e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(2); 2263-2271

Original Research Article

Shock Index and Modified Shock Index as Predictors of Post-Induction Hypotension during General Anaesthesia

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Received: 01-11-2025 / Revised: 15-12-2025 / Accepted: 21-01-2026
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Conflict of interest: Nil

Abstract

Background: Post-induction hypotension (PIH) is a common haemodynamic complication of general anaesthesia and may compromise vital organ perfusion. Early identification of susceptible patients may facilitate preventive haemodynamic management. Shock Index (SI) and Modified Shock Index (MSI) are simple bedside indices that may help predict PIH.

Objective: To evaluate and compare pre-induction SI and MSI as predictors of post-induction hypotension in patients undergoing general anaesthesia.

Materials and Methods: This prospective observational study included 90 adult patients undergoing elective surgery under general anaesthesia. Pre-induction heart rate, systolic blood pressure (SBP), diastolic blood pressure, and mean arterial pressure (MAP) were recorded. SI was calculated as heart rate/SBP and MSI as heart rate/MAP. Haemodynamic parameters were monitored following induction, and patients were categorized according to the occurrence of PIH. Receiver operating characteristic (ROC) analysis was performed to assess the predictive performance of SI and MSI.

Results: PIH occurred in 29 (32.2%) patients. Pre-induction SI (0.71 ± 0.11 vs. 0.59 ± 0.09; $p<0.001$) and MSI (0.91 ± 0.14 vs. 0.76 ± 0.11; $p<0.001$) were significantly higher in patients who developed PIH. SI ≥0.65 predicted PIH with 79.3% sensitivity, 72.1% specificity, and an area under the curve (AUC) of 0.804. MSI ≥0.85 showed 82.8% sensitivity, 77.0% specificity, and an AUC of 0.846.

Conclusion: Both SI and MSI were useful predictors of PIH, with MSI demonstrating slightly better predictive performance. These simple, non-invasive indices may facilitate early identification of patients at increased haemodynamic risk during induction of general anaesthesia.

Keywords: Shock Index; Modified Shock Index; post-induction hypotension; general anaesthesia; haemodynamic instability; mean arterial pressure.

DOI: 10.25258/ijcpr.18.2.371

Introduction

General anaesthesia is an integral component of modern surgical practice; however, induction is frequently accompanied by haemodynamic alterations. Among these, post-induction hypotension (PIH) is an important perioperative complication. Anaesthetic induction may cause vasodilatation, attenuation of sympathetic tone, myocardial depression, and reduction in systemic vascular resistance.[1] These effects may be further aggravated by decreased venous return following positive-pressure ventilation, particularly in elderly, hypovolaemic patients and those with cardiovascular disease or limited physiological reserve.[2] Post-induction hypotension is clinically important because adequate arterial pressure is required to maintain perfusion of vital organs. Both the severity and duration of perioperative hypotension have been associated with adverse outcomes, including myocardial injury, acute kidney injury, cerebral hypoperfusion, prolonged hospital stay, and mortality.[3] Therefore, identifying patients who are susceptible to hypotension before induction may permit appropriate preventive measures and improve perioperative haemodynamic stability.[4] Routine pre-induction assessment primarily relies on individual variables such as heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure, and mean arterial pressure (MAP).[5] However, apparently normal blood pressure may be maintained during early circulatory compromise through compensatory tachycardia and vasoconstriction. Consequently, individual vital signs may not adequately identify patients with

reduced cardiovascular reserve. The Shock Index (SI) is a simple haemodynamic parameter calculated as the ratio of HR to SBP. It was first described by Allgöwer and Burri in 1967.[6] In healthy adults, SI is generally approximately 0.5–0.7, whereas increasing values may indicate haemodynamic compromise. Because SI incorporates changes in both HR and SBP, it may detect early circulatory deterioration before overt hypotension develops. It has been investigated as a prognostic marker in trauma, haemorrhage, sepsis, critical illness, myocardial infarction, pulmonary embolism, and emergency airway management.[7] Shock Index = Heart Rate / Systolic Blood Pressure Evidence from emergency airway studies also supports its ability to predict subsequent haemodynamic deterioration.[8] Reported that post-intubation hypotension occurred in nearly one-quarter of initially normotensive emergency patients and was associated with increased mortality and longer ICU and hospital stay. Further demonstrated that a pre-intubation SI ≥0.90 predicted post-intubation hypotension and ICU mortality. [9,10]

