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Original Research Article

Correlation of perfusion index with baseline heart rate and post spinal hypotension in pregnant parturients undergoing lower segment cesarian section: A prospective observational study

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Abstract

Background: Post-spinal hypotension is a frequent complication in parturients undergoing lower segment cesarean section (LSCS). Perfusion Index (PI), a non-invasive indicator derived from pulse oximetry, is being explored as a predictor for intraoperative hypotension. This study aimed to evaluate the correlation between pre-induction PI, baseline heart rate (HR), and the incidence of post-spinal hypotension.

Materials and Methods: A prospective observational study was conducted on 150 pregnant women undergoing elective LSCS under spinal anaesthesia. Pre-induction PI and HR were recorded, and hemodynamic changes were monitored post-spinal block. Hypotension was defined as a >20% fall in baseline systolic blood pressure (SBP) or mean arterial pressure (MAP). Statistical analyses included Mann-Whitney U test, chi-square test, ROC curve analysis, and correlation coefficients.

Results: The incidence of SBP-based hypotension was 36.7%, and MAP-based hypotension was 52%. No statistically significant correlation was found between PI and hypotension ($p>0.05$). ROC analysis revealed poor predictive accuracy for PI, with an AUC of 0.542 for SBP and 0.534 for MAP. However, patients who developed hypotension had significantly higher pre-induction HRs (SBP: 97.02 ± 14.73 vs. 88.4 ± 13.67 bpm; MAP: 94.44 ± 15.23 vs. 88.44 ± 13.35 bpm; $p<0.05$).

Conclusion: Pre-induction PI alone is not a reliable predictor of post-spinal hypotension. However, elevated baseline heart rate is significantly associated with hypotension and may serve as a more useful clinical marker.

Keywords: Perfusion index, Heart rate, Spinal anaesthesia, Hypotension, Cesarean section, LSCS, Predictive marker.

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1. Introduction

Spinal anaesthesia is the preferred anaesthetic technique for lower-segment cesarean section (LSCS) due to its ease of administration, rapid onset, and ability to provide adequate surgical anaesthesia along with postoperative analgesia. However, one of its commonest complications is post-spinal hypotension, which can significantly impact both maternal and fetal well-being. The incidence of hypotension following spinal anaesthesia is considerably higher in pregnant women (60-80%) compared to the general population (15-33%).[1] The primary causes of post-spinal hypotension include sympathetic blockade, decreased vascular resistance, low volume status, reduced cardiac output, and peripheral venous pooling due to aortocaval compression. Research indicates that peripheral vascular tone decreases in term parturients due to various factors.[2-6] In multiparous women, this decline results in pooling of blood in the extremities, even before the administration of spinal anaesthesia.[7] Additionally, pregnant women exhibit heightened sensitivity to local anaesthetics and decreased responsiveness to vasopressors, further exacerbating hypotension.[8] This hypotension may cause

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severe adverse effects in mothers, including nausea, vomiting, and dizziness, and may also contribute to the umbilical arterial acidosis and bradycardia in infants.[9]

Various parameters like perfusion index (PI), plethysmographic variability index (PVI), heart rate (HR), and heart rate variability (HRV) may serve as predictors of hypotension.[10] Earlier plethysmographic pluse wave amplitude (PPWA) was assessed either visually or using specialized hardware and connections. Now-a-days, a numerical value representing PPWA, the PI, has been incorporated into the modern pulse oximeters.[11]

PI is defined as the ratio of pulsatile blood flow to non-pulsatile blood flow in the peripheral vascular tissue. PI is influenced by the vascular tone of peripheral blood vessels.[12] A higher baseline PI has been associated with an increased risk of post-spinal hypotension, as it indicates a lower vascular tone and a greater tendency for blood pooling. The PI is calculated using the formula PI = [AC/DC] × 100, where AC represents the variation in infrared light absorption caused by the changing diameters of pulsatile arterial vessels, and DC corresponds to the constant absorption by non-pulsatile components such as venous blood, bone, and soft tissue.[13] This index reflects the ratio of pulsatile to non-pulsatile blood flow at the sensor site, providing a non-invasive estimate of the peripheral perfusion. Normally, the PI ranges from 0.02% to 20%, with lower values indicating weak pulse signals or high vasomotor tone, and higher values representing strong pulse signals or low vasomotor tone.[11] This physiological variability makes PI a potentially useful, yet complex, parameter for assessing circulatory dynamics.

