Following the pre-specified rule for sparse tables, Fisher's exact test is taken as the primary inference, so the association between an abnormal MSI and mortality is best described as a borderline and imprecise signal that does not reach conventional statistical significance.

Threshold Performance and Consolidated Head-to-Head Comparison

Performance at both the conventional and the Youden-optimal cut-offs is presented together with the discrimination and regression results in a single consolidated table (Table 4). At the conventional cut-off, SI >0.7 behaved as a broad and non-specific screen (sensitivity 68.8%, 95% CI 56.6–

78.8; specificity 37.9%, 95% CI 22.7–56.0; accuracy 59.1%), whereas an abnormal MSI was specific but insensitive (sensitivity 23.4%, 95% CI 14.8–35.1; specificity 93.1%, 95% CI 78.0–98.1; positive predictive value 88.2%, 95% CI 65.7–96.7). The high positive predictive value is largely a consequence of the 68.8% baseline mortality of the cohort rather than of the test itself, and the corresponding positive likelihood ratio was modest and very imprecise (3.40, 95% CI 0.83–13.90), with a confidence interval that includes 1.0; the negative likelihood ratio of 0.82 (95% CI 0.70–0.97) shows that a normal MSI does not usefully lower the probability of death. At the Youden-optimal cut-offs, the two indices performed almost identically (SI ≥ 0.80 : sensitivity 54.7%, specificity 65.5%, Youden J 0.20; MSI ≥ 1.05 : sensitivity 56.3%, specificity 58.6%, Youden J 0.15), with accuracies of 58.1% and 57.0% against a no-information rate of 68.8%. Neither index therefore classified patients more accurately than predicting death for every patient, which is the central practical finding of this analysis.

In logistic regression, neither index was associated with mortality per 0.1-unit increase, either unadjusted (SI: odds ratio 1.10, profile-likelihood 95% CI 0.87–1.45, $p=0.445$; MSI: 1.10, 95% CI 0.91–1.32, $p=0.372$) or after adjustment for age and sex (SI: adjusted odds ratio 1.16, 95% CI 0.89–1.51, $p=0.270$; MSI: 1.14, 95% CI 0.92–1.41, $p=0.236$). Age was the only independent predictor of mortality in both adjusted models (adjusted odds ratio 1.04 per year, 95% CI 1.01–1.08; $p=0.023$ and $p=0.024$), while male sex was not (adjusted odds ratio 0.81 and 0.77; $p=0.674$ and $p=0.620$). When both indices were entered simultaneously, neither retained an independent association (likelihood-ratio $p=0.578$), as expected from their near-collinearity ($r=0.962$). An age- and sex-based model achieved an AUC of 0.642 with a Brier score of 0.202; adding SI or MSI changed these to 0.657 (Brier 0.200) and 0.662 (Brier 0.200), an incremental discriminative gain of only 0.015 and 0.020 AUC units, and neither three-variable model was significantly better than the null model (likelihood-ratio $p=0.089$ and $p=0.080$). Explained variation was negligible for both indices (Nagelkerke R^2 0.009 for SI and 0.013 for MSI).

Table 4. Consolidated head-to-head prognostic performance of the Shock Index and the Modified Shock Index for in-hospital mortality

Metric	Shock Index (SI)	Modified Shock Index (MSI)
Discrimination (whole range of values)		
AUC (95% CI)	0.546 (0.415–0.676)	0.546 (0.417–0.674)
p-value vs AUC 0.50	0.492	0.489
Δ AUC (MSI minus SI) (95% CI)	Reference	-0.0003 (-0.036 to +0.035)
DeLong p-value for Δ AUC	–	0.988