The Modified Shock Index (MSI) substitutes MAP for SBP and is calculated as HR divided by MAP. Since MAP incorporates both systolic and diastolic arterial pressures and more closely reflects systemic perfusion pressure, MSI may provide a more comprehensive assessment of haemodynamic status.[11] Modified Shock Index = Heart Rate / Mean Arterial Pressure demonstrated that MSI provided useful prognostic information regarding mortality in emergency patients and may perform better than conventional SI or isolated vital signs.[12] Both SI and MSI are particularly attractive for perioperative use because they are simple, non-invasive, inexpensive, rapidly calculated, and require no additional investigations or monitoring equipment. Despite their established utility in trauma, emergency medicine, and critically ill populations, evidence regarding the comparative ability of pre-induction SI and MSI to predict hypotension following general anaesthesia remains limited. Identification of reliable cut-off values could allow early recognition of high-risk patients and facilitate fluid optimization, modification of induction-agent dosing, prophylactic vasopressor administration, and closer haemodynamic monitoring. [13,14] Therefore, the present study is undertaken to evaluate Shock Index and Modified Shock Index as predictors of post-induction hypotension during general anaesthesia and to compare their predictive performance for identifying patients at increased risk of haemodynamic instability.

Materials and Methods

This prospective observational study was conducted in the Department of Anaesthesiology .The study included adult patients undergoing elective surgical procedures under general anaesthesia with endotracheal intubation.

Study Population: A total of 90 patients who fulfilled the eligibility criteria and were scheduled to undergo surgery under general anaesthesia were enrolled in the study. Written informed consent was obtained from all participants before inclusion.

Inclusion Criteria: Patients aged 18–65 years, belonging to American Society of Anesthesiologists (ASA) physical status I–III, and scheduled for elective surgery under general anaesthesia with endotracheal intubation were included.

Exclusion Criteria: Patients with preoperative haemodynamic instability, cardiac arrhythmias, severe valvular or ischemic heart disease, uncontrolled hypertension, severe anaemia, sepsis, significant autonomic dysfunction, pregnancy, or those receiving vasopressor or inotropic support before induction were excluded. Patients requiring emergency surgery or in whom the planned anaesthetic technique was changed were also excluded.

Pre-Anaesthetic Assessment: All patients underwent routine pre-anaesthetic evaluation on the day before surgery. Demographic characteristics including age, sex, body weight, height, body mass index, ASA physical status, associated comorbidities, and current medications were recorded. Routine preoperative investigations were reviewed according to institutional protocol. Patients were advised standard fasting according to institutional guidelines. Antihypertensive and other medications were continued or withheld as per routine perioperative practice.

Monitoring and Baseline Haemodynamic Measurements: On arrival in the operating room, standard ASA monitors were applied, including electrocardiography, non-invasive blood pressure monitoring, and pulse oximetry. An intravenous line was secured and appropriate intravenous fluid therapy was initiated.After a resting period of approximately 5 minutes, baseline heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and peripheral oxygen saturation (SpO_2) were recorded. Blood pressure was measured using an appropriately sized cuff with the patient in the supine position. The baseline values used for calculation of haemodynamic indices were recorded immediately before induction of anaesthesia.

Calculation of Shock Index and Modified Shock Index

The pre-induction Shock Index (SI) was calculated using the following formula:

Shock Index = Heart Rate / Systolic Blood Pressure

The Modified Shock Index (MSI) was calculated as:

Modified Shock Index = Heart Rate / Mean Arterial Pressure

Both SI and MSI were calculated from the haemodynamic parameters recorded immediately before induction of general anaesthesia.

Anaesthetic Technique: All patients received a standardized general anaesthetic technique. After preoxygenation with 100% oxygen for 3 minutes, anaesthesia was induced with intravenous propofol at a dose of approximately 1.5–2.5 mg/kg, titrated according to clinical response.

An opioid such as fentanyl 1–2 µg/kg was administered for analgesia. Neuromuscular blockade was achieved using an appropriate non-depolarizing neuromuscular blocking agent.