One previous study set the cutoff PI at 3.5 and found that 60% of cases with PI >3.5 developed hypotension, with sensitivity of 81% and specificity of 86%.[7] They concluded that a higher PI is associated with a greater incidence of hypotension. Another study reported an increase in the incidence of hypotension with PI >3.85, with a sensitivity of 69.84% & specificity of 82.28%.[8] In contrast, Yokose et al reported no association between PI and hypotension.[9] They concluded instead that pre-anaesthetic heart rate is a predictor of hypotension after spinal anaesthesia in LSCS, with a sensitivity of 86% and specificity of 50%. Given the discrepancy in existing literature, additional studies are necessary to draw definitive conclusions.

We hypothesized that PI is an indicator of post-spinal hypotension in patients undergoing LSCS. This study was planned with the primary objective of finding correlation of PI with post-spinal hypotension during LSCS. The secondary objectives were to find out correlation of baseline heart rate with baseline PI, correlation of baseline HR with post-spinal hypotension, baseline cut-off of PI to predict hypotension, and number of patients requiring phenylephrine to treat hypotension.

2. Materials and Methods

This prospective observational study was conducted at a tertiary care center from July to December 2024 after obtaining Ethics Committee (I.E.S.C./416/2024) and CTRI approval (CTRI/2024/06/069553). Inclusion criteria were age 20–35 years, gestational age 36–40 weeks, elective LSCS, and American Society of Anaesthesiologists (ASA) Physical Status II. Exclusion criteria included refusal to participate, emergency LSCS, body mass index (BMI) >40, gestational age >40 weeks, cardiac or cerebrovascular disease, coagulopathy, sensory block above T6 level, any contraindication to spinal anaesthesia, placenta previa, pregnancy induced hypertension, antepartum hemorrhage, and other comorbidities. Based on a study conducted by Dr. Duggappa et al.[8] (correlation coefficient 0.416), sample size was calculated to be 43 patients per group. A total of 150 patients were recruited for better validity and to account for any potential dropouts.

The procedure was explained to the patients and a written, informed consent was obtained. After necessary investigations, preloading was done with 500 ml Ringer's lactate (RL) over 15–20 minutes. Standard ASA monitors were attached. A pulse oximeter (Philips Efficia) was placed on the left middle finger and wrapped in a towel to minimize confounding factors like hypothermia and contamination by ambient light. The table was tilted 15° left to prevent aortocaval compression. Oxygen was administered at 4 L/min. After 5 minutes of stabilization, PI and HR were recorded every 20 seconds, and blood pressure (BP) every minute for 3 minutes. Measurements were repeated post-positioning for spinal anaesthesia.

Subarachnoid block was given at L3–L4 intervertebral space using a 26G Quincke Babcock needle with 2.2 ml of 0.5% heavy bupivacaine in sitting position and under aseptic precautions. A sensory level of T6 was targetted. PI, HR, systolic BP (SBP), and diastolic BP (DBP) were recorded every 2 minutes until delivery to avoid confounders like medications, bleeding, and uterine blood shifts. Hypotension was defined as >20% drop in SBP, and bradycardia as HR <50 bpm. Phenylephrine (100 mcg IV) was given for SBP <90 mmHg; bradycardia was treated with Atropine or Glycopyrrolate.

Categorical variables were expressed as numbers and percentages, and quantitative data as mean ± standard deviation (SD) or median with interquartile range (IQR). Data normality was assessed using the Shapiro-Wilk test. The Mann-Whitney U test was used for non-normally distributed quantitative data, and the Chi-square test for categorical variables. Point biserial correlation assessed the association between pre-induction PI and hypotension (based on SBP and MAP). Spearman correlation evaluated the relationship between PI and HR over time. ROC curve analysis determined the cutoff, sensitivity, specificity, and predictive

values of PI for hypotension. Data were analyzed using SPSS (IBM v25.0); $p < 0.05$ was considered to be significant.

