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Original Research Article

Prediction of Spinal Anaesthesia-Induced Hypotension Using Perfusion Index, Pleth Variability Index and Shock Index in Patients Undergoing Lower Abdominal and Lower Limb Surgeries

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Abstract:

Background: Spinal anaesthesia (SA) -induced hypotension is a common complication caused by sympathetic blockade and reduced venous return. Early prediction may permit timely preventive measures.

Aim: To evaluate and compare perfusion index, pleth variability index and shock index for predicting hypotension following SA.

Methods: This prospective observational study included 142 adults undergoing lower abdominal or lower limb surgery under SA at NRI Institute of Medical Sciences, Visakhapatnam, from November 2025 to May 2026. Baseline haemodynamic variables, perfusion index, pleth variability index and shock index were recorded. Hypotension was defined as a ≥20% reduction in systolic or mean arterial pressure or systolic pressure below 90 mmHg. Receiver operating characteristic analysis and multivariable logistic regression were performed.

Results: Hypotension occurred in 58 patients (40.8%). Baseline perfusion index, pleth variability index and shock index were significantly higher in hypotensive patients. Perfusion index showed an AUC of 0.86 at a cut-off above 3.65, while pleth variability index showed an AUC of 0.84 at a cut-off above 16%. Shock index demonstrated modest accuracy with an AUC of 0.69. The combined model achieved an AUC of 0.91.

Conclusion: Perfusion index and pleth variability index were useful non-invasive predictors, while shock index had limited independent predictive value.

Keywords: Perfusion Index; Pleth Variability Index; Shock Index; Spinal Anaesthesia; Hypotension.

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Introduction

Spinal anaesthesia (SA) is widely preferred for lower abdominal and lower limb surgeries (LALLS) because it provides rapid onset, effective sensory and motor blockade, reduced airway manipulation and satisfactory postoperative analgesia. However, SA-induced hypotension remains a frequent complication resulting from sympathetic blockade, arterial vasodilatation, venous pooling and reduced venous return. Severe or prolonged hypotension may cause nausea, vomiting, dizziness, myocardial ischaemia, cerebral hypoperfusion and delayed recovery, particularly in elderly patients and those with limited cardiovascular reserve. Conventional blood pressure (BP) monitoring detects hypotension only after haemodynamic deterioration has occurred; therefore, simple preoperative predictors could facilitate early fluid optimisation, vasopressor preparedness and individualised monitoring. Perfusion index reflects peripheral vascular tone through the ratio of pulsatile to non-pulsatile blood flow, whereas the pleth variability index represents respiratory variation in the perfusion index and may indicate intravascular volume responsiveness. Recent evidence suggests that these pulse oximetry-derived indices possess moderate ability to predict hypotension following SA [1,2]. Shock index, calculated by dividing heart rate (HR) by systolic blood pressure (SBP), provides an additional simple measure of cardiovascular compensation, although its independent predictive performance may be limited [3]. Combining these physiologically distinct parameters may improve risk stratification compared with using any single index [4]. Therefore, the present study aimed to evaluate and compare the ability of baseline perfusion index, pleth variability index and shock index to predict SA-induced hypotension among patients undergoing LALLS.

Methods

This prospective observational study was conducted in the Department of Anaesthesiology at NRI Institute of Medical Sciences, Visakhapatnam, from November 2025 to May 2026. Adult patients aged 18–65 years, belonging to the American Society of Anesthesiologists physical status I or II, who were scheduled to undergo elective lower abdominal or lower limb surgery under SA were enrolled after obtaining approval from the Institutional Ethics Committee and written informed consent. Patients with uncontrolled hypertension, significant cardiac disease, arrhythmias, peripheral vascular disease, autonomic neuropathy, severe anaemia, sepsis, hypovolaemia, pregnancy, contraindications to SA, or those receiving vasoactive drugs were excluded. Patients in whom spinal block failed, required conversion to general anaesthesia, or had incomplete haemodynamic data were also excluded. Demographic and clinical information, including age, sex, body mass index, ASA physical status, diagnosis, type of surgery and duration of surgery, was recorded. All patients were advised standard fasting and premedication protocols. On arrival in the operating theatre, routine monitoring consisting of non-invasive blood pressure, electrocardiography and pulse oximetry was instituted. An intravenous line was secured, and crystalloid fluid was administered according to institutional practice.