Following adequate muscle relaxation, direct or video laryngoscopy was performed and the trachea was intubated with an appropriately sized cuffed endotracheal tube.

After confirmation of correct tube placement, controlled mechanical ventilation was initiated. Anaesthesia was maintained with an inhalational anaesthetic agent in an oxygen-air mixture, and ventilation was adjusted to maintain normocapnia.

Haemodynamic Monitoring After Induction: Heart rate, SBP, DBP, and MAP were recorded at baseline and at predefined intervals following induction of anaesthesia.

Haemodynamic measurements were documented immediately after induction and at 1, 3, 5, and 10 minutes after tracheal intubation or until the start of surgical stimulation, whichever occurred earlier.

Any hypotensive episode occurring during this period was recorded along with the lowest SBP and MAP values.

Definition of Post-Induction Hypotension: Post-induction hypotension was defined as either:

- a reduction in systolic blood pressure of ≥20% from the pre-induction baseline value, or
- an absolute systolic blood pressure <90 mmHg, occurring after induction of general anaesthesia and before significant surgical stimulation.

Patients were subsequently classified into two groups according to whether post-induction hypotension occurred:

- PIH group: patients who developed post-induction hypotension.
- Non-PIH group: patients who did not develop post-induction hypotension.

Management of Hypotension and Bradycardia: Hypotension was treated according to standard institutional practice. Initial management included intravenous fluid bolus administration and reduction of anaesthetic depth, where appropriate. If hypotension persisted or was clinically significant, intravenous mephentermine 3–6 mg in incremental boluses or phenylephrine 50–100 µg in incremental boluses was administered and repeated according to the haemodynamic response. Bradycardia associated with haemodynamic compromise was treated with intravenous atropine 0.6 mg, with additional doses administered when clinically indicated.

Study Outcomes: The primary outcome was the occurrence of post-induction hypotension and its association with pre-induction SI and MSI.

The secondary outcomes included comparison of SI and MSI between patients with and without PIH, determination of optimal cut-off values for predicting PIH, and comparison of their predictive accuracy using receiver operating characteristic analysis.

Data Collection: Demographic characteristics, ASA physical status, comorbidities, baseline haemodynamic variables, induction-agent doses, pre-induction SI and MSI, haemodynamic measurements after induction, occurrence of PIH, lowest recorded blood pressure, and requirement for vasopressor therapy were recorded in a predefined study proforma.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using appropriate statistical software such as SPSS version [27] (IBM Corp., Armonk, NY, USA).Continuous variables were expressed as mean ± standard deviation or median with interquartile range according to their distribution. Categorical variables were expressed as frequencies and percentages.

The normality of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables between patients with and without PIH were compared using the independent Student's t-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate.

The association of pre-induction SI and MSI with post-induction hypotension was assessed using logistic regression analysis. Receiver operating characteristic (ROC) curves were generated for SI

and MSI, and the area under the curve (AUC) with 95% confidence intervals was calculated.