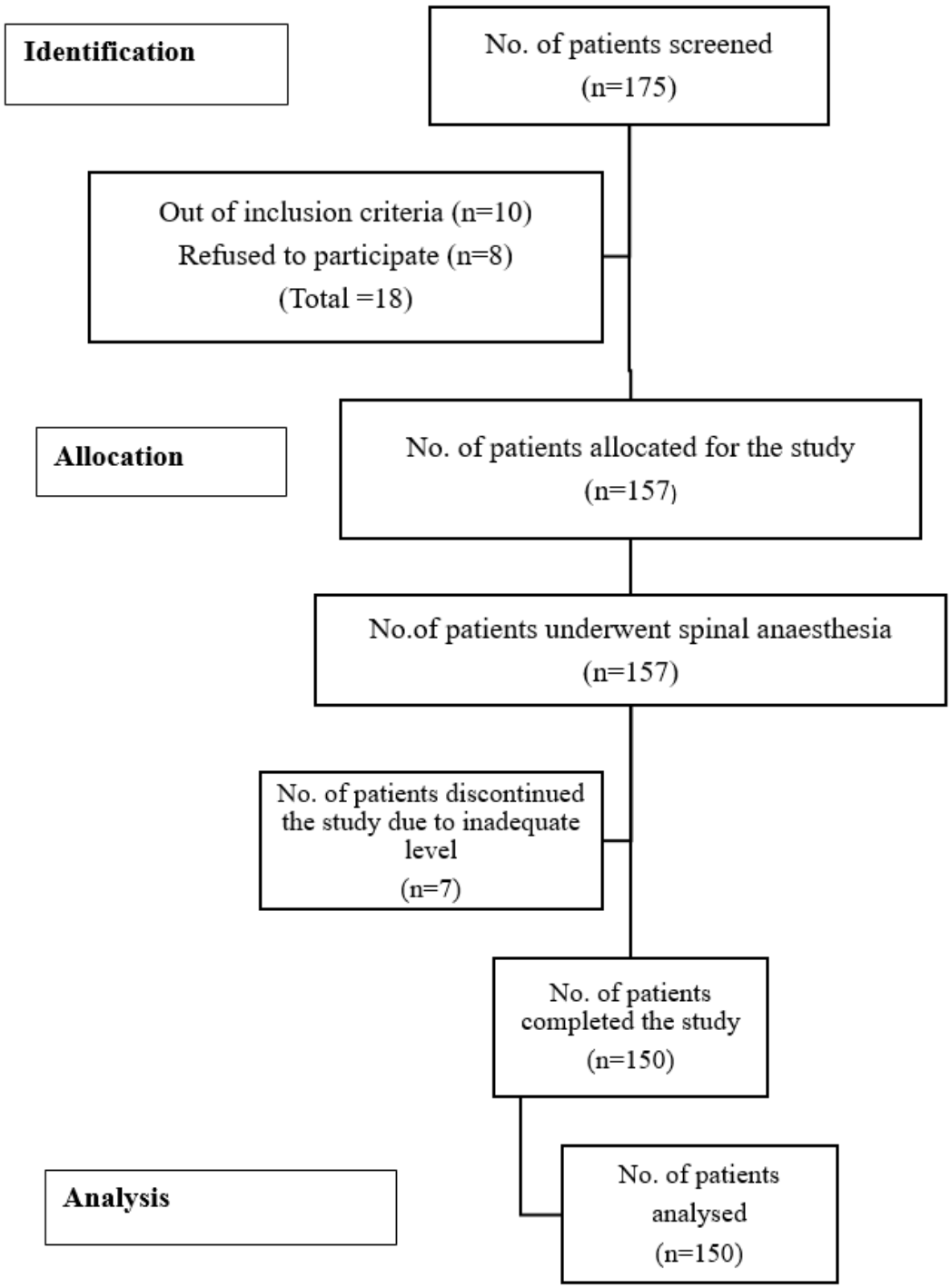

Figure 1: Consolidated standards of reporting trials (CONSORT) flow chart (n= Number of patients)

3. Results

Table 1: Patient characteristics

Parameter	Mean ± SD
Age	25.12 ± 3.8
Height (cm)	155.17 ± 6.92
Weight (kg)	64.8 ± 11.31
BMI (kg/m^2)	27.05 ± 5.16
Gestational age (weeks)	37.05 ± 1.46
Hemoglobin (g/dl)	11.44 ± 1.57

The mean pre-induction PI was 4 ± 3.13 and the pre-induction HR averaged 91.56 ± 14.62 bpm. PI and HR increased when the patients were seated for administering spinal anaesthesia and declined after administration. Mean pre-induction SBP was 118.19 ± 12.04 mmHg, with the lowest value of 109.52 ± 14.36 mmHg at 4 minutes post-induction. MAP also dropped significantly to 79.99 ± 13.07 mmHg at the same time point. (**Table 2**)

Association between pre-induction PI and hypotension is depicted in **Table 3**. The results suggest that PI is not a strong predictor of post-spinal hypotension.