Before SA, patients rested in the supine position for at least five minutes in a quiet operating room maintained at a stable temperature. Baseline HR, SBP, diastolic NP, mean arterial pressure (MAP) and oxygen saturation were recorded as the average of three consecutive readings obtained at one-minute intervals. A compatible pulse oximetry sensor capable of displaying perfusion index and pleth variability index was applied to the index finger of the hand without an intravenous cannula. The hand was kept immobile, warm and positioned at heart level. Perfusion index and pleth variability index values were recorded only after a stable plethysmographic waveform had been obtained for at least one minute. Shock index was calculated by dividing the baseline HR by the corresponding SBP. SA was administered under strict aseptic precautions in the sitting or lateral position at the L3–L4 or L4–L5 interspace using a 25- or 26-gauge spinal needle. After confirmation of free cerebrospinal fluid flow, a standard dose of hyperbaric bupivacaine, with or without an adjuvant according to the surgical requirement, was injected intrathecally. The patient was immediately placed in the supine position, and the sensory block level was assessed using loss of sensation to pinprick. HR and non-invasive NP were recorded every minute for the first ten minutes after SA and every three minutes thereafter until completion of surgery. SA-induced hypotension was defined as a reduction in SBP of at least 20% from baseline, a fall in MAP of at least 20%, or an absolute SBP below 90 mmHg. The time of onset, lowest recorded NP, number of hypotensive episodes and total vasopressor requirement were documented. Hypotension was treated with intravenous fluids, leg elevation and incremental doses of mephentermine or another vasopressor according to departmental protocol. Bradycardia, defined as a HR below 50 beats per minute, was treated with intravenous atropine when clinically indicated. Patients were classified into hypotension and non-hypotension groups. Baseline perfusion index, pleth variability index and shock index were compared between the groups. Data were analysed using SPSS version 23. Continuous variables were expressed as mean with standard deviation or median with interquartile range, while categorical variables were presented as frequency and percentage. Independent t-test or Mann–Whitney U test and Chi-square or Fisher's exact test were applied as appropriate. Receiver operating characteristic curve analysis was performed to determine optimal cut-off values, sensitivity, specificity, area under the curve and predictive accuracy of each index. Multivariable logistic regression was used to identify independent predictors of hypotension, and a p-value below 0.05 was considered statistically significant.

Results

Among the 142 patients, 58 (40.8%) developed SA-induced hypotension. Patients who developed hypotension were significantly older and more frequently achieved a sensory block level of T6 or above, while sex, body mass index, surgical category and duration of surgery were comparable between the groups (Table 1). Baseline perfusion index, pleth variability index and shock index were significantly higher in the hypotension group. The strongest between-group differences were observed for perfusion index and pleth variability index, both with p-values below 0.001 (Table 2). Patients with hypotension experienced a greater percentage reduction in SBP and significantly lower minimum systolic and MAPs. Vasopressor administration was required in 93.1% of hypotensive patients compared with 6.0% of non-hypotensive patients. Bradycardia, nausea and vomiting were also significantly more frequent in the hypotension group (Table 3). Receiver operating characteristic analysis showed that perfusion index had the highest individual predictive accuracy, with an area under the curve of 0.86 at a cut-off above 3.65. Pleth variability index demonstrated comparable predictive performance, whereas shock index showed only modest discrimination. The combined model incorporating all three indices achieved an area under the curve of 0.91, with 87.9% sensitivity and 83.3% specificity. In multivariable analysis, perfusion index and pleth variability index remained independent predictors of hypotension, while shock index was not independently significant (Table 4).

Table 1: Demographic and perioperative characteristics among the study members

Variable	Hypotension group (n=58)	Non-hypotension group (n=84)	Test statistic	p-value
Age*	48.6 ± 12.4	43.1 ± 13.0	t=2.52	0.013
Male**	34 (58.6)	52 (61.9)	χ^2=0.15	0.698
Female**	24 (41.4)	32 (38.1)		
BMI, kg/m²*	25.8 ± 3.6	24.9 ± 3.4	t=1.51	0.133
ASA physical status II**	35 (60.3)	37 (44.0)	χ^2=3.65	0.056
Lower abdominal surgery**	27 (46.6)	42 (50.0)	χ^2=0.16	0.688
Lower limb surgery**	31 (53.4)	42 (50.0)		
Duration of surgery, minutes*	92.4 ± 24.8	87.1 ± 22.6	t=1.32	0.19
Upper sensory level ≥T6**	26 (44.8)	20 (23.8)	χ^2=6.96	0.008
* mean ± SD; **n (%)				