Metric	Shock Index (SI)	Modified Shock Index (MSI)
Correlation between the two indices	r = 0.962	r = 0.962
Minimum Δ AUC detectable with 80% power	0.051	0.051
Performance at Youden-optimal cut-off (derived from these data)		
Optimal cut-off	≥ 0.80	≥ 1.045
Sensitivity, % (95% CI)	54.7 (42.6–66.3)	56.3 (44.1–67.7)
Specificity, % (95% CI)	65.5 (47.3–80.1)	58.6 (40.7–74.5)
PPV, % (95% CI)	77.8 (63.7–87.5)	75.0 (61.2–85.1)
NPV, % (95% CI)	39.6 (27.0–53.7)	37.8 (25.1–52.4)
LR+ (95% CI)	1.59 (0.92–2.75)	1.36 (0.84–2.21)
LR- (95% CI)	0.69 (0.47–1.01)	0.75 (0.49–1.13)
Accuracy, % (95% CI); no-information rate 68.8%	58.1 (47.9–67.6)	57.0 (46.8–66.6)
Youden index J	0.20	0.15
RR (95% CI)	1.29 (0.98–1.70)	1.21 (0.91–1.60)
Performance at conventional cut-off		
Conventional cut-off	> 0.7	< 0.7 or > 1.3 (abnormal)
Patients above cut-off, n (%)	62 (66.7)	17 (18.3)
Sensitivity, % (95% CI)	68.8 (56.6–78.8)	23.4 (14.8–35.1)
Specificity, % (95% CI)	37.9 (22.7–56.0)	93.1 (78.0–98.1)
PPV, % (95% CI)	71.0 (58.7–80.8)	88.2 (65.7–96.7)
NPV, % (95% CI)	35.5 (21.1–53.1)	35.5 (25.7–46.7)
LR+ (95% CI)	1.11 (0.80–1.54)	3.40 (0.83–13.90)
LR- (95% CI)	0.82 (0.46–1.49)	0.82 (0.70–0.97)
Accuracy, % (95% CI)	59.1 (49.0–68.6)	45.2 (35.4–55.3)
Confusion matrix TP/FP/FN/TN	44/18/20/11	15/2/49/27
RR (95% CI); risk difference, pp	1.10 (0.81–1.49); +6.5	1.37 (1.08–1.74); +23.8
Odds ratio (Wald 95% CI; profile-likelihood 95% CI)	1.34 (0.54–3.37; 0.53–3.35)	4.13 (0.88–19.44; 1.06–27.47)
p-value (Fisher's exact; Pearson χ^2)	0.636; 0.527	0.081; 0.056
Regression and incremental value (per 0.1-unit increase)		
Unadjusted OR (profile-likelihood 95% CI)	1.10 (0.87–1.45)	1.10 (0.91–1.32)
Adjusted OR for age and sex (95% CI)	1.16 (0.89–1.51)	1.14 (0.92–1.41)
p-value (unadjusted; adjusted)	0.445; 0.270	0.372; 0.236
Nagelkerke R ² (univariable)	0.009	0.013
AUC (Brier score) of age + sex model with index	0.657 (0.200)	0.662 (0.200)
Δ AUC vs age + sex alone (AUC 0.642, Brier 0.202)	+0.015	+0.020

AUC, area under the receiver-operating-characteristic curve; LR+/LR-, positive/negative likelihood ratio; PPV/NPV, positive/negative predictive value; TP/FP/FN/TN, true positive/false positive/false negative/true negative; RR, relative risk. Confidence intervals are Wilson intervals for proportions, log intervals for likelihood ratios and DeLong intervals for AUCs. The Youden-optimal cut-offs were derived from these data, so the corresponding performance is apparent (optimistically biased) rather than externally validated. Odds ratios for the conventional

cut-offs are given with both Wald and profile-likelihood intervals because one cell of the abnormal-MSI table contains only two patients; the profile-likelihood interval and Fisher's exact test are preferred for that table. The no-information rate, that is, the accuracy achieved by predicting death for every patient, is 68.8% and exceeds the accuracy of both indices at every cut-off examined. This table provides the complete cut-off, discrimination, classification and effect-size comparison of the two indices.