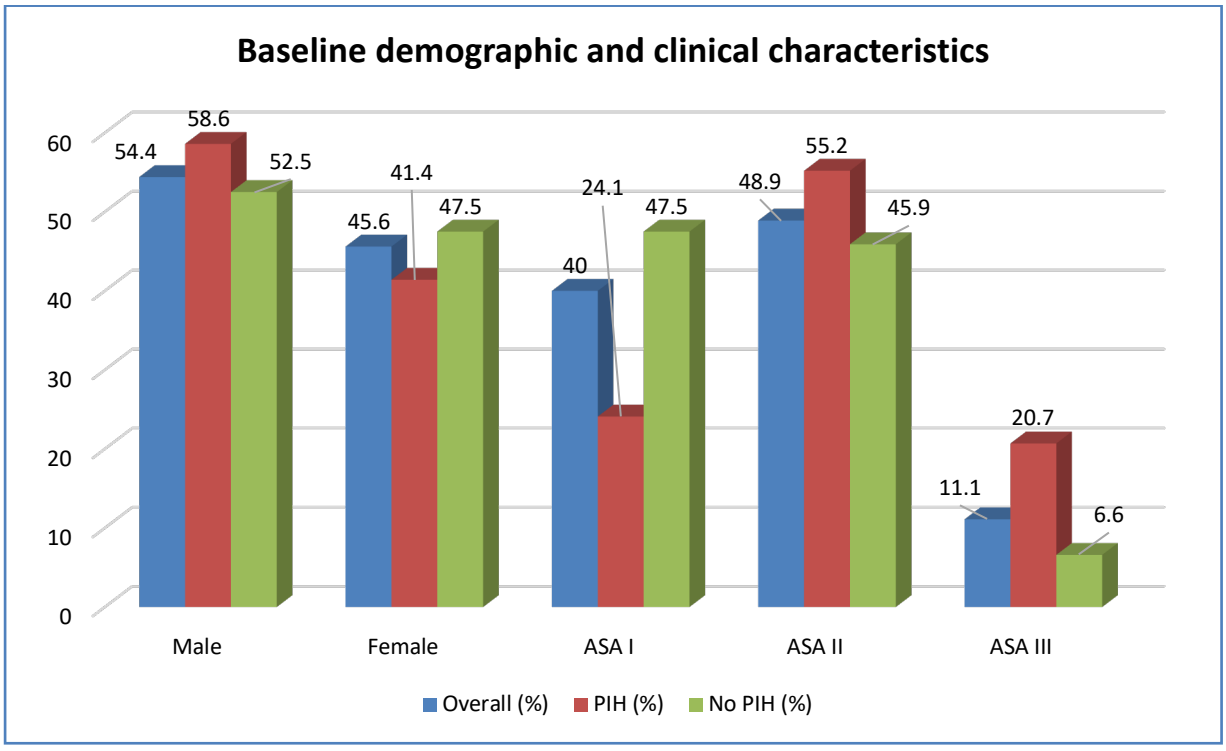
The optimal cut-off values were determined using the Youden index. Sensitivity, specificity, positive predictive value, negative predictive value, and diagnostic accuracy were calculated for the identified cut-offs. A p-value <0.05 was considered statistically significant.

Results

A total of 90 patients undergoing elective surgery under general anaesthesia were included in the study. Post-induction hypotension (PIH) occurred in 29 (32.2%) patients, while 61 (67.8%) patients remained haemodynamically stable during the post-induction period. The mean age of the study population was 44.1 ± 12.8 years, and 49 (54.4%) patients were male. Patients who developed PIH were significantly older than those who did not develop PIH (49.2 ± 11.6 vs. 41.7 ± 12.7 years; p=0.009). Sex distribution and body mass index were comparable between the groups. A higher proportion of patients with PIH belonged to ASA physical status III (20.7% vs. 6.6%; p=0.044) (Table 1, Figure 1).

Table 1: Baseline demographic and clinical characteristics

Variable	Overall (n=90)	PIH (n=29)	No PIH (n=61)	p-value
Age (years), mean ± SD	44.1 ± 12.8	49.2 ± 11.6	41.7 ± 12.7	0.009
Male, n (%)	49 (54.4)	17 (58.6)	32 (52.5)	0.585
Female, n (%)	41 (45.6)	12 (41.4)	29 (47.5)	
BMI (kg/m²), mean ± SD	24.8 ± 3.5	25.2 ± 3.7	24.6 ± 3.4	0.445
ASA I, n (%)	36 (40.0)	7 (24.1)	29 (47.5)	
ASA II, n (%)	44 (48.9)	16 (55.2)	28 (45.9)	0.044
ASA III, n (%)	10 (11.1)	6 (20.7)	4 (6.6)	