Table 2: Baseline haemodynamic variables and predictive indices

Parameter	Hypotension group	Non-hypotension group	Test statistic	p-value
HR in beats/min	83.7 ± 10.8	79.9 ± 9.7	t=2.20	0.029
SBP in mmHg	124.6 ± 12.2	129.8 ± 11.8	t=−2.55	0.012
MAP in mmHg	91.2 ± 8.9	94.1 ± 8.4	t=−1.98	0.049
Perfusion index	4.82 ± 1.46	2.91 ± 1.17	t=8.62	<0.001
Pleth variability index, %	19.7 ± 5.2	13.4 ± 4.3	t=7.90	<0.001
Shock index	0.68 ± 0.11	0.62 ± 0.09	t=3.58	<0.001

Table 3: Intraoperative haemodynamic outcomes

Outcome	Hypotension group	Non-hypotension group	Test statistic	p-value
Maximum SBP fall*	28.6 ± 6.4	11.7 ± 4.9	t=17.81	<0.001
Lowest SBP, mmHg	82.9 ± 8.6	109.7 ± 9.8	t=−16.75	<0.001
Lowest MAP, mmHg	61.8 ± 7.2	78.9 ± 7.5	t=−13.60	<0.001
Time to hypotension, minutes	6.8 ± 3.1	—	—	—
Recurrent hypotension**	19 (32.8)	0	—	—
Vasopressor requirement**	54 (93.1)	5 (6.0)	χ^2=109.23	<0.001
Mephentermine dose, mg	8.9 ± 4.1	0.4 ± 1.7	U=268.0	<0.001
Bradycardia**	10 (17.2)	4 (4.8)	χ^2=6.11	0.013
Nausea or vomiting**	13 (22.4)	5 (6.0)	χ^2=8.50	0.004
*Maximum reduction in SBP from baseline, %, mean ± SD; **n (%)				

Table 4: Predictive performance and multivariable analysis

Predictor	Optimal cut-off	AUC (95% CI)	Sen	Spe	Adjusted OR (95% CI)	p-value
Perfusion index	>3.65	0.86 (0.80–0.92)	82.80%	78.60%	2.46 (1.71–3.54)	<0.001
Pleth variability index	>16%	0.84 (0.77–0.90)	79.30%	76.20%	1.29 (1.16–1.44)	<0.001
Shock index	>0.65	0.69 (0.60–0.78)	65.50%	64.30%	1.18 (0.86–1.63)	0.306
Combined PI + PVI + SI model	—	0.91 (0.86–0.96)	87.90%	83.30%	—	<0.001
Sensitivity (Sen); Specificity (Spe)						

Discussion

The present prospective study evaluated baseline perfusion index, pleth variability index and shock index as non-invasive predictors of SA-induced hypotension among 142 patients undergoing LALLS. Hypotension occurred in 58 patients, giving an incidence of 40.8%. This finding confirms that hypotension remains a frequent complication of SA even in predominantly ASA physical status I–II patients undergoing elective non-obstetric surgery. The haemodynamic response to SA is primarily caused by sympathetic blockade, which produces arterial and venous vasodilatation, peripheral pooling of blood, reduction in systemic vascular resistance and decreased venous return. A reduction in cardiac output may become particularly prominent when sympathetic blockade is extensive or when the patient has reduced intravascular volume or limited cardiovascular reserve. Hofhuizen et al. demonstrated that hypotension following SA may result from a reduction in systemic vascular resistance, cardiac output or both, with elderly patients showing greater vulnerability to impaired compensatory mechanisms [5]. Similarly, Yotsuya et al. identified volume deficit and extensive sympathetic blockade as important contributors to intraoperative hypotension during SA [6]. The incidence observed in the present study was clinically important because hypotensive patients experienced greater reductions in systolic and MAPs, more frequent vasopressor administration, bradycardia, nausea and vomiting. These findings support the need to identify susceptible patients before administering SA rather than relying exclusively on intermittent NP measurements after haemodynamic deterioration has already developed.

Patients who developed hypotension were significantly older than those who remained haemodynamically stable, with mean ages of 48.6 ± 12.4 and 43.1 ± 13.0 years, respectively. Furthermore, a sensory block reaching T6 or above was significantly more common in the hypotension group. Increasing age is associated with reduced baroreceptor responsiveness, diminished β-adrenergic sensitivity, impaired autonomic compensation, increased vascular stiffness and a lower ability to augment HR and vascular resistance after sympathetic blockade. Akelma et al., in a prospective study of patients undergoing lower-extremity orthopaedic surgery, reported that SA-induced hypotension was particularly relevant among older patients and demonstrated the potential value of PI for preoperative risk assessment [4]. Background clinical factors should therefore be considered together with pulse oximetry-derived indices. The significant association between a higher sensory block and hypotension in the current study was physiologically expected because a more extensive block causes a greater reduction in sympathetic vascular tone and venous return. Silwal et al. also found that a high thoracic sensory block was associated with post-spinal hypotension, although their population consisted of women undergoing non-elective caesarean section [3]. In the present study, sex, body mass index, surgical category and duration of surgery were not significantly different between groups. These findings suggest that the observed differences in PI and PVI were unlikely to be explained merely by imbalance in these characteristics. However, age and block height should remain part of any clinically useful predictive model because no single peripheral perfusion parameter fully represents the multifactorial haemodynamic response to SA.

