Bangladesh Crit Care J September 2026; 14 (2): 74-83

Original Article

Effectiveness of the Modified Shock Index as a Predictor of Post-intubation Hypotension

Mohammad Abbas Uddin Ahmed[1], Md. Tariqul Islam[2], Mohammad Ibrahim Khalil[3], Fahmida Sharmin[4]

DOI: https://doi.org/10.3329/bccj.v14i2.94146

Abstract:

Background: *Emergency intubation in critically ill patients may cause post-intubation hypotension, impairing organ perfusion and increasing mortality. Early risk identification is therefore important. The Modified Shock Index (MSI), calculated as heart rate divided by mean arterial pressure, offers a physiologically plausible alternative to the conventional Shock Index (SI).*

Objective: *To compare MSI and SI for predicting post-intubation hypotension in adult critically ill patients undergoing emergency intubation in the ICU.*

Methods: *This prospective observational study enrolled 80 haemodynamically stable adults requiring emergency intubation at Dhaka Medical College Hospital between July 2019 and June 2020. Pre-intubation vital signs were recorded and both indices calculated. Hypotension was defined as systolic blood pressure ≤90 mmHg, mean arterial pressure ≤65 mmHg, or vasopressor initiation within 30 minutes, with a structured assessment at 20 minutes to facilitate early response. Receiver operating characteristic (ROC) analysis and the Youden index assessed predictive performance and optimal cutoffs. Paired DeLong testing compared areas under the curve (AUCs).*

Results: *Hypotension occurred in 42 patients (52.5%). MSI had a numerically higher AUC than SI: 0.830 (95% CI 0.733–0.927) versus 0.751 (95% CI 0.642–0.860), but paired DeLong testing did not establish superiority ($z = 1.53$, $p = 0.127$). MSI remained independently associated with hypotension after adjustment for age, sex, traumatic brain injury, and comorbidity (adjusted odds ratio 193.7; 95% CI 17.3–2172; $p < 0.001$). Organophosphate poisoning was excluded from the model because all 10 affected patients developed hypotension (complete separation; Fisher's exact $p = 0.0012$, assessed separately). Optimal cutoffs were 1.15 for MSI and 0.85 for SI. Sensitivity, specificity, positive predictive value, and negative predictive value were 90.5%, 76.3%, 80.9%, and 87.9% for MSI, compared with 81.0%, 63.2%, 70.8%, and 75.0% for SI.*

Conclusion: *MSI showed numerically higher discrimination than SI and remained independently associated with post-intubation hypotension, although the AUC difference was not statistically significant. An MSI ≥1.15 may help identify patients for haemodynamic optimisation. Larger multicentre studies are needed to confirm its additional predictive value over SI.*

Keywords: *Emergency airway management; Intensive care unit; Haemodynamic instability; Modified Shock Index; post-intubation hypotension; Rapid sequence intubation; Shock Index.*

Introduction

Emergency endotracheal intubation in the ICU differs fundamentally from elective airway management performed under controlled operative conditions. ICU patients who require emergency intubation are often physiologically compromised, with reduced cardiovascular reserve, ongoing organ dysfunction, and preexisting haemodynamic stress. When sedative and neuromuscular blocking agents are administered and positive pressure ventilation is initiated, a complex set of physiological perturbations occurs and venous return falls, cardiac preload is reduced, and both vasodilation and myocardial depression may follow. The cumulative haemodynamic consequence can be abrupt and clinically serious.[1,2]

Post-intubation hypotension has been identified as one of the most frequent and consequential complications of emergency airway management in the ICU. Even brief periods of circulatory compromise following intubation have been associated with impaired tissue oxygenation, increased organ dysfunction, and higher mortality.[3-5] Identifying which patients are at greatest risk before the procedure allows clinicians to prepare targeted preventive measures, including fluid loading, vasopressor initiation, or modification of induction agent choice and dose.[6,7]

Individual vital signs such as heart rate and blood pressure are routinely available at the bedside but may be misleading in critically ill patients, because compensatory sympathetic responses can maintain these parameters within apparently normal ranges even when significant circulatory compromise is present.[8] It is equally important to recognise, however, that heart rate and blood pressure are non-specific signals that can be altered by factors entirely unrelated to circulating volume or cardiac output, an issue that is addressed explicitly later in this manuscript. Composite physiological indices that combine multiple variables into a single score may offer better sensitivity for detecting subclinical haemodynamic instability, provided their inputs are interpreted with these confounders in mind.

Received : February 22, 2026; Accepted :

The Shock Index, originally described by Allgöwer and Burri in 1967, is calculated by dividing heart rate by systolic blood pressure and has been widely used in trauma and emergency settings as a marker of circulatory compromise.[9,10] Elevated SI has been associated with adverse outcomes across a range of critical illness presentations.[10,11] However, SI has a fundamental limitation: it uses systolic blood pressure, which reflects only peak ventricular ejection pressure and does not capture diastolic function or systemic vascular resistance. Mean arterial pressure, by contrast, integrates both systolic and diastolic components and provides a more accurate representation of the average perfusion pressure driving blood flow to vital organs throughout the cardiac cycle.[12]

The Modified Shock Index addresses this limitation by substituting mean arterial pressure for systolic blood pressure in the denominator. Studies in emergency department and critical care populations have evaluated SI and MSI as predictors of post-intubation haemodynamic outcomes.[13,14] However, data specifically from ICU patients undergoing emergency intubation are limited, and critically ill patients represent a physiologically distinct group from those encountered in the emergency department.

The present study was therefore designed to evaluate and compare the predictive accuracy of MSI and SI for post-intubation hypotension in adult ICU patients undergoing emergency endotracheal intubation, and to identify optimal diagnostic thresholds for each index using ROC curve analysis. In light of peer review, this revised manuscript additionally gives explicit consideration to confounding influences on heart rate, to multivariable adjustment, and to diagnosis specific physiology (traumatic brain injury and organophosphate poisoning) that may affect the interpretation of MSI in this cohort.

Methods

Study design and setting: This was a prospective observational study conducted in the Intensive Care Unit, Department of Anaesthesia, Analgesia, Palliative and Intensive Care Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh. Data were collected between July 2019 and June 2020. Ethical approval was obtained from the Institutional Ethical Review Committee of Dhaka Medical College (Ref: ERC DMC/ECC/2020/63(R), dated 02/03/2020). Written informed consent was obtained from the legal guardian of each patient before enrolment.

