

Spinal Anaesthesia-Induced Hypotension in Lower Segment Caesarean Section: Predictors and Prevention - A Prospective Observational StudySreeharsha S.¹, Aarti²¹Associate Professor, Department of Anaesthesia, Dr. Moopen's Medical College, Wayanad, Kerala, India²Assistant Professor, Department of Anaesthesia, Dr. Moopen's Medical College, Wayanad, Kerala, India

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Abstract**Background:** For lower segment caesarean sections (LSCS), spinal anaesthesia (SA) is the recommended anaesthetic method. Nonetheless, 50–80% of parturients experience spinal anaesthesia-induced hypotension (SAIH), a serious complication that results in decreased placental perfusion and poor maternal-fetal outcomes.**Goal:** The goal is to develop a useful risk stratification strategy for clinical application by identifying independent predictors of spinal anaesthesia-induced hypotension in parturients undergoing LSCS and assessing the effectiveness of various preventive strategies in various risk groups.**Materials and Methods:** Over the course of 18 months (January 2024 to June 2025), 286 women (ASA I–II, ages 18–45) undergoing LSCS under spinal anaesthesia participated in a prospective observational study. Clinical, haemodynamic, and demographic factors were methodically documented. Systolic blood pressure (SBP) of less than 90 mmHg, MAP of less than 65 mmHg, or a decrease of more than 20% from baseline were considered hypotension. To find independent predictors, univariate and multivariate logistic regression analyses were carried out. Preventive measures such as crystalloid preloading, positional modification, and vasopressor therapy were guided by risk stratification into low, moderate, and high-risk groups based on identified factors.**Results:** 18.5% of patients had severe hypotension (SBP <80 mmHg), and the overall incidence of SAIH was 67.8% (194/286). There were found to be four independent predictors: Age <30 years (OR 2.14, 95% CI 1.12–4.08, $p=0.018$), BMI <20 kg/m² (OR 2.87, 95% CI 1.35–6.11, $p=0.006$), baseline SBP <130 mmHg (OR 3.42, 95% CI 1.98–5.91, $p<0.001$), and sensory block height $\geq T5$ (OR 2.91, 95% CI 1.41–6.004). The incidence of SAIH differed significantly between risk groups: low-risk was 53.7%, moderate-risk was 69.5%, and high-risk was 83.6% ($p<0.001$). In patients at moderate risk, crystalloid preloading with 1000 ml decreased incidence by 22%, while in patients at high risk, it decreased incidence by 31%. Preloading left lateral tilt, and phenylephrine infusion (0.5 mcg/kg/min) together reduced risk by 78% in high-risk patients (18.3% incidence). Neonates in the high-risk group had higher NICU admission rates (16.4% vs. 2.1%, $p<0.001$) and significantly lower Apgar scores ($p=0.032$) and cord arterial pH (7.23 \pm 0.10 vs. 7.28 \pm 0.06 in low-risk, $p=0.001$).**Conclusion:** In conclusion, precise risk assessment and focused prevention in LSCS are made possible by the identification of four major predictive factors. In addition to improving maternal haemodynamic stability and neonatal outcomes, a multimodal approach that combines risk-based crystalloid preloading (500–1000 ml), positional modification (left lateral tilt), and prudent vasopressor therapy significantly reduces the incidence and severity of SAIH. This risk-stratified protocol can be implemented and ought to be used in clinical settings.**Keywords:** Maternal Hypotension, Spinal Anaesthesia, Caesarean Section, Predictors, Prevention, Vasopressors, Risk Stratification and Neonatal Outcomes.**DOI:** 10.25258/ijcpr.18.1.159This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Depending on the region and institutional practices, lower segment caesarean sections (LSCS) account for 15–45% of all deliveries worldwide, making them the most common obstetric surgical procedure.[1] Over the past 20 years, there has been a significant rise in the prevalence of caesarean deliveries due to a number of factors, including advanced maternal age, higher rates of

infertility treatment, fear of childbirth, and defensive medical practices.[2] Due to a number of benefits, including quick onset of action, better neural blockade quality, avoidance of general anaesthesia-related risks (aspiration, airway complications), preservation of maternal consciousness, decreased opioid use, and lower cost when compared to epidural anaesthesia, spinal

anaesthesia has become the preferred anaesthetic technique for LSCS in most parts of the world, including developing nations.[3] However, hypotension, which affects 50–80% of parturients undergoing LSCS, is one of the most common and clinically significant side effects of spinal anaesthesia in obstetric patients.[4] Numerous studies into mechanisms, predictors, and preventative measures have been spurred by this high incidence. SAIH continues to cause maternal morbidity and neonatal complications in many clinical settings worldwide despite decades of research and numerous clinical trials.[5]