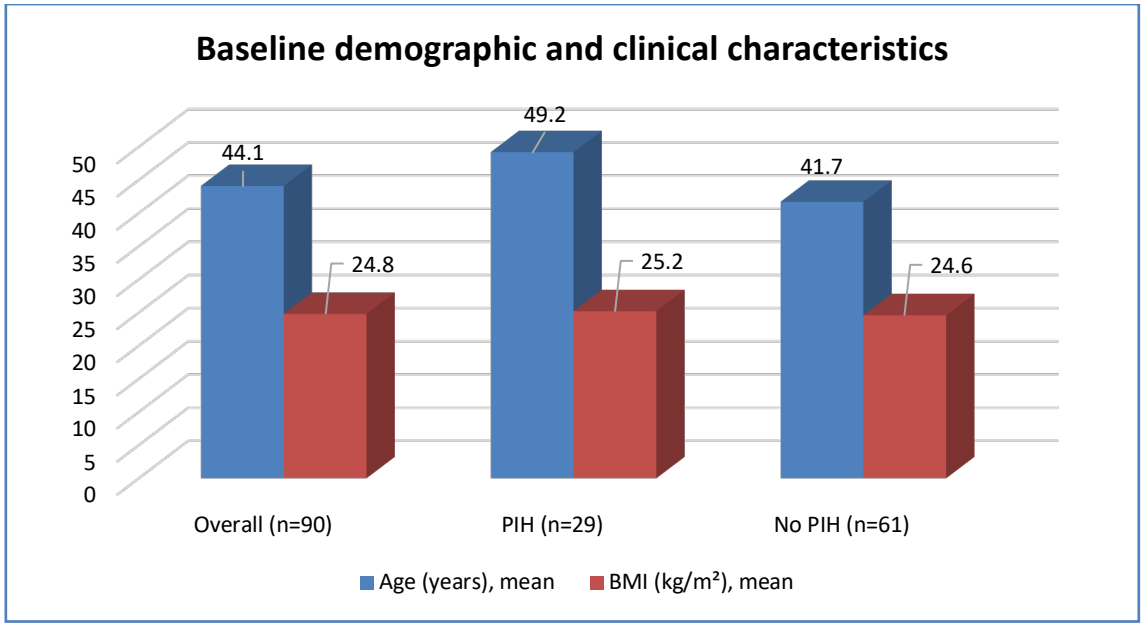


Figure 1: Baseline demographic and clinical characteristics

Pre-induction haemodynamic parameters are presented in Table 2, Patients who subsequently developed PIH had a significantly higher baseline HR (86.8 ± 10.9 vs. 78.1 ± 9.7 beats/min; p<0.001) and significantly lower SBP and MAP. Consequently, both pre-induction SI and MSI were significantly higher among patients who developed PIH.

Mean SI was 0.71 ± 0.11 in the PIH group compared with 0.59 ± 0.09 in the non-PIH group (p<0.001), while MSI was 0.91 ± 0.14 and 0.76 ± 0.11, respectively (p<0.001) (Table 2, Figure 2)

Table 2: Comparison of pre-induction haemodynamic parameters

Parameter	PIH (n=29)	No PIH (n=61)	p-value
Heart rate (beats/min)	86.8 ± 10.9	78.1 ± 9.7	<0.001
SBP (mmHg)	122.7 ± 11.5	133.1 ± 12.4	<0.001
DBP (mmHg)	75.4 ± 8.9	80.2 ± 9.1	0.020
MAP (mmHg)	95.6 ± 9.4	103.0 ± 9.7	0.001
Shock Index	0.71 ± 0.11	0.59 ± 0.09	<0.001
Modified Shock Index	0.91 ± 0.14	0.76 ± 0.11	<0.001

Values are expressed as mean ± SD.