Study population: Consecutive adult patients aged 18 years or older who required emergency endotracheal intubation in the ICU were considered for enrolment. Only patients who were apparently haemodynamically stable immediately before intubation were included, so that the predictive value of SI and MSI could be assessed under conditions representative of routine clinical practice.

Inclusion criteria: (i) age ≥18 years; (ii) requirement for emergency endotracheal intubation in the ICU; (iii) pre-intubation systolic blood pressure ≥90 mmHg or mean arterial pressure ≥65 mmHg; (iv) no vasopressor administration within 60 minutes before intubation.

Exclusion criteria: Patients were excluded if intubation was performed outside the ICU, or if they had any of the following conditions: acute coronary syndrome, active malignancy, end stage renal disease, uncontrolled hypertension, diabetes mellitus, or congestive heart failure.

Pre-intubation heart rate can be altered independently of circulating volume by rate controlling or rate modifying medication (beta blockers, calcium channel blockers, digoxin), by sympathomimetic or vasodilator drugs, by cardiac arrhythmia (particularly atrial fibrillation and other tachyarrhythmias), and by autonomic dysfunction arising from primary neurological disease. These variables were not systematically recorded as exclusion or stratification criteria in the original protocol; this is acknowledged as a limitation and is discussed in detail below, and their prospective collection is recommended for future validation studies of MSI.

Data collection and study procedure: Baseline heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded from calibrated bedside monitors immediately before intubation. Shock Index was calculated as heart rate divided by systolic blood pressure. Modified Shock Index was calculated as heart rate divided by mean arterial pressure. All intubations were performed by experienced intensivists following a standardised ICU rapid sequence intubation protocol using appropriate sedative and neuromuscular blocking agents selected at the discretion of the treating clinician; the specific agent, dose, number of intubation attempts, pre-intubation fluid bolus volume, and timing of any vasopressor initiation were not systematically captured in a structured case report form, and are acknowledged as a data collection gap (see Strengths and Limitations). Haemodynamic parameters were formally reassessed at 20 minutes after intubation, with continued bedside monitoring to 30 minutes to capture the full outcome window defined below.

Outcome definition: Post-intubation hypotension was defined as the occurrence of any of the following within 30

1. Anaesthesiologist, Department of Anaesthesia, Analgesia, Palliative and Intensive Care Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh
2. Assistant Professor of Critical Care Medicine, Dhaka Medical College, Dhaka, Bangladesh.
3. Anaesthesiologist, Department of Anaesthesia, Analgesia, Palliative and Intensive Care Medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh
4. Assistant Professor of Radiology, NIDCH, Dhaka

Corresponding Author:

Dr Mohammad Abbas Uddin Ahmed
MD (Critical Care Medicine)
Anaesthesiologist
Department of Anaesthesia, Analgesia, Palliative and Intensive Care Medicine,
Dhaka Medical College Hospital, Dhaka, Bangladesh
Email: drabbasccm@gmail.com

minutes of intubation: systolic blood pressure ≤90 mmHg; mean arterial pressure ≤65 mmHg; or initiation of vasopressor therapy. The 20-minute bedside reassessment reported in Table VI and Table VII was the primary structured measurement point within this 30-minute window; no additional hypotensive events were identified between 20 and 30 minutes in this cohort, and the terms are used interchangeably in the Results only for this reason. A convenience sample of 80 patients meeting the inclusion criteria was enrolled over the study period.

Statistical analysis: Data were analysed using IBM SPSS Statistics, version 22 (IBM Corp., Armonk, NY, USA), with supplementary DeLong's test and multivariable logistic regression performed on the same 80 patient dataset. Continuous variables are expressed as mean ± standard deviation. Categorical variables are presented as frequency and percentage. ROC curves were generated for both SI and MSI, and the area under the curve (AUC) was calculated with 95% confidence intervals. The optimal cutoff value for each index was identified using the Youden index. Sensitivity, specificity, PPV, NPV, and a measure of agreement (kappa coefficient) were calculated at the optimal cutoff. DeLong's test was used to formally compare the two correlated ROC curves for SI and MSI. A multivariable logistic regression model was fitted with post-intubation hypotension as the outcome and MSI, age, sex, traumatic brain injury, and comorbidity as covariates; organophosphate poisoning was assessed separately by Fisher's exact test because it produced complete separation when entered into the regression model. The chi-square test was used to assess the unadjusted association between elevated SI and MSI and the occurrence of post-intubation hypotension. A two-tailed p value of <0.05 was considered statistically significant.

Because SI and MSI were derived from the same 80 patients, DeLong's test for two correlated ROC curves was performed on the patient level dataset to test whether the AUC difference between MSI and SI was statistically significant. A multivariable logistic regression model (post-intubation hypotension as the dependent variable; MSI, age, sex, traumatic brain injury, and presence of a comorbidity as covariates) was also fitted to assess whether MSI retained predictive value after adjustment. Organophosphate poisoning could not be entered as a covariate because every affected patient developed the outcome (complete separation), which is reported descriptively instead using Fisher's exact test. Full results of both analyses are given below.

Results

Eighty patients were enrolled. The overall mean age was 35.98 ± 14.95 years (range 18–70). The 21-to-30-year age group was most heavily represented, accounting for 28.8% of the cohort, followed by patients older than 50 years (26.3%). Male patients comprised 56.25% of the study population and female patients 43.75%. The mean age of male patients was 38.42 ± 15.15 years and of female patients 32.85 ± 14.29 years (Table I).

Table I: Age and gender distribution of study subjects (N=80)

Age (years)	Male	Female	Total
≤20	7 (15.6%)	10 (28.6%)	17 (21.3%)
21–30	12 (26.7%)	11 (31.4%)	23 (28.8%)
31–40	8 (17.8%)	4 (11.4%)	12 (15.0%)
41–50	4 (8.9%)	3 (8.6%)	7 (8.8%)
>50	14 (31.1%)	7 (20.0%)	21 (26.3%)
Mean ± SD	38.42 ± 15.15	32.85 ± 14.29	35.98 ± 14.95
Range	18–70	19–64	18–70
Total	45 (56.25%)	35 (43.75%)	80 (100%)

Table II shows baseline haemodynamic parameters. The mean heart rate was 105 ± 15.5 bpm; elevated heart rate was present in 54 patients (67.5%). The mean systolic blood pressure was 121.45 ± 28.59 mmHg and mean arterial pressure was 91.40 ± 20.19 mmHg, indicating that although patients met the haemodynamic stability criteria for enrolment, a substantial proportion had evidence of background physiological stress. Table III shows the calculated SI and MSI values. The mean SI was 0.91 ± 0.21 and the mean MSI was 1.22 ± 0.27, with the higher MSI mean reflecting the proportionally lower denominator when MAP rather than systolic blood pressure is used.