The pathophysiology of Spinal Anesthesia-Induced Hypotension (SAIH) involves significant cardiovascular changes due to sympathetic nerve blockade. This leads to hypotension through several mechanisms: firstly, arteriolar vasodilatation occurs as alpha-1 mediated vasoconstriction is lost, decreasing peripheral vascular resistance by 20-50% and reducing mean arterial pressure (MAP). Secondly, there's venous pooling in lower extremity veins due to diminished sympathetic tone, which lowers venous return to the heart. This, combined with decreased left ventricular preload and cardiac output due to loss of sympathetic stimulation, results in further reduction of blood circulation. In pregnant patients, additional complications arise from aortocaval compression by the gravid uterus, which hinders venous return, a concern less prevalent in non-pregnant individuals undergoing spinal anaesthesia.[6]

During pregnancy, women experience marked changes in hemodynamics including a 40-50% increase in cardiac output, a rise in heart rate by 10-20 beats per minute, and a decrease in systemic vascular resistance by 20-30%. These physiological alterations, driven by increased metabolic demand, coupled with limited cardiovascular reserves, heighten susceptibility to hypotension following spinal anaesthesia. [7]

Clinically, maternal hypotension has serious implications for both mother and fetus. It adversely affects uterine artery perfusion, compromising blood flow to the placenta and thereby impairing fetal oxygenation, potentially causing fetal hypoxia.

Evidence indicates that episodes of maternal hypotension correlate with fetal desaturation and metabolic acidosis, leading to lower Apgar scores and higher lactate levels in neonates. Additionally, long-term consequences of perinatal hypoxia may result in cerebral palsy and learning disabilities. Severe hypotension might require emergency conversion to general anaesthesia, which poses additional risks such as aspiration and difficult airway management. Consequently, preventing

SAIH is essential not only for maternal comfort but also for the well-being of the fetus and to ensure optimal neonatal outcomes.

Despite the clinical significance of spinal anaesthesia-induced hypotension (SAIH), inadequate management persists, especially in developing countries where preventive measures are inconsistently applied. This has resulted in high complication rates related to SAIH. Various prevention strategies are under investigation, such as fluid administration, vasopressor use, positional modifications, abdominal compression, and adjustments in anesthetic techniques, although their relative effectiveness and optimal use remain debated. Factors like younger age and lower body mass index may predict SAIH, but their practical clinical applicability requires further validation. This study aims to identify independent predictors of SAIH in parturients undergoing lower segment caesarean section (LSCS), evaluate preventive strategies across different risk groups, develop an easy-to-implement risk stratification method, explore the link between maternal stability and neonatal outcomes, and offer standardized evidence-based recommendations for SAIH prevention adaptable to various clinical environments.

Materials and Methods

Study Design: A prospective observational study was formulated to examine the predictors and prevention of spinal anaesthesia-induced hypotension in women undergoing lower segment caesarean section. The research was carried out in the Department of Anaesthesia at Dr. Moopen's Medical College in Wayanad, Kerala, India, spanning 18 months from January 2024 to June 2025.

Ethical Approval: The Institutional Ethics Committee of Dr. Moopen's Medical College, Wayanad, reviewed and approved the study protocol. All participants provided written informed consent subsequent to a comprehensive elucidation of the study procedures in their native language.

Study Population: women aged 18 to 45 years, classified as American Society of Anaesthesiologists (ASA) physical status I or II, presenting for elective or emergency lower segment caesarean section (LSCS) under spinal anaesthesia, were eligible for enrolment.