Baseline perfusion index was substantially higher in patients who subsequently developed hypotension, with mean values of 4.82 ± 1.46 compared with 2.91 ± 1.17 in the non-hypotension group. PI reflects the ratio between the pulsatile and non-pulsatile components of the photoplethysmographic signal. A higher baseline PI generally indicates lower peripheral vascular tone and greater peripheral vasodilatation. Such patients may have less capacity for compensatory vasoconstriction when SA produces additional sympathetic blockade. In the present study, PI produced an AUC of 0.86, with an optimal cut-off above 3.65, sensitivity of 82.8% and specificity of 78.6%. These findings were comparable to several previous investigations. Harde et al. reported an AUC of 0.917 for baseline PI and found that a PI above 2.9 predicted post-spinal hypotension with 83.08% sensitivity and 96% specificity in parturients [7]. Nandini et al. observed that patients with a baseline PI of at least 3.5 had a markedly higher incidence of hypotension and suggested that values above 3.9 increased specificity [8]. Inamanamelluri et al. similarly reported a significant relationship between elevated baseline PI and hypotension following subarachnoid block [9]. Jabarulla et al. also supported the role of PI as an easily obtained predictor in patients undergoing caesarean delivery [10]. Although earlier studies were obstetric, the present findings extend the usefulness of PI to a mixed non-obstetric population undergoing LALLS. The cut-off differences between studies are likely attributable to variations in patient age, pregnancy-related vasodilatation, room temperature, sensor site, spinal drug dose, fluid administration, hypotension definitions and timing of haemodynamic measurements.

Pleth variability index was also significantly higher in the hypotension group, with mean values of 19.7 ± 5.2% compared with 13.4 ± 4.3% among patients without hypotension. At a cut-off above 16%, PVI achieved an AUC of 0.84, sensitivity of 79.3% and specificity of 76.2%. PVI represents respiratory variation in the perfusion index over a respiratory cycle and is influenced by intravascular volume, respiratory pattern, vascular tone and cardiopulmonary interaction. A higher PVI may indicate greater respiratory variation in peripheral pulsatile flow and possible preload dependence. When sympathetic blockade reduces venous return, patients with reduced effective circulating volume or greater preload responsiveness may be more likely to develop hypotension. Sun et al. evaluated both PI and PVI before SA and demonstrated that pulse oximetry-derived parameters could contribute to the prediction of hypotension [11]. Yüksek et al. reported that PVI predicted a reduction in MAP among geriatric patients, although its discrimination was modest, with an AUC of 0.651 and a cut-off of approximately 15.45 [12]. The cut-off found in the present study was close to this value. A recent systematic review and meta-analysis by Kondo et al., including 19 studies and 1,437 patients, concluded that PI and PVI possessed moderate predictive ability, with summary AUC values of approximately 0.75 [1]. The higher AUCs in the current study may reflect standardised baseline measurements, a relatively homogeneous elective surgical population and the use of repeated measurements after a period of supine rest. Nevertheless, PVI can be affected by spontaneous breathing irregularity, movement, anxiety, peripheral temperature, low signal quality and changes in vasomotor tone. It should therefore be interpreted only after confirming a stable plethysmographic waveform.

Shock index was significantly higher in patients who developed hypotension, with values of 0.68 ± 0.11 compared with 0.62 ± 0.09. However, its AUC was only 0.69, with sensitivity of 65.5% and specificity of 64.3% at a cut-off above 0.65. Moreover, shock index did not remain an independent predictor in multivariable analysis. Shock index combines HR and SBP and can reveal compensated circulatory stress when either variable alone remains within its conventional reference interval. In the present study, the hypotension group had a higher baseline HR and lower SBP, explaining the higher calculated index. Nevertheless, relatively small changes in anxiety, pain, medication, hydration and resting HR can influence shock index. Furthermore, healthy elective surgical patients may maintain a normal HR despite reduced preload, thereby limiting the index's sensitivity. Silwal et al. found that a preoperative shock index of at least 0.9 was associated with post-spinal hypotension during non-elective caesarean delivery, but shock index alone had limited discrimination, with AUCs of only 0.53–0.56 [3]. Their conclusion that SI becomes more useful when integrated with other risk factors is consistent with the present findings. The lower optimal cut-off of 0.65 in the current study may be explained by differences between non-pregnant elective patients and emergency obstetric patients, in whom physiological increases in HR and altered vascular resistance are common. Thus, SI may serve as a simple supplementary bedside indicator but should not replace direct assessment of peripheral vascular tone or volume-related dynamic indices. The lack of independent significance after adjustment indicates that SI may largely reflect information already contained within HR, systolic pressure, PI and PVI.