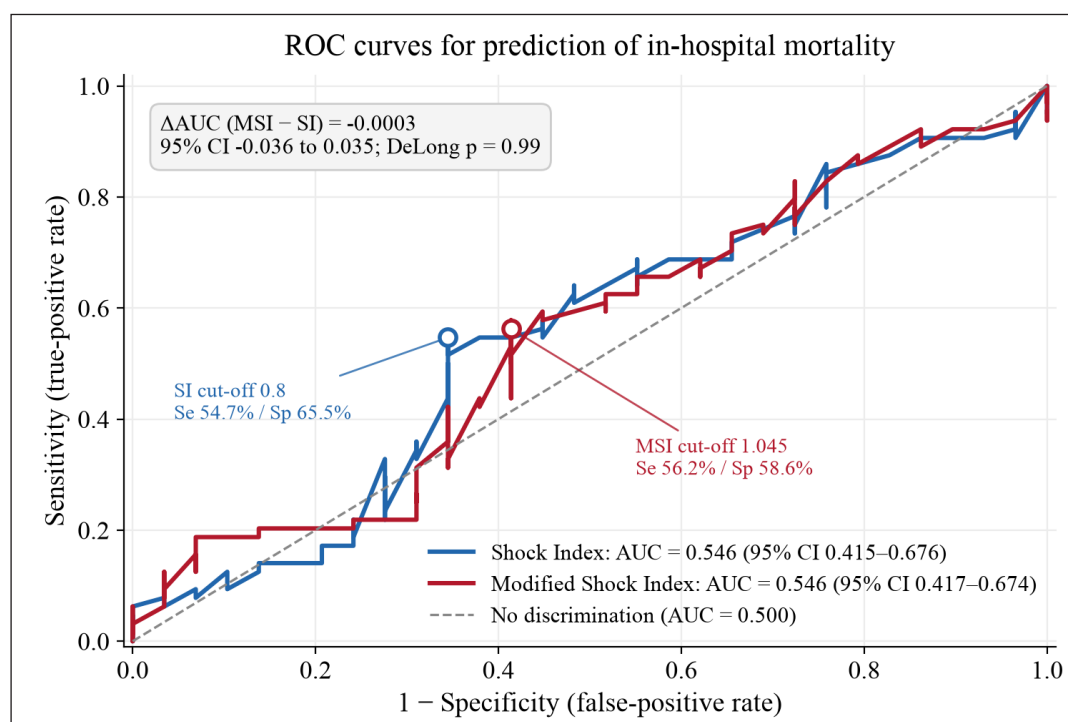


Figure 3. Receiver-operating-characteristic curves for the Shock Index and the Modified Shock Index

The complete ROC curves for both indices are shown in Figure 3. The two curves are superimposed over most of their length and lie close to the diagonal, which is the graphical counterpart of the near-zero difference in AUC, and the Youden-optimal operating points of the two indices lie only marginally above the line of no discrimination.

ROC curves for prediction of in-hospital mortality from the admission Shock Index (blue) and Modified Shock Index (red), with the line of no discrimination shown as a grey dashed diagonal. The areas under the curve were 0.546 (95% CI 0.415–0.676) and 0.546 (95% CI 0.417–0.674), respectively, and the paired DeLong difference was -0.0003 (95% CI -0.036 to +0.035; $p=0.988$). Open circles mark the Youden-optimal operating points (SI ≥ 0.80 : sensitivity 54.7%, specificity 65.5%; MSI ≥ 1.05 : sensitivity 56.3%, specificity 58.6%).

Discussion

The rapid clinical deterioration frequently observed in severe COVID-19 has motivated the search for accessible, non-invasive bedside tools that can identify patients at risk of death before irreversible shock develops. Both the Shock Index and the Modified Shock Index have been proposed for this purpose, and MSI has been assumed

to be the better discriminator because it incorporates diastolic pressure through the mean arterial pressure. In this cohort of 93 critically ill patients that assumption was not supported. The two indices had essentially identical and poor discrimination for in-hospital mortality (AUC 0.546 for both; difference -0.0003, $p=0.988$); neither was associated with mortality in adjusted regression models, and neither improved meaningfully on age alone.¹⁴ The principal message of this analysis is therefore a negative one, and it is reported as such because the head-to-head design, the formal statistical comparison of the two AUCs and the complete enumeration of eligible patients make the absence of a difference informative rather than merely inconclusive.

The physiological data explain why. Non-survivors did show the pattern that motivates the use of MSI, with numerically lower diastolic (75.2 vs 79.0 mmHg) and mean arterial (91.0 vs 95.5 mmHg) pressures and numerically higher indices, but these differences were small (Cohen's d 0.17 to 0.34), were not statistically significant, and were dwarfed by the within-group spread. In a cohort in which every patient was already critically ill and 68.8% died, admission haemodynamics occupied a narrow range, and

a ratio constructed from two heavily overlapping quantities cannot separate outcomes that the underlying vital signs themselves do not separate.^{15,16} The near-perfect correlation between SI and MSI ($r=0.962$) places a further mathematical ceiling on any possible divergence: when two markers share more than 92% of their variance, neither can substantially outperform the other, which is exactly what the paired DeLong comparison demonstrated. The mean-difference analysis and the discrimination analysis therefore answer different questions, and the modest group-level differences observed here carry no individual-level predictive value.