Table 2: Descriptive statistics of perfusion index, heart rate, systolic blood pressure and mean arterial pressure

Time	PI (Mean ± SD)	HR (Mean ± SD)	SBP (Mean ± SD)	MAP (Mean ± SD)
Pre-induction	4 ± 3.13	91.56 ± 14.62	118.19 ± 12.04	89.9 ± 10.42
In sitting position	3.2 ± 2.36	96.01 ± 15.87	125.38 ± 12.9	94.59 ± 11.75
At 0 minute	3.32 ± 2.62	94.13 ± 14.87	119.48 ± 13.2	89.45 ± 11.61
At 2 minutes	3.39 ± 2.8	93.89 ± 16.46	110.92 ± 14.66	80.98 ± 12.38
At 4 minutes	3.57 ± 3.04	89.8 ± 16.38	109.52 ± 14.36	79.99 ± 13.07
At 6 minutes	3.34 ± 2.52	87.71 ± 16.76	109.21 ± 13.88	79.99 ± 11.94
At 8 minutes	3.51 ± 2.9	86.33 ± 15.87	110.74 ± 12.71	79.99 ± 10.92
At 10 minutes	3.5 ± 2.77	87.04 ± 15.16	111.58 ± 14.02	80.46 ± 10.68
At 12 minutes	3.71 ± 2.99	85.34 ± 14.19	110.9 ± 11.1	79.74 ± 9.76
At 14 minutes	3.32 ± 2.63	83.23 ± 13.58	112.29 ± 11.87	79.94 ± 11.27
At 16 minutes	3 ± 2.2	84.04 ± 14.08	112.64 ± 12.68	82.52 ± 10.62
At 18 minutes	3.38 ± 2.45	82.22 ± 14.96	108.56 ± 11.83	80.22 ± 8.83
At 20 minutes	3.96 ± 2.54	79.6 ± 6.88	113.2 ± 5.81	77.8 ± 5.26
At 22 minutes	2.6 ± 0	98 ± 0	122 ± 0	85±0

PI: Perfusion index, HR: Heart rate, SBP: Systolic blood pressure, MAP: Mean arterial pressure, SD: Standard deviation

Table 3: Association between hypotension and pre-induction perfusion index

Association of Hypotension with pre-induction PI						Association of pre-induction PI with hypotension		
	Hypotension	PI<3.5	PI>3.5	Total	p value	PI of patients with hypotension (Mean ± SD)	PI of patients without hypotension (Mean ± SD)	p value
SBP	No n (%)	61 (66.3%)	34 (58.6%)	95 (63.3%)	0.342	4.27+3.04	3.85+3.19	0.396
	Yes n (%)	31 (33.7%)	24 (41.4%)	55 (36.7%)				
	Total n (%)	92 (100%)	58 (100%)	150 (100%)				
MAP	No n (%)	45 (48.9%)	27 (46.5%)	72 (48%)	0.778	3.89+2.92	4.11+3.36	0.477
	Yes n (%)	47 (51.1%)	31 (53.4%)	78 (52%)				
	Total n (%)	92 (100%)	58 (100%)	150 (100%)				

PI: Perfusion index, SBP: Systolic blood pressure, MAP: Mean arterial pressure, n: Frequency, %: Percentage, SD: Standard deviation

Table 4: Receiver operating characteristic (ROC) curve of pre induction perfusion index for predicting hypotension

Variables	SBP	MAP
Area under the ROC curve (AUC)	0.542	0.534
Standard Error	0.0508	0.0478
95% Confidence interval	0.442 to 0.641	0.440 to 0.627
P value	0.4122	0.4818
PI Cut off	>6.2	≤1.7
Sensitivity (95% CI)	30.91% (19.1 - 44.8%)	32.05% (21.9 - 43.6%)
Specificity (95% CI)	83.16% (74.1 - 90.1%)	84.72% (74.3 - 92.1%)
PPV (95% CI)	51.5% (33.5 - 69.2%)	69.4% (51.9 - 83.7%)
NPV (95% CI)	67.5% (58.2 - 75.9%)	53.5% (43.9 - 62.9%)
Diagnostic accuracy	64.00%	57.33%