Table II: Pre-intubation haemodynamic parameters (N=80)

Parameter	Mean ± SD	Range	Elevated n (%)
Heart rate (bpm)	105 ± 15.5	85–120	54 (67.5%)
SBP (mmHg)	121.45 ± 28.59	90–150	23 (28.8%)
DBP (mmHg)	76.45 ± 17.66	55–95	17 (21.3%)
MAP (mmHg)	91.40 ± 20.19	70–112	27 (33.8%)

Table III: Pre-intubation Shock Index (SI) and Modified Shock Index (MSI) values (N=80)

Index	Mean ± SD	Range (Min Max)
Shock Index (SI)	0.91 ± 0.21	0.60–1.40
Modified Shock Index (MSI)	1.22 ± 0.27	0.70–1.70

The primary reasons for ICU admission are shown in Table IV. Traumatic brain injury was the most common indication, present in 15 patients (18.8%), followed by eclampsia in 11 (13.8%), and pneumonia and organophosphate poisoning each in 10 (12.5%). Neurological conditions and poisoning together accounted for more than half of admissions. Traumatic brain injury (18.8%) and organophosphate poisoning (12.5%) together account for a substantial minority of the cohort, and both conditions can alter heart rate through mechanisms unrelated to circulating volume, raised intracranial pressure via the Cushing reflex, and cholinergic excess or therapeutic atropinisation, respectively. The three most frequent comorbidities were COPD in 9 patients (11.3%), bronchial asthma in 8 (10.0%), and epilepsy in 8 (10.0%) (Table V).

Table IV: Primary diagnoses of study subjects (N=80)

Primary Diagnosis	Frequency (n)	Percentage (%)
Traumatic brain injury	15	18.8
Eclampsia	11	13.8
Pneumonia	10	12.5
Organophosphate poisoning	10	12.5
Meningoencephalitis	7	8.8
Acute exacerbation of COPD	5	6.3
Near hanging	5	6.3
Acute exacerbation of bronchial asthma	5	6.3
Guillain–Barré syndrome	4	5.0
Traumatic cervical cord compression	3	3.8
Paraquat poisoning	3	3.8
Myasthenic crisis	2	2.5
Total	80	100

Table V: Comorbidities of study subjects (N=80)

Comorbidity	Frequency (n)	Percentage (%)
Chronic obstructive pulmonary disease	9	11.3
Bronchial asthma	8	10.0
Epilepsy	8	10.0

After intubation, the mean systolic blood pressure fell to 95.62 ± 22.71 mmHg, mean diastolic blood pressure to 55.06 ± 15.16 mmHg, and mean MAP to 68.93 ± 16.35 mmHg, reflecting the haemodynamic impact of sedation and positive pressure ventilation (Table VI). Post-intubation hypotension, assessed within the full 30-minute outcome window defined in Methods, occurred in 42 patients (52.5%), while 38 patients (47.5%) maintained adequate blood pressure throughout the observation period (Table VII).

Table VI: Post-intubation blood pressure parameters (N=80)

Parameter	Mean ± SD	Range
Systolic blood pressure (mmHg)	95.62 ± 22.71	70–120
Diastolic blood pressure (mmHg)	55.06 ± 15.16	40–70
Mean arterial pressure	68.93 ± 16.35	53–85

Table VII: Occurrence of post-intubation hypotension within the 30-minute outcome window (N=80)

Outcome	Frequency (n)	Percentage (%)
Hypotension within 30 minutes	42	52.5
No hypotension within 30 minutes	38	47.5
Total	80	100

ROC curve analysis and AUC comparison

The AUC for MSI was 0.830 (95% CI: 0.733–0.927) compared with 0.751 (95% CI: 0.642–0.860) for SI, indicating a numerically higher AUC for MSI in identifying patients who will develop post-intubation hypotension.

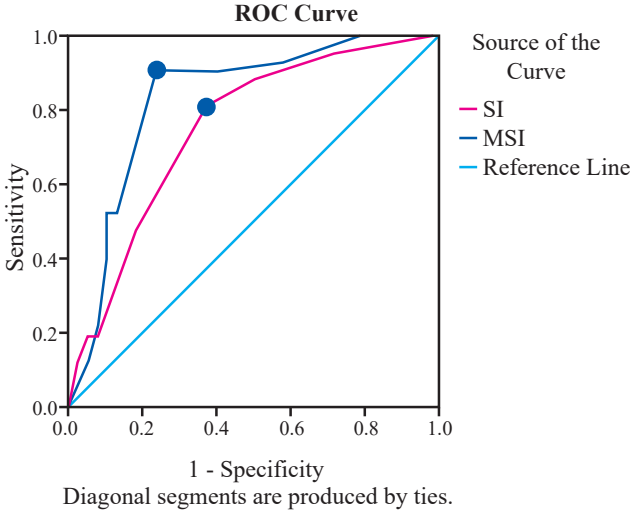


Figure 1: Receiver operating characteristic (ROC) curves comparing the Shock Index (SI) and Modified Shock Index (MSI) for predicting post-intubation hypotension (N = 80).

MSI showed a numerically higher area under the curve (AUC) than SI (0.830, 95% CI 0.733–0.927 vs 0.751, 95% CI 0.642–0.860). The difference between the two ROC curves was not statistically significant on paired DeLong testing (z = 1.53, p = 0.127).

DeLong's test for two correlated ROC curves was performed on the patient level dataset (N=80) to formally compare the AUCs. The between curve comparison did not reach conventional statistical significance (z = 1.53, p = 0.127). Therefore, although MSI had a numerically higher AUC than SI in this cohort, statistical superiority over SI was not confirmed at this sample size; a larger multicentre sample would be needed to test this comparison with adequate power.

Table VIII: Area under the ROC curve for SI and MSI in predicting post-intubation hypotension (N=80). Between curve comparison: paired DeLong test on patient level data, z = 1.53, p = 0.127.