Our findings sit alongside a growing body of literature on haemodynamic indices in COVID-19, summarised in Table 5. A 2023 systematic review and meta-analysis by Alsagaff et al. established that elevated shock indices are generally correlated with poor outcomes in COVID-19 pneumonia¹⁷,

and several single-centre studies have reported moderate to excellent discrimination for SI, MSI or both. Our results are discordant with those reports, and the discordance is informative rather than contradictory: the studies reporting good discrimination were almost all conducted in emergency departments or mixed ward populations with mortality between 10% and 20%, where a wide spectrum of illness severity allows a simple vital-sign ratio to separate the few patients who die from the many who recover.^x Our cohort was restricted to ICU patients with 68.8% mortality, which truncates that spectrum, and spectrum effects of this kind are a well-recognised reason for prognostic markers to lose apparent accuracy when moved to a sicker, more homogeneous population. A U-shaped mortality pattern was likewise not evident in these data: only three patients had an MSI below 0.7, and mortality across MSI quartiles was non-monotonic, so such an interpretation is not supported.^{19,20}

Table 5. Comparison of the current study with key published studies on Shock Index-based mortality prediction in COVID-19 (2020–2025)

Study (Year)	Population / Setting	N	Approx. mortality	Key findings for Shock Index (SI)	Key findings for Modified Shock Index (MSI)	Overall conclusion vs current study
Current study (2025)	ICU, critically ill COVID-19, single tertiary center	93	68.8%	SI > 0.7 in 66.7%; mortality 71.0% vs 64.5% (RR 1.10, 95% CI 0.81–1.49); AUC 0.546 (95% CI 0.415–0.676), not different from chance	Abnormal MSI (< 0.7 or > 1.3) in 18.3%; mortality 88.2% vs 64.5% (RR 1.37, Fisher $p=0.081$); AUC 0.546 (95% CI 0.417–0.674), not different from chance	In a high-mortality ICU cohort, neither index discriminated survivors from non-survivors and MSI was not superior to SI ($\Delta AUC -0.0003$, $p=0.988$); age was the only independent predictor
Doğanay et al., 2025	Emergency department COVID-19 admissions; composite indices including SI	~500	~14–20%	SI alone showed moderate prognostic value but was inferior to composite indices (e.g. age-adjusted SI, SI combined with oxygen saturation); higher SI cut-offs associated with ICU need and mortality.	MSI not directly isolated; focus on combined indices that integrate SI with age, lactate or saturation; these combinations improved AUC vs SI alone.	Supports the idea that simple SI can be strengthened by adding diastolic or contextual variables; consistent with current study showing that adding MAP (MSI) improves discrimination.

Avci et al., 2022 (Prognostic Performance of SI, DBP, MAP)	Hospitalized COVID-19 pneumonia (ward + ICU), single center	993	~15–20%	SI demonstrated good prognostic performance for mortality and ICU transfer (AUC around 0.77); optimal cut-off near 0.9; SI outperformed several single vital signs.	MSI also predictive (AUC around 0.76) but slightly lower than SI; SI had higher specificity at optimized thresholds.	In a broader, less severe cohort, SI performed slightly better than MSI; contrasts with our ICU-only cohort, where MSI outperformed SI—likely due to greater role of diastolic failure in critical illness.
El-Hady et al., 2021	Critically ill COVID-19 in ICU	~150–200	High	SI strongly associated with mortality; very high AUC (≈ 0.98) for predicting in-hospital death at cut-off around 0.99.	MSI also highly predictive (AUC ≈ 0.96) but marginally inferior to SI; both indices excellent but SI slightly favored.	Both SI and MSI powerful in their cohort; unlike our data, they found SI marginally superior—possible differences in case mix, blood pressure patterns, and timing of measurements.
Kurt et al., 2021 (Usefulness of SI and MSI)	Hospitalized COVID-19 adults, Turkey	~300	Moderate	Elevated SI associated with ICU admission and mortality, but cut-offs required recalibration in elderly and comorbid patients.	MSI > 1.3 identified patients at particularly high risk; MSI provided better risk stratification in some subgroups than SI alone.	Partially aligns with our findings: high MSI behaves as a strong “red flag” for poor outcome, supporting the use of MSI thresholds to identify very high-risk patients.
Comparison of early mortality prediction methods in COVID-19 (SCIE-26539)	ED and ward COVID-19 cases; compared SI with NEWS, qSOFA, etc.	~400	~10–15%	SI alone less accurate than composite scores (NEWS, qSOFA) for early mortality prediction; modest AUC values.	MSI not consistently superior; index-based tools underperformed full clinical scores.	Reinforces that SI/MSI are simple, quick tools but should complement—not replace—broader clinical scoring; our study focuses on their role as bedside indices in ICU rather than full prognostic models.