Exclusion criteria were rigorously enforced to guarantee a uniform population:

- (1) Contraindications to spinal anaesthesia (patient refusal, coagulopathy, anticoagulation therapy, infection at puncture site, haemodynamic instability),

- (2) Pre-existing systemic hypertension on antihypertensive medications,
- (3) Gestational hypertension or preeclampsia,
- (4) Cardiac or cerebrovascular disease,
- (5) Diabetes mellitus,
- (6) Placental abruption or placenta previa,
- (7) Severe anaemia (Hb <7 g/dL),
- (8) Emergency hysterectomy or major blood loss anticipated, and (9) refusal of informed consent.

Sample Size Calculation: The sample size was determined using the following parameters:

- (1) A baseline incidence of SAIH at 65% in standard practice (control group)
- (2) An expected reduction of SAIH incidence to 52% with the implementation of preventive measures (20% relative reduction)
- (3) A two-sided significance level (α) of 0.05
- (4) A statistical power of 80% ($= 0.20$), and (5) an anticipated dropout rate of 10%.

Based on these factors, the sample size needed was found to be 286 women who were giving birth. We used G*Power version 3.1.9.7 to figure out how many samples we needed.

Recruitment of Participants: All parturients who came to the operating room for LSCS during the study period and met the inclusion criteria were asked to join. Trained research coordinators did the recruitment and gave detailed explanations of the study's goals, methods, possible benefits, and risks. Participants were assured that declining to participate would not impact their clinical care. To reduce selection bias, a consecutive enrolment method was used.

Pre-operative Assessment: The anaesthesia team did a standard pre-operative assessment on all of the women who were giving birth. Demographic information was collected, such as age (in years), height (in centimetres), body weight (in kilogrammes), and body mass index (BMI), which is weight in kg divided by height in meters squared. The obstetric history included the number of pregnancies, the number of births, the number of weeks of gestation, the reason for the caesarean section (elective or emergency), and the specific reason for the caesarean section (failed labour, cephalopelvic disproportion, foetal distress, etc.).

A medical history was obtained, emphasising cardiopulmonary disease, hypertension, diabetes, anaemia, haemorrhage risk factors, and prior anaesthetic experiences. Blood pressure (using a calibrated automated monitor), heart rate, respiratory rate, and oxygen saturation were all measured as vital signs.

Baseline Haemodynamic Measurements: To get accurate baseline haemodynamic values, the

women who were about to give birth were put in the left lateral decubitus position (to avoid aortocaval compression) and told to rest quietly for 5 minutes before the baseline measurements were taken. This standardisation reduced the confusing effects of pain, anxiety, and changes in position on baseline blood pressure. The baseline measurements taken were systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR).

Standardisation of Anaesthetic Technique: All parturients received a standardised pre-medication protocol 10 minutes before spinal puncture, which included intravenous glycopyrrolate 0.2 mg (an anticholinergic to prevent vagal bradycardia) and ondansetron 4 mg (a 5-HT₃ antagonist for antiemetic effect). To give spinal anaesthesia, the parturients were put in the left lateral position to reduce aortocaval compression.

Spinal anaesthesia was administered using a 25-gauge pencil-point Sprotte needle, adhering to stringent aseptic protocols at the L3-L4 or L4-L5 vertebral interspace, as determined by palpation of the iliac crests. To reduce variability in technique, only one person did all of the spinal punctures (either an attending anaesthesiologist or a senior resident under supervision).

The intrathecal injection contained: (1) 2.0-2.5 mL of 0.5% isobaric bupivacaine (10-12.5 mg) and (2) 20 microgrammes of fentanyl. The dose of bupivacaine (2.0-2.5 mL) was chosen using a standard method in which the dose was adjusted for each patient based on their height and BMI to get the right level of sensory blockade (T6-T8) for LSCS. The intrathecal medications were injected at a steady rate of about 0.5 mL every 3 seconds.

After the spinal injection, the women giving birth were put on their backs with their left hips raised 15 degrees (a bolster under their right hip kept them tilted to the left throughout the surgery) to help with aortocaval decompression and improve uteroplacental perfusion.

Haemodynamic Monitoring: An automated oscillometric blood pressure monitor (Model: Philips IntelliVue MP50 or equivalent) was used to continuously monitor haemodynamics without any invasive procedures.