Index	AUC	p value	95% CI Lower	95% CI Upper
Shock Index (SI)	0.751	< 0.001	0.642	0.860
Modified Shock Index (MSI)	0.830	< 0.001	0.733	0.927

Optimal cutoffs by Youden index: Youden index analysis identified an optimal SI cutoff of 0.85, at which sensitivity was 0.810 and specificity 0.632 (Youden index 0.441; Table IX). For MSI, the optimal cutoff was 1.15, at which sensitivity was 0.905 and specificity 0.763, yielding a Youden index of 0.668 (Table X). The higher Youden index for MSI is consistent with a better balance between sensitivity and

specificity across the tested thresholds in this cohort, subject to the caveats on statistical comparison and confounding noted above.

Table IX: Youden index analysis for Shock Index (SI) in predicting post-intubation hypotension

SI Cutoff	Sensitivity	Specificity	Youden Index
0.65	0.952	0.289	0.242
0.75	0.881	0.500	0.381
0.85 ✓	0.810	0.632	0.441 (optimal)
0.95	0.571	0.763	0.335
1.05	0.476	0.816	0.292
1.12	0.190	0.921	0.112

Table X: Youden index analysis for Modified Shock Index (MSI) in predicting post-intubation hypotension

MSI Cutoff	Sensitivity	Specificity	Youden Index
0.75	1.000	0.026	0.026
0.85	1.000	0.211	0.211
0.95	0.929	0.421	0.350
1.05	0.905	0.605	0.510
1.15 ✓	0.905	0.763	0.668 (optimal)
1.25	0.810	0.789	0.599
1.33	0.524	0.868	0.392
1.38	0.524	0.895	0.419

Using the SI cutoff of 0.85, 34 of 48 patients with SI ≥0.85 developed post-intubation hypotension (70.8%), compared with 8 of 32 patients with SI <0.85 (25.0%; $p < 0.001$; Table XI). The unadjusted association was numerically stronger for MSI: 38 of 47 patients with MSI ≥1.15 developed hypotension (80.9%), compared with 4 of 33 patients with MSI <1.15 (12.1%; $p < 0.001$; Table XII). As these are unadjusted chi-square associations, they do not account for the diagnosis mix and medication related confounders discussed below and should not be interpreted as evidence of an independent causal effect of MSI.

Table XI: Association between Shock Index (SI ≥0.85) and post-intubation hypotension (N=80*)*

SI	Total n (%)	Hypotension Yes n (% of row)	Hypotension No n (% of row)	p value
≥ 0.85	48 (60.0)	34 (70.8)	14 (29.2)	
< 0.85	32 (40.0)	8 (25.0)	24 (75.0)	< 0.001
Total	80 (100)	42 (52.5)	38 (47.5)	

Table XII: Association between Modified Shock Index (MSI ≥1.15) and post-intubation hypotension (N=80)

MSI	Total n (%)	Hypotension Yes n (% of row)	Hypotension No n (% of row)	p value
≥ 1.15	47 (58.8)	38 (80.9)	9 (19.1)	
< 1.15	33 (41.3)	4 (12.1)	29 (87.9)	< 0.001
Total	80 (100)	42 (52.5)	38 (47.5)	

Table XIII summarises the full diagnostic performance of both indices at their optimal cutoffs. MSI outperformed SI numerically across every metric in this single cohort: sensitivity 90.5% vs 81.0%, specificity 76.3% vs 63.2%, PPV 80.9% vs 70.8%, NPV 87.9% vs 75.0%, and kappa coefficient 0.619 vs 0.466. As above, these are descriptive, unadjusted comparisons from one dataset rather than formally tested differences between correlated diagnostic tests.

Table XIII: Comparative diagnostic performance of SI and MSI in predicting post-intubation hypotension at optimal cutoff values (N=80)

Index	Cutoff	Sens (%)	Spec (%)	PPV (%)	NPV (%)	Kappa	AUC (95% CI)
SI	0.85	81.0	63.2	70.8	75.0	0.466	0.751 (0.642–0.860)
MSI	1.15	90.5	76.3	80.9	87.9	0.619	0.830 (0.733–0.927)

A multivariable logistic regression model was fitted with post-intubation hypotension as the outcome and MSI, age, sex, traumatic brain injury, and presence of a comorbidity (COPD, asthma, or epilepsy) as covariates (Table XIV). All 10 patients with organophosphate poisoning developed post-intubation hypotension (0 of 10 did not), a pattern of complete separation that prevents a stable coefficient for this variable being estimated by maximum likelihood; this association is instead reported descriptively (10/10 vs 32/70 in non OPC patients; Fisher's exact $p = 0.0012$) rather than included in the regression model. In the adjusted model, MSI remained statistically significantly associated with post-intubation hypotension (adjusted OR 193.7, 95% CI 17.3–2172, $p < 0.001$), while age, sex, TBI, and comorbidity were not independently significant. The very wide confidence interval and large point estimate for MSI reflect its narrow numerical range in this cohort (a one-unit change in MSI represents a very large physiological shift) and the modest sample size. The estimate should therefore be interpreted as supporting the direction and persistence of the association after available adjustment, rather than as a precise or fully generalisable effect size. This finding addresses part of the concern that MSI's apparent predictive value might simply reflect uncontrolled confounding: after adjusting for age, sex, TBI, and comorbidity, the association between MSI and post-intubation hypotension persisted, but important unmeasured confounders remained unavailable for adjustment.

Table XIV: Multivariable logistic regression for post-intubation hypotension (N=80). Organophosphate poisoning excluded due to complete separation (10/10 patients developed the outcome; Fisher's exact p = 0.0012 for this association considered separately)

Covariate	Adjusted OR	95% CI	p value
MSI (per unit)	193.7	17.3, 2172	< 0.001
Age (per year)	0.96	0.92, 1.01	0.13
Male sex	1.33	0.41, 4.29	0.64
Traumatic brain injury	2.32	0.52, 10.30	0.27
Any comorbidity (COPD/asthma/epilepsy)	1.07	0.23, 4.98	0.93

Discussion

This prospective observational study was designed to determine whether the Modified Shock Index offers better predictive accuracy than the conventional Shock Index for post-intubation hypotension in adult ICU patients undergoing emergency endotracheal intubation. The findings show a numerically favourable picture for MSI, and its association with the outcome persisted after multivariable adjustment. The AUC of 0.830 for MSI versus 0.751 for SI is directionally consistent with a possible advantage for MSI, but a formal paired DeLong test on the patient level data did not confirm this difference as statistically significant (p = 0.127), and this should temper how strongly the AUC comparison itself is interpreted. At their respective optimal cutoffs, MSI achieved a sensitivity of 90.5% and specificity of 76.3% compared with 81.0% and 63.2% for SI. The NPV of 87.9% for MSI versus 75.0% for SI, and the kappa coefficient of 0.619 versus 0.466, are consistent with better agreement for MSI in this cohort, but, like the AUC comparison, represent single cohort point estimates rather than formally tested differences, and should be read alongside the limitations discussed below before being generalised. The adjusted analysis suggests that MSI remained independently associated with post-intubation hypotension after adjustment for age, sex, TBI, and comorbidity (Table XIV). This supports a plausible clinical implication that a pre-intubation MSI of ≥1.15 may be a useful prompt for active haemodynamic preparation before airway intervention, used as one input among several rather than in isolation, even though its formal superiority over SI remains unconfirmed.