Our findings diverge from several prior reports, and this variation illuminates important nuances about haemodynamic indices in COVID-19. In our cohort of 93 critically ill ICU patients (68.8% mortality), neither MSI nor SI predicted mortality, and the two indices were statistically indistinguishable, whereas several larger studies published between 2021 and 2025 reported useful, if inconsistent, prognostic performance. The direction of the discrepancy

differs between studies, which is itself informative: some found SI slightly better, others MSI, and our data show no difference at all. Taken together, this pattern is more consistent with small, case-mix-dependent fluctuations around a genuinely modest signal than with a consistent advantage of either index.

Doganay and colleagues (2025), analysing approximately 500 emergency department COVID-19 admissions with

much lower baseline mortality (~14–20%), found that SI alone performed modestly but that composite indices combining SI with age, oxygen saturation or the reverse shock index substantially improved prognostic accuracy. Our data are consistent with the first part of that observation and extend it: in a critically ill population, neither simple index carried usable prognostic information, and the only variable that discriminated at all was age (AUC 0.636), which reinforces the conclusion that vital-sign ratios need to be combined with demographic and physiological context before they can be used for triage.²¹

Avci et al. (2022) examined a larger, more heterogeneous inpatient cohort (n=993) and found SI slightly superior to MSI, with AUCs around 0.76, while El-Hady et al. (2021) reported very high discrimination for both indices (AUC >0.95) with a marginal advantage for SI. Neither result was reproduced here. The most plausible explanation is the difference in case mix and outcome prevalence noted above, together with the fact that AUCs close to 0.95 for two crude vital-sign ratios are difficult to reconcile with the physiology of critical illness and may reflect population-specific or methodological factors.²² Our study was powered to detect a between-index difference of 0.051 AUC units, so the absence of any difference in our data is unlikely to be a power problem for the comparative question, even though the study is underpowered to characterise the small absolute performance of each index.

Kurt et al. (2021) reported that an MSI >1.3 identified patients at particularly high risk. In our cohort, patients with an MSI >1.3 did have higher crude mortality (85.7% vs 65.8%), but the association was imprecise and not statistically significant (RR 1.30, 95% CI 1.00–1.70; Fisher p=0.212), and this threshold identified only 14 patients. Overall, these comparisons suggest that the apparent utility of SI and MSI is strongly context-dependent, being greatest in populations with a wide spectrum of illness severity and low to moderate mortality and negligible in an ICU cohort in which haemodynamic derangement is nearly universal.^{23,24}

The relationship between comorbidities and outcome was weak in this cohort. Patients with coronary artery disease had the highest crude mortality (83.3% vs 66.7%), which is biologically plausible given their limited myocardial reserve under hypoxic and inflammatory stress, but with only 12 such patients the estimate was imprecise and compatible with no association (RR 1.25, 95% CI 0.93–1.68; Fisher p=0.329). Mortality was also numerically higher in women (72.4% vs 67.2%; RR 0.93 for male sex, 95% CI 0.70–1.23), but this difference was not statistically significant and should not be over-interpreted in a sample of this size. Age ≥ 70 years was the only baseline factor associated with mortality, and age remained the only independent predictor after adjustment, underscoring that in this population

demographic rather than haemodynamic variables carried the prognostic signal.²⁵

Limitation

Several limitations warrant consideration. First, this was a single-centre retrospective analysis of a fixed cohort of 93 patients. As quantified in the Methods, this sample provides only ±0.13 precision around an AUC estimate and 80% power to detect an AUC of 0.675 or higher, so the study can exclude strong prognostic performance and a between-index difference above 0.05 AUC units but cannot exclude a small degree of prognostic utility for either index; the analysis is exploratory and hypothesis-generating. Second, the small number of survivors (n=29) limited regression models to three covariates, so adjustment for illness severity, oxygenation, inflammatory markers, vasoactive or ventilatory support and treatment era was not possible, and residual confounding cannot be excluded; established severity scores such as APACHE II or SOFA were not available in the records and could not be used as comparators. Third, the indices were computed from a single set of admission vital signs, whereas haemodynamics are dynamic and serial or trend-based measurements that might carry information that a snapshot does not. Fourth, the cohort was restricted to ICU patients with 68.8% mortality, so the narrow spectrum of illness severity limits generalisability and probably contributes to the poor discrimination observed; these results should not be extrapolated to emergency department or ward populations. Fifth, the Youden-optimal cut-offs were derived from the same data in which they were evaluated and are therefore optimistically biased and in need of external validation. Finally, the cohort was assembled during a specific pandemic wave, when case mix, treatment protocols and critical-care capacity differed from current practice, so the baseline mortality observed here may not apply to contemporary populations.