SBP: Systolic blood pressure, MAP: Mean arterial pressure, PI: Perfusion index, 95% CI: 95% confidence interval

Table 5: Association of requirement of phenylephrine with pre-induction perfusion index and heart rate

Pre-induction PI		<=3.5(n=92)	>3.5(n=58)	Total	P value
Requirement of Phenylephrine doses (Chi square test)	No n (%)	64 (69.57%)	38 (65.52%)	102 (68%)	0.605*
	Yes n (%)	28 (30.43%)	20 (34.48%)	48 (32%)	
	Total n (%)	92 (100%)	58 (100%)	150 (100%)	
Pre-induction HR (beats per minute) Mean ± SD		91.96 ± 14.29	90.93 ± 15.25	91.56 ± 14.62	0.677‡

PI: Perfusion index, HR: Heart rate, n: Frequency, %: Percentage, SD: Standard deviation

Table 6: Association of pre-induction heart rate with hypotension

Hypotension according to		Pre-induction HR (per minute)	p value
SBP (n=150)	No (n=95) Mean ± SD	88.4 ± 13.67	0.0004
	Yes (n=55) Mean ± SD	97.02 ± 14.73	
MAP (n=150)	NO (n=72) Mean ± SD	88.44 ± 13.35	0.012
	Yes (n=78) Mean ± SD	94.44 ± 15.23	

The study evaluated pre-induction PI as a predictor of hypotension. The AUC was 0.542 for SBP (p = 0.4122) and 0.534 for MAP (p = 0.4818), indicating weak predictive value. The optimal PI cut-off for hypotension was >6.2 (30.91% sensitivity, 83.16% specificity) for SBP, and ≤1.7 (32.05% sensitivity, 84.72% specificity) for MAP. (**Table 4**)

Hypotensive patients were found to have a significantly higher mean pre-induction HR than normotensive patients for both SBP-based (p = 0.0004) and MAP-based hypotension (p = 0.012). This suggests that elevated pre-induction HR increases the risk of post-spinal hypotension (**Table 6**).

4. Discussion

Hypotension is a common complication following spinal anaesthesia for cesarean delivery. Several studies have evaluated the role of PI in predicting hypotension after spinal anaesthesia in cesarean section. However, no reliable monitoring system has been found to predict its occurrence and allow timely preventive measures.

This study examined the correlation between PI and post-spinal hypotension in LSCS patients, attempted to identify a cutoff PI for predicting hypotension and also examined the relationship between pre-induction PI, HR and the number of Phenylephrine doses required. Additionally, the study also explored the correlation between HR and hypotension.

Previous studies have shown conflicting results on the predictive value of pre-induction PI for hypotension during cesarean section. Given PI's frequent fluctuations, the method of averaging significantly affects interpretation. In our study, we minimized variability by allowing 5 minutes for patient stabilization to reduce anxiety-related effects. PI and HR were then recorded every 20 seconds for 3 minutes, and BP

every minute for 3 minutes. Mean values from these recordings were taken as baseline.

Although PI has been suggested as a predictor of post-spinal hypotension, its predictive value was found to be very limited in our study. The mean pre-induction PI was 4 ± 3.13, with no significant correlation to hypotension incidence. In the PI <3.5 group, 33.7% developed SBP hypotension versus 41.38% in the PI >3.5 group (p = 0.342). For MAP-based hypotension, 51.09% with PI <3.5 and 53.45% with PI >3.5 developed hypotension (p = 0.778). These results indicate PI is not a strong predictor of post-spinal hypotension. Mean PI in hypotensive patients was found to be 4.27 ± 3.04 (SBP-based) and 3.89 ± 2.92 (MAP-based), versus 3.85 ± 3.19 and 4.11 ± 3.36, respectively, in non-hypotensive patients. These differences were not statistically significant (p > 0.05), supporting that PI alone is not a strong predictor of hypotension. Toyama et al.[7] reported that PI >3.5 predicted hypotension with 81% sensitivity and 86% specificity, this difference may be because they defined hypotension as a >25% drop in SBP and used 6% carboxyethyl starch for pre-loading. It's unclear if PI was measured before or after preloading, and their method for determining baseline PI was not specified. Where as in our study we defined hypotension as more than 20% fall in SBP and recorded base line PI after preloading with RL.