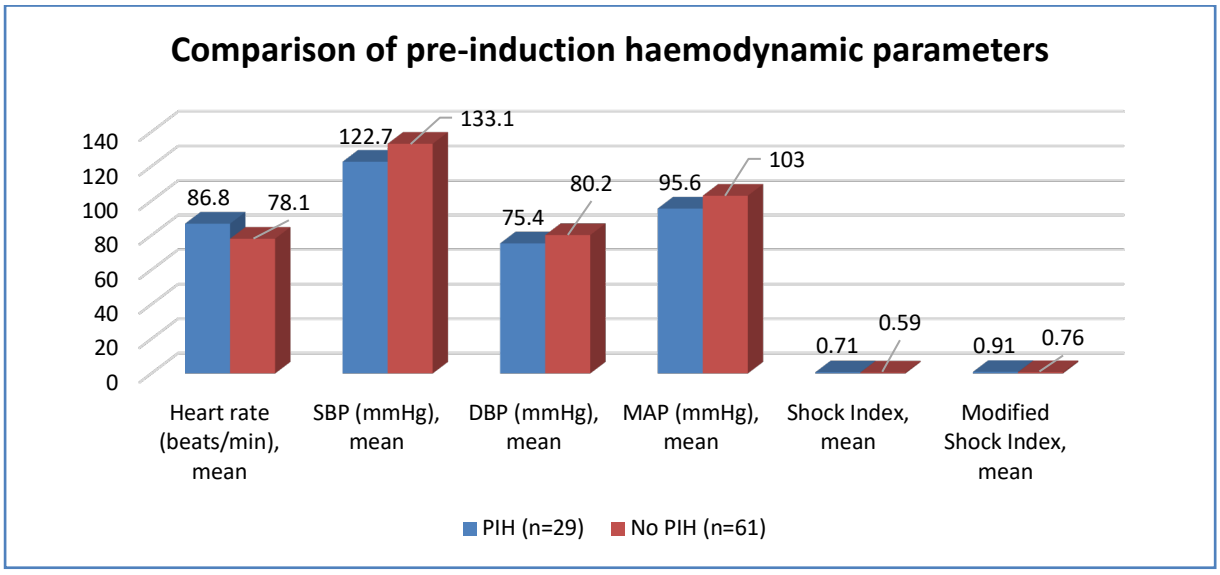


Figure 2: Comparison of pre-induction haemodynamic parameters

Following induction, a progressive reduction in arterial pressure was observed, with the greatest difference between groups occurring during the first 3 minutes.

At 1 minute after induction, MAP was 68.2 ± 8.7 mmHg in patients with PIH compared with 83.6 ± 9.1 mmHg in patients without PIH (p<0.001). This difference remained significant at 3 and 5 minutes.

By 10 minutes, MAP had partially recovered in both groups but remained significantly lower among patients who developed PIH (Table 3,Figure 3).

Table 3: Haemodynamic changes following induction

Time point	PIH (n=29)	No PIH (n=61)	p-value
SBP (mmHg)			
Baseline	122.7 ± 11.5	133.1 ± 12.4	<0.001
1 min	86.9 ± 10.4	113.7 ± 12.1	<0.001
3 min	88.6 ± 9.8	111.8 ± 11.7	<0.001
5 min	93.8 ± 10.5	114.5 ± 11.4	<0.001
10 min	105.2 ± 11.3	120.6 ± 11.8	<0.001
MAP (mmHg)			
Baseline	95.6 ± 9.4	103.0 ± 9.7	0.001
1 min	68.2 ± 8.7	83.6 ± 9.1	<0.001
3 min	67.5 ± 7.9	82.4 ± 8.8	<0.001
5 min	71.3 ± 8.1	84.7 ± 8.5	<0.001
10 min	79.8 ± 8.6	89.1 ± 8.9	<0.001

Values are expressed as mean ± SD.