Post-intubation hypotension occurred in 52.5% of patients in this cohort, confirming that it is not an infrequent complication but rather a common and expected challenge in the critically ill ICU population. This incidence aligns with rates reported across the published literature. Jaber et al. conducted a landmark prospective study of ICU intubation complications and reported that severe cardiovascular collapse occurred in a substantial proportion of patients, with hypotension representing one of the most frequent adverse events.[15] Heffner et al. specifically examined post-intubation hypotension in emergency airway management and documented its association with increased morbidity, including a threefold higher risk of adverse outcomes in affected patients.[16,17] Smischney et al. reported that post-intubation hypotension was associated with increased in hospital mortality and longer hospital length of stay on multivariable analysis.[5] Ko et al. and Wardi et al. further reported that circulatory deterioration following emergency intubation was linked to post-intubation cardiac arrest, a catastrophic complication that is substantially more common than widely appreciated.[18,19]

The high prevalence of hypotension in the present cohort, despite patients meeting haemodynamic stability criteria at enrolment, demonstrates that a normal pre-intubation blood pressure does not reliably predict haemodynamic tolerance of the intubation procedure. This observation reinforces the well-established principle that individual vital signs are insufficient for risk stratification in critically ill patients.[8]

Confounders affecting heart rate and the validity of MSI: A central limitation of any heart rate-based index, including both SI and MSI, is that heart rate is a nonspecific physiological variable that can be altered by numerous factors independent of circulating volume or cardiac output. Rate controlling medication such as beta blockers, calcium channel blockers, and digoxin can blunt or abolish the tachycardic response to hypovolaemia or shock, artificially lowering MSI in a patient who is nonetheless haemodynamically fragile. Conversely, sympathomimetic agents, vasodilators, fever, pain, and anxiety can elevate heart rate independent of true circulatory compromise, artificially raising MSI in an otherwise stable patient. Cardiac arrhythmias, particularly atrial fibrillation and other tachyarrhythmias, produce heart rates that do not reflect stroke volume or perfusion in the way that a regular sinus tachycardia would, and may therefore distort both the numerator of MSI and its clinical meaning. Autonomic dysfunction secondary to underlying neurological disease can similarly produce heart rates that are dissociated from circulating volume status. None of these variables were systematically recorded or adjusted for in the present analysis, and this represents an important limitation. It cannot be excluded that some of the observed predictive performance of MSI in this cohort is attributable, in part, to these unmeasured factors rather than to circulatory compromise alone. Future prospective validation of MSI should record concurrent medication use and cardiac rhythm at the time of index calculation and should ideally exclude or stratify by arrhythmia and rate altering drug exposure.

Multivariable adjustment: Post-intubation hypotension is multifactorial, and the univariate ROC and chi-square analyses alone cannot establish that MSI is an independent predictor once other relevant variables are accounted for. In response to this concern, a multivariable logistic regression model was fitted (Table XIV) with MSI, age, sex, traumatic brain injury, and comorbidity as covariates. MSI remained independently associated with post-intubation hypotension after this adjustment (adjusted OR 193.7, 95% CI 17.3–2172, p < 0.001), which is reassuring evidence that its unadjusted association with the outcome is not simply a proxy for age, sex, TBI status, or comorbidity burden in this cohort. This

does not, however, fully resolve this concern: several other plausible confounders discussed elsewhere in this manuscript, concurrent rate altering medication, cardiac arrhythmia, induction agent choice and dose, and fluid or vasopressor administration, were not recorded and could not be entered into this model, and organophosphate poisoning could not be included because of complete separation. The regression should therefore be read as adjusting for the confounders that were measurable in this dataset, not as a complete multivariable resolution of this concern.

Traumatic brain injury and the Cushing response: Traumatic brain injury constituted the largest single diagnostic category in this cohort (18.8%), and raised intracranial pressure can produce the Cushing response, a combination of systemic hypertension, reflex bradycardia, and irregular respiration mediated by brainstem autonomic centres. In patients with significant intracranial hypertension, heart rate may therefore be inappropriately low relative to true circulatory status, which would tend to lower both SI and MSI and could mask haemodynamic vulnerability, or could produce paradoxical MSI behaviour around the time of induction if the Cushing response is abruptly lost following sedation and loss of the intracranial pressure stimulus. Because heart rate is central to both indices, the inclusion of a substantial proportion of neurological patients introduces a specific and plausible source of bias. A sensitivity analysis restricted to non TBI patients, or a formal interaction test between diagnosis category and MSI performance, would help determine whether the predictive value of MSI observed in the full cohort is preserved, attenuated, or reversed in patients with raised intracranial pressure. This subgroup analysis was not possible with the present sample size but is recommended as a priority for future, larger studies.

Organophosphate poisoning and atropinisation: A comparable concern applies to the 10 patients (12.5%) admitted with organophosphate poisoning. Cholinergic toxicity itself typically produces bradycardia, while the mainstay of treatment, atropine, is deliberately titrated to reverse muscarinic bradycardia and can produce iatrogenic tachycardia independent of circulatory status. Heart rate in these patients at the time of pre-intubation assessment may therefore reflect the degree and timing of atropinisation as much as, or more than, true haemodynamic reserve. As with TBI, this represents a plausible and diagnosis specific distortion of the MSI signal. Notably, all 10 organophosphate poisoning patients in this cohort developed post-intubation hypotension (Fisher's exact $p = 0.0012$ versus the remaining patients), a rate so uniform that it could not be entered into the multivariable model above without producing an unstable, non-identifiable coefficient (complete separation). This near uniform outcome may reflect genuine severe haemodynamic vulnerability in poisoning patients, confounding from atropinisation and cholinergic toxicity as described above, or both; the present data cannot distinguish between these explanations. Reporting the atropine dose and timing relative to intubation and considering a dedicated analysis of poisoning patients in a larger sample, would strengthen future work in this area.