Malavika et al.[14] reported a cut off PI of 4.25 with sensitivity and specificity of 74.5% and 89.7%, respectively, to detect hypotension on parturients with non-severe pre-eclampsia undergoing LSCS. Joseph George et al[15] also concluded that parturient with baseline PI >3.6 are at higher risk of post spinal hypotension in LSCS. In our study, we excluded patients with pre-eclampsia, and hence, pre-eclampsia could have been a confounding factor in these studies.

Kumar et al.[12] concluded that patients with baseline PI >3.5 had a higher risk of developing post-spinal hypotension compared to those with PI <3.5. This difference in results could be due to the use of prophylactic phenylephrine infusion by Upendra Kumar et al. Duggappa et al.[8] reported a cutoff PI of 3.85 (69.84% sensitivity, 82.28% specificity) for predicting hypotension. This difference could originate from the fact that they defined hypotension as MAP less than 65 mmHg and measured PI before preloading the patients.

The area under the receiver operating characteristic (ROC) curve (AUC) for SBP was 0.542 (p = 0.4122) and for MAP 0.534 (p = 0.4818), indicating weak predictive ability. This again suggests that PI alone is unreliable for identifying patients at risk of post-spinal hypotension. The study found optimal PI cutoffs: for SBP hypotension, PI >6.2 had 30.91% sensitivity and 83.16% specificity; for MAP hypotension, PI ≤1.7 had 32.05% sensitivity and 84.72% specificity. These results show that while PI may reflect hemodynamic status, its predictive ability to detect hypotension is limited.

We also assessed relationship between baseline HR and baseline PI by Spearman rank correlation. Mean pre-induction HR was 91.96 ± 14.29 bpm for PI ≤3.5 and 90.93 ± 15.25 bpm for PI >3.5, with no significant difference (p = 0.677). While pre-induction PI and HR showed a downward trend post-spinal anaesthesia, the correlation strength between these parameters was not statistically significant. We could find no other study assessing correlation between these two variables.

We compared correlation of HR with hypotension and found that pre-induction HR in patients with hypotension (SBP) was 97.02 ± 14.73 per minute which was significantly higher as compared to patients without hypotension (88.4 ± 13.67 per minute) (p=0.0004). Similarly, pre-induction HR in patients with hypotension (MAP) was 94.44 ± 15.23 per minute which was again significantly higher as compared to patients without hypotension (88.44 ± 13.35 per minute) (p=0.012). Our findings align with those of Yokose et al.[4] who defined hypotension as SBP <80 mmHg and administered 6% carboxyethyl starch post-spinal anaesthesia. Using a PI cutoff of 3.5, they found PI unreliable and identified pre-anaesthetic HR as a better predictor (86% sensitivity, 50% specificity) for hypotension.

We assessed the relationship between pre-induction PI and phenylephrine requirement for managing post-spinal hypotension in LSCS patients. Phenylephrine was needed in 30.43% of patients with PI ≤3.5 and 34.48% with PI >3.5, with no significant difference (p = 0.605), suggesting PI is not a reliable predictor of vasopressor need (**Table 5**). In contrast, Inamanamelluri R et al.[1] found a significant difference, possibly due to using a lower PI cutoff (2.85) and defining hypotension based on MAP, influencing vasopressor administration.

Since PI is commonly influenced by factors such as movement, temperature, psychological stress, and anxiety, which can trigger sympathetic activation, a large sample size would have been desirable. The variation in fasting duration or decrease in oral intake due to anxiety might also have a confounding effect on PI. Future research on correlation of inferior vena cava diameter as a predictor of fluid status and PI is needed. Studies comparing invasive method of hemodynamic monitoring with PI may shed more light for its utility.

5. Conclusion

Perfusion index (PI) as a predictor of post-spinal hypotension lacks significant association with hypotension and does not support for standalone clinical use. Pre-induction heart rate (HR) shows better predictive value for post-spinal hypotension in patients undergoing lower segment cesarean section (LSCS).

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None.

7. Conflict of Interest

None.

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