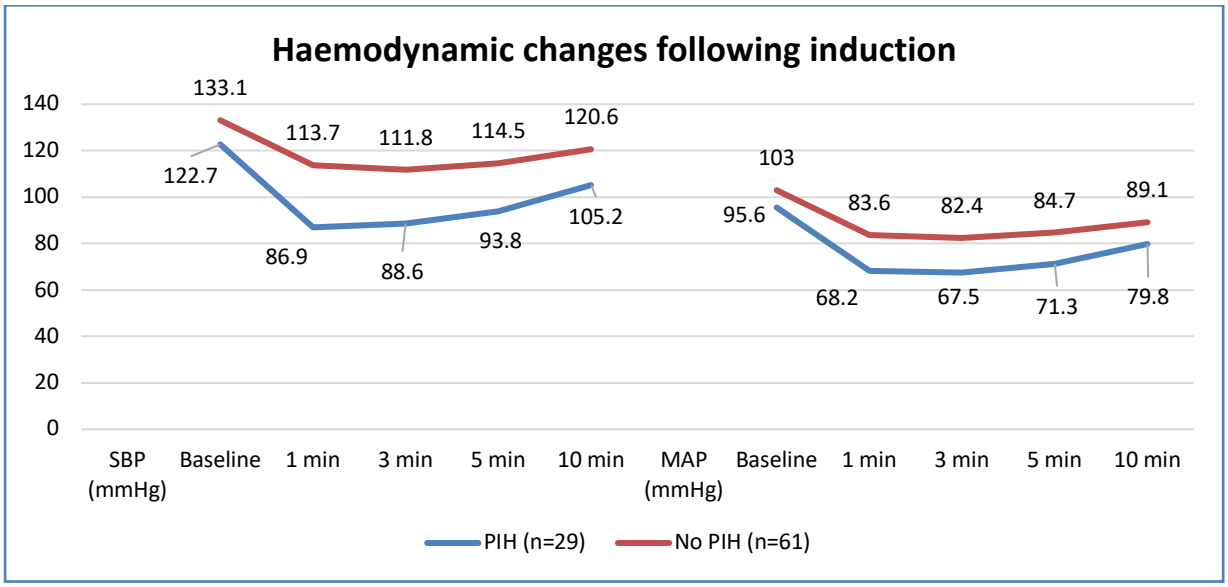


Figure 3: Haemodynamic changes following induction

Overall, 29 of 90 patients (32.2%) fulfilled the predefined criteria for PIH. Among these, 18 (62.1%) had a ≥20% reduction in SBP from baseline, while 11 (37.9%) recorded an absolute SBP <90 mmHg. Vasopressor treatment was required in 18 (62.1%) patients with PIH compared with 3 (4.9%) patients without PIH (p<0.001). Intravenous fluid bolus administration was also significantly more frequent in the PIH group (Table 4).

Table 4: Post-induction hypotension and interventions

Outcome	PIH (n=29)	No PIH (n=61)	p-value
Lowest SBP (mmHg), mean ± SD	84.7 ± 8.6	108.9 ± 10.2	<0.001
Lowest MAP (mmHg), mean ± SD	64.8 ± 7.3	79.6 ± 8.1	<0.001
SBP reduction ≥20%, n (%)	18 (62.1)	0	<0.001
SBP <90 mmHg, n (%)	11 (37.9)	0	<0.001
Vasopressor required, n (%)	18 (62.1)	3 (4.9)	<0.001
IV fluid bolus required, n (%)	15 (51.7)	8 (13.1)	<0.001
Bradycardia requiring treatment, n (%)	3 (10.3)	1 (1.6)	0.092

Receiver operating characteristic (ROC) analysis demonstrated that both pre-induction SI and MSI had good discriminatory ability for predicting PIH. SI yielded an area under the curve (AUC) of 0.804 (95% CI: 0.708–0.900; p<0.001), whereas MSI showed a higher AUC of 0.846 (95% CI: 0.761–0.931; p<0.001). An SI cut-off of ≥0.65 predicted PIH with 79.3% sensitivity and 72.1% specificity. An MSI cut-off of ≥0.85 demonstrated 82.8% sensitivity and 77.0% specificity. MSI therefore showed numerically better overall discrimination than SI (Table 5).

Table 5: ROC analysis of Shock Index and Modified Shock Index for prediction of PIH

Predictor	AUC (95% CI)	Optimal cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)	p-value
Shock Index	0.804 (0.708–0.900)	≥0.65	79.3	72.1	57.5	88.0	74.4	<0.001
Modified Shock Index	0.846 (0.761–0.931)	≥0.85	82.8	77.0	63.2	90.4	78.9	<0.001

On univariable analysis, increasing age, higher ASA physical status, lower baseline SBP, higher SI, and higher MSI were associated with PIH.

In multivariable logistic regression, MSI ≥0.85 remained independently associated with PIH (adjusted OR 4.82; 95% CI: 1.67–13.91; p=0.004). SI ≥0.65 was also associated with increased odds of PIH, although the strength of association was comparatively lower (adjusted OR 3.21; 95% CI: 1.13–9.12; p=0.028).

Increasing age remained an independent predictor (Table 6).

Table 6: Logistic regression analysis for predictors of post-induction hypotension

Predictor	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Age, per year increase	1.05 (1.01–1.09)	0.011	1.04 (1.00–1.09)	0.041
ASA III	3.92 (1.02–15.10)	0.047	2.48 (0.54–11.43)	0.244
Baseline SBP, per mmHg	0.93 (0.89–0.97)	<0.001	0.96 (0.91–1.01)	0.108
SI ≥0.65	6.84 (2.43–19.25)	<0.001	3.21 (1.13–9.12)	0.028
MSI ≥0.85	9.17 (3.09–27.22)	<0.001	4.82 (1.67–13.91)	0.004

Discussion

The present study evaluated Shock Index (SI) and Modified Shock Index (MSI) as predictors of post-induction hypotension (PIH) in 90 adults undergoing general anaesthesia. PIH occurred in 29 patients (32.2%). Patients who developed PIH had significantly higher pre-induction SI and MSI, and both indices demonstrated good discriminatory ability, with MSI showing numerically better predictive performance.