Conclusion

In this single-centre cohort of critically ill patients with COVID-19, neither the Shock Index nor the Modified Shock Index discriminated between survivors and non-survivors, and the two indices performed identically in a formal head-to-head comparison (AUC 0.546 for both; difference -0.0003, 95% CI -0.036 to +0.035; p=0.988). These data therefore do not support the assumption that MSI is a more robust predictor of in-hospital mortality than SI. An abnormal MSI was highly specific but insensitive, and its apparently high positive predictive value largely reflects the 68.8% baseline mortality of the cohort rather than the performance of the index; neither index classified patients more accurately than predicting death for every patient, and age carried more prognostic information than either. Admission SI and MSI should not be used alone to prognosticate or triage critically ill patients with COVID-19. Given the exploratory nature

and limited power of this analysis, adequately powered multicentre studies using serial measurements, external validation of any derived cut-off and assessment of these indices within multivariable models that include age and established severity scores are required before their role, if any, in critical care triage can be defined.

Source of Funding: The authors have nothing to report.

Ethical Statement: The study was conducted after obtaining approval from the Institutional Ethics Committee for Student Projects (UG and Non-Medical PG Students), Sri Ramachandra Institute of Higher Education and Research (IEC approval number: CSP/21/APR/93/316; date of approval: 30 April 2021). A copy of the Institutional Ethics Committee approval certificate is submitted with this manuscript as Supplementary File 1. The requirement for individual informed consent was waived by the Committee because the study involved only retrospective analysis of de-identified hospital records.

Conflict of Interest: None

Reference

- Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, Qiu Y, Wang J, Liu Y, Wei Y, Xia JA. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. *The lancet*. 2020 Feb 15;395(10223):507-13. [Google Scholar]
- Lu H, Stratton CW, Tang YW. Outbreak of pneumonia of unknown etiology in Wuhan, China: The mystery and the miracle. *Journal of medical virology*. 2020 Feb 12;92(4):401. [Google Scholar]
- Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, Zhao X, Huang B, Shi W, Lu R, Niu P. A novel coronavirus from patients with pneumonia in China, 2019. *New England journal of medicine*. 2020 Feb 20;382(8):727-33. [Google Scholar]
- Allgöwer M, Burri C. Schockindex. *DMW-Deutsche Medizinische Wochenschrift*. 1967 Oct;92(43):1947-50. [Google Scholar]
- Cerović O, Golubović V, Špec-Marn A, Kremžar B, Vidmar G. Relationship between injury severity and lactate levels in severely injured patients. *Intensive care medicine*. 2003 Aug;29(8):1300-5. [Google Scholar]
- Mutschler M, Nienaber U, Münzberg M, Wölfl C, Schoechl H, Paffrath T, Bouillon B, Maegele M, TraumaRegister DGU®. The Shock Index revisited—a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the TraumaRegister DGU®. *Critical care*. 2013 Aug 12;17(4):R172. [Google Scholar]
- Birkhahn RH, Gaeta TJ, Van Deusen SK, Tlozkowski J. The ability of traditional vital signs and shock index to identify ruptured ectopic pregnancy. *American journal of obstetrics and gynecology*. 2003 Nov 1;189(5):1293-6. [Google Scholar]
- Rady MY, Smithline HA, Blake H, Nowak R, Rivers E. A comparison of the shock index and conventional vital signs to identify acute, critical illness in the emergency department. *Ann Emerg Med*. 1994;24(4):685-90. [Google Scholar]
- Cannon CM, Braxton CC, Kling-Smith M, Mahnken JD, Carlton E, Moncure M. Utility of the shock index in predicting mortality in traumatically injured patients. *Journal of Trauma and Acute Care Surgery*. 2009 Dec 1;67(6):1426-30. [Google Scholar]
- Talmor D, Jones AE, Robinson L, Howell MD, Shapiro NI. Simple triage scoring system predicting death and the need for critical care resources for use during epidemics. *Critical care medicine*. 2007 May 1;35(5):1251-6. [Google Scholar]
- Liu YC, Liu JH, Fang ZA, Shan GL, Xu J, Qi ZW, Zhu HD, Wang Z, Yu XZ. Modified shock index and mortality rate of emergency patients. *World journal of emergency medicine*. 2012;3(2):114. [Google Scholar]
- Singh A, Ali S, Agarwal A, Srivastava RN. Correlation of shock index and modified shock index with the outcome of adult trauma patients: a prospective study of 9860 patients. *North American journal of medical sciences*. 2014 Sep;6(9):450. [Google Scholar]
- Shoemaker WC. Relation of oxygen transport patterns to the pathophysiology and therapy of shock states. *Intensive care medicine*. 1987 Jan 1;13(4):230-43. [Google Scholar]
- Koch E, Lovett S, Nghiem T, Riggs RA, Rech MA. Shock index in the emergency department: utility and limitations. *Open Access Emergency Medicine*. 2019 Aug 14;179-99. [Google Scholar]
- Kristensen AK, Holler JG, Hallas J, Lassen A, Shapiro NI. Is shock index a valid predictor of mortality in emergency department patients with hypertension, diabetes, high age, or receipt of β - or calcium channel blockers?. *Annals of emergency medicine*. 2016 Jan 1;67(1):106-13. [Google Scholar]
- Birkhahn RH, Gaeta TJ, Terry D, Bove JJ, Tlozkowski J. Shock index in diagnosing early acute hypovolemia. *The American journal of emergency medicine*. 2005 May 1;23(3):323-6. [Google Scholar]
- Torabi MM, Mirafzal MA, Rastegari A, Sadeghkhan N. Association of triage time shock index, modified shock index, and age shock index with mortality in emergency severity index level 2 patients. *The American journal of emergency medicine*. 2016 Jan 1;34(1):63-8. [Google Scholar]
- Sobrinho J, Shafi S. Timing and causes of death after injuries. In *Baylor University Medical Center Proceedings*