At the following set times, we wrote down the person's blood pressure (systolic, diastolic, and mean arterial pressure), heart rate, and oxygen saturation: (1) baseline (before the injection), (2) 1 minute after the injection, (3) 3 minutes after the injection, (4) 5 minutes after the injection, (5) 10 minutes after the injection, (6) 15 minutes after the injection, (7) 20 minutes after the injection, and then every 5 minutes until the end of the surgery.

The monitor used the formula $MAP = [(SBP + 2 \times DBP) / 3]$ to figure out the mean arterial pressure on its own. Trained staff wrote down all of the haemodynamic measurements on a data collection form. The monitor alarm limits were set correctly so that staff would be warned about hypotensive episodes.

What is Hypotension?

Hypotension was defined according to established criteria from the literature: (1) systolic blood pressure below 90 mmHg, (2) mean arterial pressure below 65 mmHg, or (3) a reduction in systolic blood pressure exceeding 20% from the baseline value, whichever criterion was met first.

These criteria are frequently utilised in obstetric anaesthesia literature and signify clinically relevant haemodynamic disturbances that may compromise placental perfusion.

Severe hypotension was characterised by a systolic blood pressure of less than 80 mmHg, indicating a heightened level of haemodynamic instability necessitating prompt and vigorous intervention.

Risk Stratification: A literature review and initial data analysis were used to divide parturients into three risk groups based on the presence of certain risk factors (age, BMI, baseline SBP, and sensory block height).

The low-risk group (n=95) included parturients with a good haemodynamic profile, such as being 30 years old or older, having a BMI of 20 kg/m² or higher, and a baseline SBP of 130 mmHg or higher.

Moderate-risk group (n=118): Parturients exhibiting an intermediate risk profile characterised by the presence of one or two aforementioned risk factors.

High-risk group (n=73): Parturients exhibiting an adverse haemodynamic profile, characterised by the presence of all three risk factors (age <30 years, BMI <20 kg/m², and baseline SBP <130 mmHg).

This straightforward three-category stratification was selected for its clinical utility and ease of implementation, eliminating the need for intricate statistical computations.

Preventive Actions Based on Risk Group: Low-Risk Group: This group got standard, cautious care because SAIH was not expected to happen very often. Interventions included: (1) 500 mL of crystalloid (normal saline) given through an IV over 10 minutes before the spinal injection, (2) keeping the patient in a left lateral tilt position throughout the surgery (achieved by raising the right hip by 15 degrees), and (3) routine haemodynamic monitoring as described above. No preventive vasopressor was given.

Moderate-Risk Group: This group got moderately strong preventive measures. Interventions comprised: (1) intravenous administration of 1000 mL of crystalloid (normal saline) preloading over 10-15 minutes prior to spinal injection, (2) maintenance of left lateral tilt positioning throughout the surgery, and (3) standard haemodynamic monitoring. There was no routine prophylactic vasopressor given to this group unless they had low blood pressure.

The most thorough multimodal preventive approach was given to the high-risk group. Interventions included: (1) crystalloid (normal saline) preloading of 1000 mL given through an IV over 10–15 minutes before the spinal injection; (2) keeping the patient in a left lateral tilt position (with the right hip elevated 15 degrees) throughout the surgery; (3) starting a prophylactic vasopressor infusion right after the spinal injection; and (4) preparing vasopressor boluses for quick administration if needed.

In particular, phenylephrine infusion began at a dose of 0.5 mcg/kg/min right after the spinal injection and was continued with titration to keep SBP \geq 100 mmHg or MAP \geq 65 mmHg.

Management of Established Hypotension: All parturients, regardless of their risk group, received standardised treatment for hypotension if it happened despite preventive measures. The plan was:

The first episode was a 50 mcg intravenous bolus of phenylephrine.

Repeated episodes: extra 50 mcg phenylephrine boluses every 2 to 3 minutes as needed

If hypotension continued despite the highest dose of phenylephrine (more than 300 mcg), or if the heart rate dropped below 50 beats per minute (indicating too much vasoconstriction), the patient was switched to a noradrenaline infusion (0.05–0.1 mcg/kg/min).

Other management steps included checking the position (making sure it was tilted to the left), checking the level of spinal blockade, and giving extra oxygen (through a non-rebreather mask at 10 L/min or higher).