The incidence of PIH in our study was 32.2%, which was comparable with previous reports. A prospective study of 423 adult surgical patients reported PIH in 26.95% of cases and identified increasing age, higher ASA physical status, and use of propofol or thiopental as important predictors. Sun et al. [15] reported PIH in 40 of 94 patients (42.6%) undergoing elective abdominal surgery. In emergency airway studies, Lee et al. [16] reported PIH in 29.7% of 438 patients, while Heffner et al. [17] observed PIH in 23% of 336 patients. These variations likely reflect differences in patient profile, anaesthetic technique, and definitions of hypotension. Age was significantly higher in patients who developed PIH in our study (49.2 ± 11.6 vs. 41.7 ± 12.7 years; p=0.009). Increasing age remained independently associated with PIH (adjusted OR 1.04; p=0.041). Südfeld et al. [18] in 2,037 patients undergoing general anaesthesia, similarly demonstrated that increasing age independently predicted PIH (OR 1.03 per year) and that lower pre-induction systolic arterial pressure was also an independent risk factor. Pre-induction haemodynamic parameters differed significantly between groups. Patients developing PIH had higher HR (86.8 ± 10.9 vs. 78.1 ± 9.7 beats/min), lower SBP (122.7 ± 11.5 vs. 133.1 ± 12.4 mmHg), and lower MAP (95.6 ± 9.4 vs. 103.0 ± 9.7 mmHg). Correspondingly, SI was significantly higher in the PIH group (0.71 ± 0.11 vs. 0.59 ± 0.09; p<0.001), as was MSI (0.91 ± 0.14 vs. 0.76 ± 0.11; p<0.001). At a cut-off of ≥0.65, SI predicted PIH with 79.3% sensitivity, 72.1% specificity, and an AUC of 0.804. Heffner et al. [17] previously identified SI ≥0.8 as a predictor of post-intubation hypotension, with 67% sensitivity and 80% specificity. Trivedi et al. [19] reported that SI ≥0.90 independently predicted post-intubation hypotension in ICU patients, with an adjusted OR of 3.17. In our study, SI ≥0.65 was similarly independently associated with PIH (adjusted OR 3.21; p=0.028). MSI demonstrated slightly better performance than SI. At a cut-off of

≥0.85, MSI showed 82.8% sensitivity, 77.0% specificity, 90.4% negative predictive value, and an AUC of 0.846. MSI ≥0.85 was independently associated with PIH (adjusted OR 4.82; p=0.004). Liu et al., in 22,161 emergency patients, also demonstrated that MSI provided useful prognostic information and performed better than isolated heart rate or blood pressure measurements. However, Trivedi et al. [19] found MSI less useful than SI, while Lee et al. [16] reported Similar AUCs for SI and MSI (0.611 and 0.614, respectively). Thus, our findings suggest that both SI and MSI are useful bedside predictors of PIH, with MSI showing a modest advantage. As both indices are simple, non-invasive, inexpensive, and derived from routinely measured variables, they may help identify patients requiring closer haemodynamic monitoring, fluid optimization, titrated induction, and early vasopressor support. However, the relatively small sample size and single-centre design limit generalizability, and larger multicentre studies are required to validate the identified cut-off values.

Conclusion

Pre-induction Shock Index and Modified Shock Index were useful predictors of post-induction hypotension in patients undergoing general anaesthesia. Both indices were significantly higher in patients who developed hypotension, with MSI demonstrating slightly better predictive performance than SI. These simple, non-invasive, and readily available parameters may help identify patients at increased haemodynamic risk before induction. Their use may facilitate closer monitoring, timely fluid optimization, and early vasopressor support.

Limitations

The study was limited by its relatively small sample size and single-centre design, which may restrict the generalizability of the findings. Intermittent non-invasive blood pressure monitoring might have missed brief episodes of hypotension. SI and MSI may also be influenced by factors affecting heart rate and blood pressure, including medications, anxiety, comorbidities, and volume status. Larger multicentre studies are required to validate the proposed predictive cut-off values.

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