Physiological rationale for MSI over SI: The limitations of isolated vital signs for detecting haemodynamic vulnerability are well recognised. Heart rate and blood pressure may remain within acceptable ranges in critically ill patients due to compensatory sympathetic activation, even when intravascular volume, cardiac reserve, and organ perfusion are substantially compromised.[8] In this state of compensated shock, the removal of sympathetic stimulation by induction agents or the abrupt reduction in venous return caused by positive pressure ventilation may unmask haemodynamic fragility that was not apparent from the resting vital signs alone. Composite physiological indices that combine multiple variables have therefore been developed to improve early detection of occult circulatory compromise.[9,10] Smischney et al. reported that both higher pre-intubation SI and MSI values were significantly associated with post-intubation haemodynamic derangement in ICU patients, supporting the concept that pre procedure index values carry predictive information beyond individual vital signs.[20]

The Shock Index, first described by Allgöwer and Burri in 1967, was originally developed as a simple marker of blood loss and circulatory compromise in haemorrhagic shock.[9] Its appeal lies in its simplicity: it requires only two measurements that are universally available without any additional equipment. Elevated SI values have since been associated with adverse outcomes across a wide range of clinical contexts including trauma, sepsis, gastrointestinal haemorrhage, pulmonary embolism, and obstetric emergencies.[10,11,21] However, SI has fundamental physiological limitations. Systolic blood pressure, which forms its denominator, reflects the peak pressure generated during ventricular ejection and is strongly influenced by arterial wall stiffness, peripheral vascular resistance, and the velocity of ventricular contraction. It does not represent the sustained driving pressure that determines organ perfusion throughout the cardiac cycle. As a result, SI may fail to detect haemodynamic insufficiency in patients with elevated vascular tone who maintain a normal or elevated systolic pressure despite reduced cardiac output and end organ perfusion.[8,12]

The Modified Shock Index addresses this limitation directly by substituting mean arterial pressure for systolic blood pressure. MAP is calculated as diastolic blood pressure plus one third of pulse pressure and integrates both the systolic ejection component and the diastolic filling component of the blood pressure waveform. It therefore represents the time averaged pressure driving blood flow to vital organs and is physiologically more meaningful as an indicator of global tissue perfusion than systolic blood pressure alone.[12] When a patient has tachycardia with only modest systolic hypertension but a relatively low diastolic blood pressure, the resulting MAP may be considerably lower than the systolic value would suggest. In such patients, SI may appear falsely reassuring while MSI more closely captures the compromised perfusion pressure. This scenario is common in critically ill patients who are peripherally vasoconstricted with a narrow pulse pressure, a haemodynamic profile that is frequently seen

in early septic shock, hypovolaemia, and cardiogenic compromise. Liu et al. demonstrated that MSI identified critical illness severity in emergency department patients more accurately than SI and was associated with superior prediction of in hospital mortality.[12] The physiological rationale described here provides a plausible mechanistic explanation for the improved unadjusted diagnostic performance of MSI observed in the present study and in prior investigations, though, as discussed above, it does not substitute for confirmatory adjusted and paired comparison analyses.

The findings of this study can be considered alongside a growing body of literature evaluating shock indices as bedside haemodynamic tools. Lee et al. found that pre-intubation age shock index, MSI, and SI were independent predictors of post-intubation hypotension in emergency department patients, with age shock index showing the best discrimination.[13] Trivedi et al. evaluated SI and MSI in haemodynamically stable ICU patients undergoing emergency intubation and found that SI ≥0.90 predicted post-intubation hypotension, whereas MSI was not associated with the measured outcomes.[14] Smischney et al. later reported that higher pre-intubation SI and MSI values were significantly associated with post-intubation haemodynamic derangement in critically ill patients.[20] Koby et al. reported that a pre-intubation SI of ≥0.8 was associated with increased risk of post-intubation hypotension, although it was not associated with longer intubation duration or higher in hospital mortality.[22] Koch et al. reviewed SI across multiple emergency conditions and highlighted that its predictive performance and useful thresholds vary according to the clinical context and patient population.[21] Taken together, these studies show that shock indices may assist risk stratification, but the literature is mixed regarding whether MSI offers an incremental advantage over SI, and the confounding and adjustment issues discussed here remain important.

The diagnostic performance metrics reported in this study deserve individual consideration. The sensitivity of 90.5% for MSI at the 1.15 cutoff means that 38 of 42 patients who subsequently developed post-intubation hypotension were correctly identified as high risk before the procedure in this unadjusted analysis. For a bedside screening tool applied in a time pressured clinical environment, this level of sensitivity is encouraging and compares favourably with other risk stratification tools used in emergency airway management.[17,15] The specificity of 76.3% indicates that among patients who did not develop hypotension, three quarters were correctly identified as low risk. The PPV of 80.9% and NPV of 87.9% are both potentially useful operationally, allowing the clinician to weigh pre-emptive intervention against the risk of unnecessary prophylactic treatment, provided the confounders described above are considered when interpreting an individual patient's MSI value. The kappa coefficient of 0.619 reflects moderate to substantial agreement in this cohort, though, as elsewhere in this Discussion, this should be read as descriptive rather than as proof that the association is independent of the unmeasured variables identified above.[5]

Understanding why post-intubation hypotension is so common in critically ill patients requires consideration of the multiple simultaneous haemodynamic insults that occur at the time of intubation. Induction agents exert direct cardiovascular effects that vary by drug class. Propofol and thiopentone reduce sympathetic tone, cause peripheral vasodilation, and may directly depress myocardial contractility.[23] Benzodiazepines similarly blunt sympathetic drive, and their combination with opioid analgesics further impairs the adrenergic response to hypotension. Ketamine, which maintains or increases sympathetic tone in patients with intact catecholamine stores, is often preferred in haemodynamically compromised patients, but its sympathomimetic effect depends on intact endogenous catecholamine reserves; in patients with catecholamine depletion from prolonged critical illness, ketamine may precipitate hypotension despite its reputation for haemodynamic stability.[24,25] The administration of neuromuscular blocking agents removes the patient's ability to generate respiratory muscle activity, and the subsequent initiation of positive pressure ventilation increases mean intrathoracic pressure. This reduces the pressure gradient driving venous return to the right heart, diminishes right ventricular preload, and consequently reduces left ventricular filling and cardiac output.[1,2] In patients who are volume depleted, vasodilated from sepsis, or have impaired ventricular function, the combination of these drug effects and the mechanical consequences of positive pressure ventilation may precipitate severe and sudden haemodynamic collapse. As noted above, the specific induction agents, doses, and fluid strategies used in this cohort were not systematically captured, which limits the ability to attribute outcomes to MSI rather than to these co-occurring management factors. Pre-emptive strategies including fluid administration, vasopressor initiation, and careful agent selection have been shown to reduce the incidence of this complication.[6,7]