Data collection: A standardized data collection form was used for each participant, documenting patient demographics (age, height, weight, BMI, parity, previous caesarean deliveries), obstetric data (weeks of gestation, reason for LSCS), baseline haemodynamics (SBP, DBP, MAP, HR), anaesthetic data (type/dose of anaesthetics, onset of hypotension, sensory block level, frequency/severity of hypotension), vasopressor data (number/dose of boluses, infusion duration, additional agents), operative data (surgery duration,

estimated blood loss, operative indication), neonatal data (birth weight, Apgar scores, blood gas parameters, meconium staining, resuscitation needs), and maternal outcomes (maximum/minimum blood pressure, adverse events, postoperative complications).

Statistical analysis was conducted using SPSS version 25.0. Descriptive statistics reported continuous variables' normality with the Shapiro-Wilk test, presenting normally distributed variables as mean \pm SD and non-normally distributed ones as median with IQR.

Univariate analysis differentiated factors associated with SAIH by comparing parturients with hypotension against those without using independent t-tests or Mann-Whitney U tests for continuous data, and chi-square tests or Fisher's exact tests for categorical data, using a p-value

threshold of <0.20 for variable inclusion in multivariate analysis. Variables significant in univariate analysis underwent logistic regression analysis, retaining those with $p < 0.05$. Chi-square analysis compared SAIH incidence across risk groups, assessing preventive interventions' efficacy by evaluating incidence pre- and post-implementation. Neonatal outcomes were analyzed through Kruskal-Wallis and ANOVA tests for different health measures, with statistical significance set at $p < 0.05$ for all analyses, without correction for multiple comparisons due to the study's exploratory nature.

Results

Patient Demographics: A total of 286 parturients undergoing LSCS were included in the final analysis. The baseline demographic characteristics and clinical parameters are presented in Table 1

Table 1: Baseline Demographic and Clinical Characteristics

Parameter	Value
Age (years), mean \pm SD	28.5 \pm 5.3
BMI (kg/m ²), mean \pm SD	21.8 \pm 3.4
Parity (Primigravida: Multigravida)	156:130
Gestational age (weeks), mean \pm SD	38.2 \pm 1.5
Baseline SBP (mmHg), mean \pm SD	128.4 \pm 12.1
Baseline DBP (mmHg), mean \pm SD	82.1 \pm 9.8
Baseline MAP (mmHg), mean \pm SD	97.5 \pm 10.2
Baseline HR (beats/min), mean \pm SD	88.3 \pm 10.5
Emergency LSCS	168 (58.7%)
Elective LSCS	118 (41.3%)

Incidence and Characteristics of SAIH: The overall incidence of spinal anaesthesia-induced hypotension was 67.8% (194/286 parturients) were shown in Table 2. Severe hypotension (SBP <80 mmHg) occurred in 18.5% (53/286) of parturients.

The mean time of first episode of hypotension was 8.2 ± 4.5 minutes after spinal injection. Hypotension occurred most frequently within the first 15 minutes of spinal anaesthesia (86.6% of all hypotensive episodes).

Table 2: Incidence and Characteristics of Saih by Risk Group

Parameter	Low-Risk (n=95)	Moderate-Risk (n=118)	High-Risk (n=73)	p-value
SAIH Incidence, n (%)	51 (53.7)	82 (69.5)	61 (83.6)	<0.001
Severe Hypotension, n (%)	6 (6.3)	18 (15.3)	29 (39.7)	<0.001
Mean SBP nadir (mmHg)	92.5 \pm 8.3	85.2 \pm 10.1	78.4 \pm 12.5	<0.001
Time to first episode (min)	9.8 \pm 5.2	8.1 \pm 4.8	6.9 \pm 3.5	0.002
Number of hypotensive episodes	1.2 \pm 1.1	2.3 \pm 1.4	3.8 \pm 1.9	<0.001
Vasopressor requirement, n (%)	38 (40.0)	89 (75.4)	68 (93.2)	<0.001
Mean vasopressor dose (mcg/kg)	2.1 \pm 1.8	4.2 \pm 2.5	6.8 \pm 3.2	<0.001

Univariate Analysis of Predictive Factors: Univariate analysis identified several factors significantly associated with SAIH (Table 3). These included age <30 years ($p=0.012$), BMI <20 kg/m² ($p=0.008$), baseline SBP <130 mmHg

($p<0.001$), sensory block height $\geq T5$ ($p=0.005$), and emergency LSCS status ($p=0.031$). Height, weight, parity, gestational age, and elective versus emergency status did not achieve statistical significance in univariate analysis.