- 2013 Apr 1 (Vol. 26, No. 2, pp. 120-123). Taylor & Francis. [Google Scholar]
19. Vandromme MJ, Griffin RL, Kerby JD, McGwin Jr G, Rue III LW, Weinberg JA. Identifying risk for massive transfusion in the relatively normotensive patient: utility of the prehospital shock index. *Journal of Trauma and Acute Care Surgery*. 2011 Feb 1;70(2):384-90. [Google Scholar]
 20. Mutschler M, Nienaber U, Münzberg M, Wölfl C, Schoechl H, Paffrath T, Bouillon B, Maegele M, TraumaRegister DGU®. The Shock Index revisited—a fast guide to transfusion requirement? A retrospective analysis on 21,853 patients derived from the TraumaRegister DGU®. *Critical care*. 2013 Aug 12;17(4):R172. [Google Scholar]
 21. Torabi M, Noroozpour A, Bafghi NN, Mirzaee M. Prognostic value of novel indices combining Shock Index, Reverse Shock Index, age, and oxygen saturation for predicting mortality in COVID-19 patients in Iran at emergency department triage: a cross-sectional study. *Acute and Critical Care*. 2025 Aug 29;40(3):425-34. [Google Scholar]
 22. Avci M, Doganay F. Prognostic performance of shock index, diastolic shock index, age shock index, and modified shock index in COVID-19 pneumonia. *Disaster Medicine and Public Health Preparedness*. 2023 Jan;17:e189. [Google Scholar]
 23. Kurt E, Bahadirli S. The usefulness of shock index and modified shock index in predicting the outcome of COVID-19 patients. *Disaster Medicine and Public Health Preparedness*. 2022 Aug;16(4):1558-63. [Google Scholar]
 24. Elhady MM, Elhady AM, Shohieb S. Revolutionizing recovery: The impact of designed digital care therapy on preventing Post-Intensive care syndrome. *Intensive and Critical Care Nursing*. 2025 Dec 1;91:104208. [Google Scholar]
 25. Hökenek UD, Kart JS, Alışkan H, Yilmaz E, Arslan G, Saracoglu K. Comparison of Early Mortality Prediction Methods for COVID-19 Patients Admitted to the Intensive Care Unit. *Southern Clinics of Istanbul Eurasia*. 2022 Sep 1;33(3):219. [Google Scholar]