The clinical implications of these findings should be framed cautiously in light of the limitations described above. When MSI is calculated to be ≥1.15 before intubation, clinicians may reasonably consider undertaking haemodynamic optimisation before proceeding with airway intervention, as one input alongside clinical judgement regarding medication history, cardiac rhythm, and underlying diagnosis. This may include administration of a fluid bolus to augment preload, initiation or up titration of a vasopressor infusion to maintain vascular tone during induction, or selection of an induction agent with a more favourable haemodynamic profile such as ketamine in a non-catecholamine depleted patient.[6,7,23] Panchal et al. demonstrated that prophylactic administration of phenylephrine during rapid sequence intubation significantly reduced the incidence of post induction hypotension compared with no pretreatment.[6] Two randomised trials found that routine administration of a 500 mL crystalloid bolus before intubation did not reduce cardiovascular collapse compared with no routine bolus, indicating that fluid administration should be individualised rather than applied automatically.[26,27]

Conversely, when MSI is <1.15, the clinician may proceed with somewhat greater reassurance, while still applying clinical judgement, particularly in patients on rate controlling medication or with arrhythmia, where a falsely low MSI is plausible. This bidirectional utility, if confirmed by adjusted and externally validated analyses, would be a useful property of a bedside index, but should not yet be regarded as established practice on the basis of this single centre dataset.

The patient population in this study reflects the typical case mix of a tertiary ICU in South Asia. Traumatic brain injury was the most common admission diagnosis, contributing to the relatively young age distribution observed compared with Western critical care populations, where cardiovascular and respiratory diseases predominate in older patients. Eclampsia accounted for 13.8% of admissions, reflecting the higher prevalence of hypertensive disorders of pregnancy in this geographic and demographic context. Organophosphate and paraquat poisoning together accounted for a further 16.3% of cases, a pattern that is distinctive to South Asian and Southeast Asian ICU populations. As discussed above, both the TBI and poisoning subgroups carry heart rate altering physiology that is specific to their aetiology, meaning the diverse case mix that gives this study external relevance in South Asian settings is the same feature that most limits the internal validity of MSI as a unified, diagnosis agnostic index in this cohort.

A practical advantage of MSI as a pre-intubation risk stratification tool, independent of the statistical caveats above, is its complete lack of procedural or resource requirements. Heart rate and mean arterial pressure are measured non-invasively and displayed continuously on bedside monitors in every ICU without exception. The MSI is calculated by a single division, taking less than five seconds. Unlike severity scores such as APACHE or SOFA, which require laboratory results, or haemodynamic indices derived from echocardiography or invasive monitoring, MSI imposes no additional cost, no additional time, and no additional technical skill.[8,12] This makes it potentially suitable for implementation in resource limited settings across South Asia, sub Saharan Africa, and other regions where advanced haemodynamic monitoring may not be available around the clock, provided clinicians are trained to interpret MSI in the context of concurrent medication, rhythm, and diagnosis rather than as an isolated number.

Strengths and Limitations

The prospective observational design of this study allowed systematic and standardised collection of haemodynamic data at defined time points before and after intubation. All airways were managed according to a uniform ICU rapid sequence intubation protocol, which reduces variability attributable to technique. The use of ROC curve analysis with Youden index derived cutoffs provides an objective and reproducible basis for the diagnostic thresholds reported.

Several limitations, some identified during peer review, should be considered when interpreting these findings:

- Confounders affecting heart rate, rate controlling and rate altering medication, cardiac arrhythmia, and autonomic dysfunction from neurological disease were not systematically recorded and could not be excluded or adjusted for, even in the multivariable model, and may have influenced MSI values independently of true circulatory status.
- Multivariable regression was performed (Table XIV) and MSI remained independently associated with the outcome after adjustment for age, sex, TBI, and comorbidity (adjusted OR 193.7, 95% CI 17.3–2172, $p < 0.001$). This strengthens, but does not fully resolve, the original concern, because induction agent, fluid/vasopressor strategy, and organophosphate poisoning (excluded due to complete separation) could not be included in the same model.
- Traumatic brain injury subgroup, the Cushing response can alter heart rate independently of circulating volume in the 15 TBI patients in this N=80 cohort, and a dedicated interaction analysis (rather than inclusion as a simple covariate) was not possible with the available sample size.
- Organophosphate poisoning subgroup, all 10 OPC poisoning patients in this cohort developed post-intubation hypotension (Fisher's exact $p = 0.0012$), a rate so uniform that the variable produced complete separation and could not be entered into the multivariable model; whether this reflects genuine severity, atropinisation related heart rate confounding, or both cannot be determined from these data.
- Intubation related variables not reported, the specific induction agent, dose, number of intubation attempts, pre-intubation fluid bolus volume, and timing of vasopressor initiation were not systematically captured, despite being established determinants of post-intubation hypotension, and could therefore not be adjusted for in the regression model.
- A formal DeLong test comparing the correlated MSI and SI ROC curves was performed on the patient level data and did not reach statistical significance ($z = 1.53$, $p = 0.127$), despite MSI showing a numerically higher AUC. The originally stated superiority of MSI over SI should therefore be regarded as suggestive rather than statistically confirmed at this sample size.
- Single centre setting and modest sample size ($n = 80$), which limited the power of the DeLong comparison and precluded inclusion of organophosphate poisoning and several other candidate confounders in the regression model.
- Outcome ascertainment was anchored to a fixed 30-minute window with a structured check at 20 minutes; hypotension occurring after this window would not have been captured.

Larger, multicentre, prospective studies that record concurrent medication, cardiac rhythm, induction agent and dose, fluid and vasopressor administration, and that apply multivariable adjustment and a formal paired comparison of ROC curves, are needed to confirm the MSI cutoff of 1.15 and to establish whether MSI provides independent predictive value over SI in a broader and more heterogeneous ICU population.[5,28,20]

Conclusion

In this single centre cohort of 80 ICU patients, MSI ≥1.15 predicted post-intubation hypotension with 90.5% sensitivity, 76.3% specificity, and an AUC of 0.830. Discrimination was numerically higher than SI, but the paired DeLong comparison was not statistically significant (p = 0.127). MSI remained associated with hypotension after adjustment for age, sex, traumatic brain injury, and comorbidity. All 10 patients with organophosphate poisoning developed hypotension, preventing its inclusion in the model; heart rate altering medication, arrhythmia, and induction, fluid, and vasopressor strategies were not systematically recorded. MSI may support bedside risk assessment and haemodynamic optimisation alongside clinical judgement, but its additional value over SI requires confirmation in larger, adequately powered studies.