Patients who developed hypotension experienced a maximum SBP reduction of 28.6 ± 6.4%, compared with 11.7 ± 4.9% in the non-hypotension group. Their lowest SBP and MAP were also substantially lower. Vasopressors were required in 93.1% of hypotensive patients but in only 6% of patients without hypotension, demonstrating that the study definition corresponded to clinically meaningful haemodynamic instability. Bradycardia occurred in 17.2% of hypotensive patients compared with 4.8% of stable patients, while nausea or vomiting occurred in 22.4% and 6%, respectively. Reduced venous return after SA can activate cardioinhibitory reflexes and cause simultaneous hypotension and bradycardia. Cerebral and gastrointestinal hypoperfusion may contribute to nausea, dizziness and vomiting. The average time to hypotension was 6.8 ± 3.1 minutes, indicating that the period immediately after intrathecal injection is critical. This early onset also strengthens the clinical relevance of obtaining predictive indices before the block. Preoperative identification of high-risk patients could permit closer NP monitoring, timely co-loading with crystalloid, preparation of vasopressor boluses or infusions and adjustment of intrathecal local anaesthetic dose. Mendonça et al. reported that ondansetron may reduce SA-induced hypotension and vasopressor use in non-obstetric surgery, although preventive pharmacological strategies should complement rather than replace appropriate monitoring [13]. Lal et al. demonstrated that preoperative inferior vena cava assessment could predict hypotension, supporting the broader principle that pre-anaesthetic evaluation of volume status may improve risk stratification [14]. Low-dose spinal techniques and combined regional approaches may also reduce severe haemodynamic disturbance in vulnerable patients, particularly those undergoing lower-limb orthopaedic procedures [15].

The combined model incorporating PI, PVI and SI achieved the best predictive performance, with an AUC of 0.91, sensitivity of 87.9% and specificity of 83.3%. This improvement was physiologically plausible because the three parameters describe different, although partially overlapping, components of cardiovascular status. PI primarily reflects peripheral vascular tone, PVI reflects respiratory variation and potential preload dependence, and SI reflects the relationship between HR and systolic pressure. A composite model may therefore recognise patients with vasodilated circulation, reduced effective preload or early compensatory cardiovascular stress more effectively than any single variable. The superiority of combined prediction is consistent with the observation by Silwal et al. that integrating SI with clinical factors improved model performance [3], and with broader evidence showing that multimodal models generally outperform isolated indices. However, the combined model should undergo external validation before routine application. PI and PVI are sensitive to environmental temperature, probe position, movement, nail abnormalities, peripheral vascular disease and device-specific algorithms. The observational, single-centre design may limit generalisability, and the inclusion of different LALLS could have introduced variability in patient positioning, block height and surgical stimulation. In addition, maximum percentage fall in systolic pressure was an outcome closely related to the definition of hypotension and should not be interpreted as an independent predictor. Despite these considerations, the study suggests that pre-spinal PI and PVI are practical, rapid and non-invasive predictors, whereas SI provides limited additional value when considered alone. The findings agree with recent meta-analytic evidence that pulse oximetry-derived indices have moderate predictive accuracy and support their integration with age, baseline haemodynamics, sensory block level and clinical assessment [1, 16]. Such risk stratification may allow targeted prophylaxis while avoiding unnecessary fluid loading or vasopressor administration in low-risk patients.

Conclusion: The study demonstrated that SA-induced hypotension occurred frequently among patients undergoing LALLS. Baseline perfusion index and pleth variability index were significantly higher in patients who developed hypotension and showed good predictive accuracy. Perfusion index was the strongest individual predictor, while shock index showed only modest discrimination and was not independently significant. Combining all three indices improved overall predictive performance. Preoperative assessment of these simple, non-invasive parameters may help identify high-risk patients, facilitate timely fluid and vasopressor preparation, and reduce haemodynamic complications following SA. Multicentre validation is required before routine clinical application.

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