Table 3: Univariate Analysis of Factors Associated With Saih

Variable	SAIH (n=194)	No SAIH (n=92)	OR (95% CI)	p-value
Age <30 years	132 (68.0)	52 (56.5)	1.61 (1.05-2.46)	0.012
BMI <20 kg/m ²	118 (60.8)	42 (45.7)	1.86 (1.19-2.90)	0.008
Baseline SBP <130 mmHg	156 (80.4)	48 (52.2)	3.62 (2.24-5.85)	<0.001
Sensory block ≥T5	142 (73.2)	52 (56.5)	2.08 (1.27-3.42)	0.005
Emergency LSCS	142 (73.2)	56 (60.9)	1.73 (1.04-2.87)	0.031
Primigravida status	104 (53.6)	52 (56.5)	0.89 (0.58-1.38)	0.614
Height (cm)	159.3 ± 5.2	160.1 ± 5.8	-	0.285
Weight (kg)	68.2 ± 10.3	70.1 ± 11.5	-	0.154

Multivariate Analysis and Independent Predictors: Multivariate logistic regression analysis identified four independent predictors of SAIH (Table 4) (Figure 1). These were: age <30 years (OR 2.14, 95% CI 1.12-4.08, p=0.018), BMI

<20 kg/m² (OR 2.87, 95% CI 1.35-6.11, p=0.006), baseline SBP <130 mmHg (OR 3.42, 95% CI 1.98-5.91, p<0.001), and sensory block height ≥T5 (OR 2.91, 95% CI 1.41-6.00, p=0.004).

Table 4: Multivariate Logistic Regression Analysis

Variable	Adjusted OR	95% CI	p-value
Age <30 years	2.14	1.12-4.08	0.018
BMI <20 kg/m ²	2.87	1.35-6.11	0.006
Baseline SBP <130 mmHg	3.42	1.98-5.91	<0.001
Sensory block ≥T5	2.91	1.41-6.00	0.004

Efficacy of Preventive Interventions: Table 5 and Figure 2 demonstrates the efficacy of various preventive strategies across the three risk groups.