Conflict of Interest and Funding

The authors declare no conflicts of interest. No external funding was received for this study.

References:

1. Franklin C, Samuel J, Hu TC. Life-threatening hypotension associated with emergency intubation and the initiation of mechanical ventilation. Am J Emerg Med. 1994;12(4):425–8.
2. Manthous CA. Avoiding circulatory complications during endotracheal intubation and initiation of positive pressure ventilation. J Emerg Med. 2010;38(5):622–31.
3. Green RS, Turgeon AF, McIntyre LA, Fox-Robichaud AE, Fergusson DA, Doucette S, et al. Post-intubation hypotension in intensive care unit patients: a multicenter cohort study. J Crit Care. 2015;30(5):1055–60.
4. Holler JG, Bech CN, Henriksen DP, Mikkelsen S, Pedersen C, Lassen AT. Nontraumatic hypotension and shock in the emergency department and the prehospital setting: prevalence, etiology, and mortality: a systematic review. PLoS One. 2015;10(3):e0119331.
5. Smischney NJ, Demirci O, Diedrich DA, Barbara DW, Sandefur BJ, Trivedi S, et al. Incidence of and risk factors for post-intubation hypotension in the critically ill. Med Sci Monit. 2016;22:346–55.
6. Panchal AR, Satyanarayan A, Bahadir JD, Hays D, Mosier J. Efficacy of bolus-dose phenylephrine for peri-intubation hypotension. J Emerg Med. 2015;49(4):488–94.
7. Jaber S, Jung B, Corne P, Sebbane M, Muller L, Chanques G, et al. An intervention to decrease complications related to endotracheal intubation in the intensive care unit: a prospective, multiple-center study. Intensive Care Med. 2010;36(2):248–55.
8. 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.
9. Allgöwer M, Burri C. Schockindex. Dtsch Med Wochenschr. 1967 Oct 27;92(43):1947–50. doi: 10.1055/s-0028-1106070.
10. 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. J Trauma. 2009;67(6):1426–30.
11. Berger T, Green J, Horeczko T, Hagar Y, Garg N, Suarez A, et al. Shock index and early recognition of sepsis in the emergency department: pilot study. West J Emerg Med. 2013;14(2):168–74.
12. Liu YC, Liu JH, Fang ZA, Shan GL, Xu J, Qi ZW, et al. Modified shock index and mortality rate of emergency patients. World J Emerg Med. 2012;3(2):114–7.
13. Lee K, Jang JS, Kim J, Suh YJ. Age shock index, shock index, and modified shock index for predicting postintubation hypotension in the emergency department. Am J Emerg Med. 2020;38(5):911–15.
14. Trivedi S, Demirci O, Arteaga G, Kashyap R, Smischney NJ. Evaluation of preintubation shock index and modified shock index as predictors of postintubation hypotension and other short-term outcomes. J Crit Care. 2015;30(4):861.e1–7.
15. Jaber S, Amraoui J, Lefrant JY, Arich C, Cohendy R, Landreau L, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study. Crit Care Med. 2006;34(9):2355–61.
16. Heffner AC, Swords DS, Neale MN, Jones AE. Incidence and factors associated with cardiac arrest complicating emergency airway management. Resuscitation. 2013;84(11):1500–4.
17. Heffner AC, Swords D, Kline JA, Jones AE. The frequency and significance of post-intubation hypotension during emergency airway management. J Crit Care. 2012;27(4):417.e9–13.
18. Ko BS, Ahn R, Ryoo SM, Ahn S, Sohn CH, Seo DW, et al. Prevalence and outcomes of endotracheal intubation-related cardiac arrest in the emergency department. Am J Emerg Med. 2015;33(11):1642–5.
19. Wardi G, Villar J, Nguyen T, Vyas A, Pokrajac N, Minokadeh A, et al. Factors and outcomes associated with inpatient cardiac arrest following emergent endotracheal intubation. Resuscitation. 2017;121:76–80.
20. Smischney NJ, Seisa MO, Heise KJ, Wiegand RA, Busack KD, Deangelis JL, et al. Predictors of hemodynamic derangement during intubation in the critically ill: a nested case-control study of hemodynamic management—Part II. J Crit Care. 2018;44:179–84.
21. Koch E, Lovett S, Nghiem T, Riggs RA, Rech MA. Shock index in the emergency department: utility and limitations. Open Access Emerg Med. 2019;11:179–99.
22. Koby A, Nwokolo J, Horn A, Banapoor B, Marchick M, Beattie LK. Using pre-Rapid Sequence Intubation Shock Index as a predictor of post-intubation hypotension, mortality, and duration of intubation. Presented at: Florida College of Emergency Physicians Symposium by the Sea; August 2015; Amelia Island, FL.
23. El-Beheiry H, Kim J, Milne B, Seegobin R. Prophylaxis against the systemic hypotension induced by propofol during rapid-sequence intubation. Can J Anaesth. 1995;42(10):875–8.
24. Morris C, Perris A, Klein J, Mahoney P. Anaesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent? Anaesthesia. 2009;64(5):532–9.
25. Miller M, Kruit N, Heldreich C, Ware S, Habig K, Reid C, et al. Hemodynamic response after rapid sequence induction with ketamine in out-of-hospital patients at risk of shock as defined by the shock index. Ann Emerg Med. 2016;68(2):181–188.e2.
26. Janz DR, Casey JD, Semler MW, et al. Effect of a fluid bolus on cardiovascular collapse among critically ill adults undergoing tracheal intubation (PrePARE): a randomised controlled trial. Lancet Respir Med. 2019;7(12):1039–47.
27. Russell DW, Casey JD, Gibbs KW, et al. Effect of fluid bolus administration on cardiovascular collapse among critically ill patients undergoing tracheal intubation: a randomized clinical trial. JAMA. 2022;328(3):270–79.
28. Smischney NJ, Seisa MO, Cambest J, Wiegand RA, Busack KD, Loftsgard TO, et al. The incidence of and risk factors for postintubation hypotension in the immunocompromised critically ill adult. J Intensive Care Med. 2019;34(7):578–86.