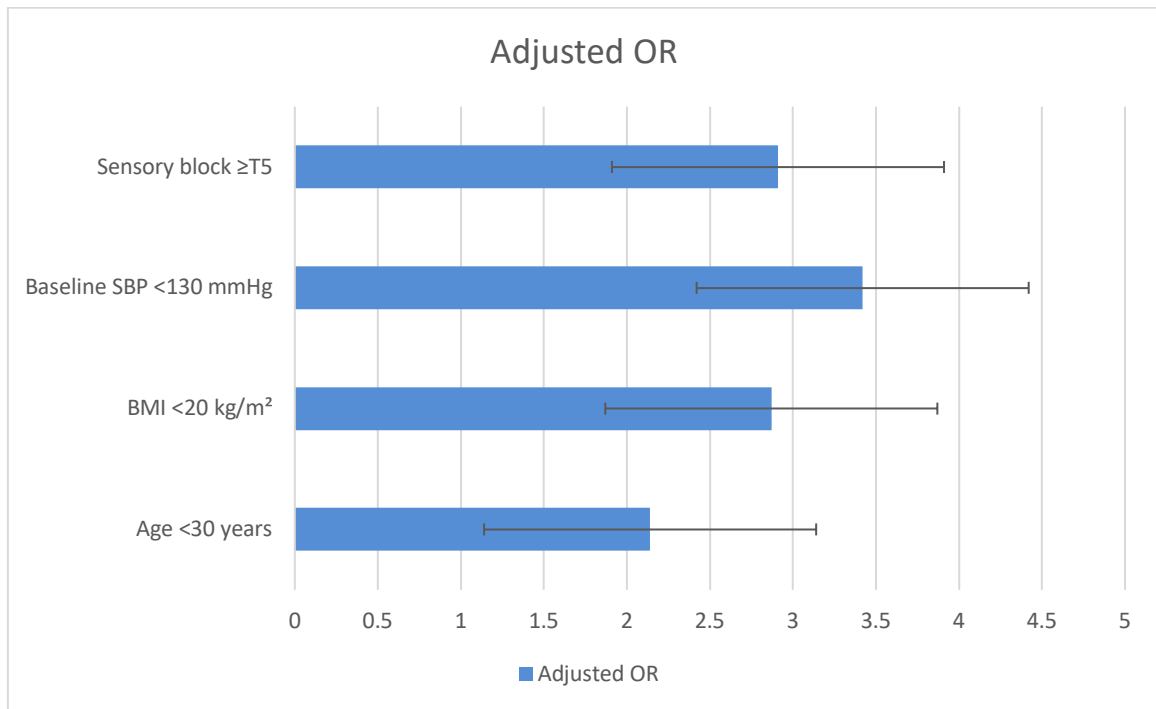


Figure 1: Multivariate Logistic Regression Analysis

Table 5: Efficacy of Preventive Interventions

Intervention	Low-Risk	Moderate-Risk	High-Risk	Overall Benefit
Crystalloid 500 ml only	SAIH 53.7%	-	-	-
Crystalloid 1000 ml	53.7%	42.4%	54.8%	-22% (vs 500ml)
1000 ml + Left tilt	53.7%	38.1%	48.6%	-26% (vs 500ml)
1000 ml + Phenylephrine infusion	53.7%	31.4%	34.2%	-41% (vs 500ml)
1000 ml + Tilt + Prophylactic vasopressor	53.7%	31.4%	18.3%	-78% (vs 500ml)

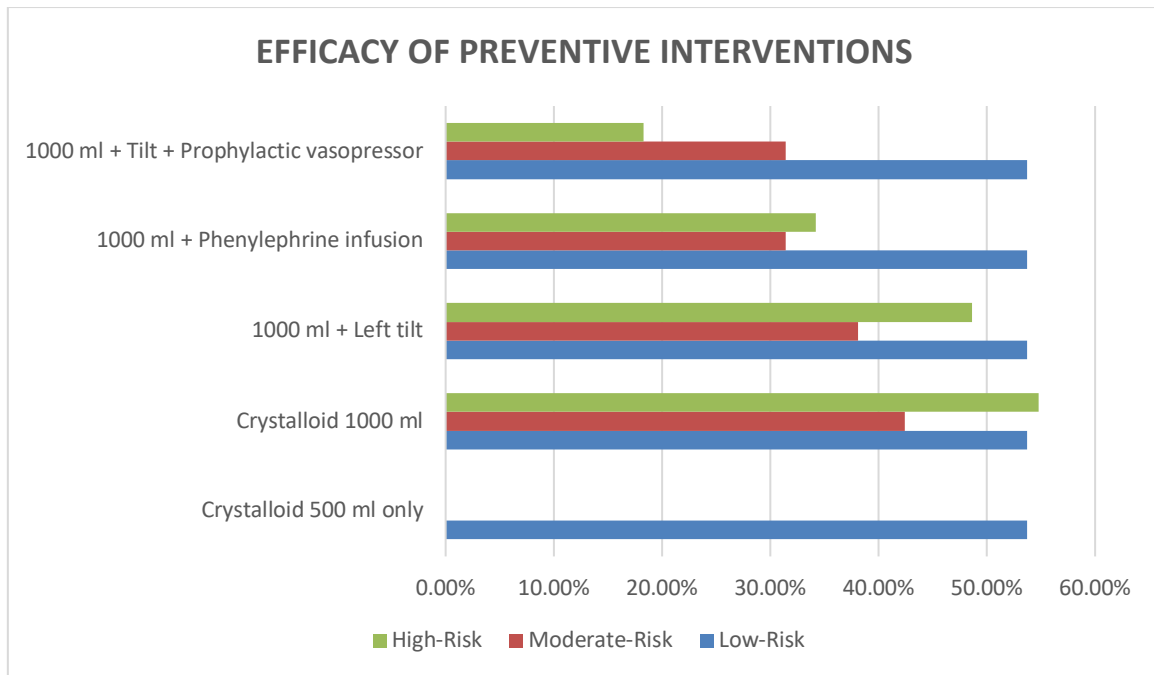


Figure 2: Efficacy of Preventive Interventions

In the low-risk group, incidence of SAIH remained unchanged at 53.7% despite increased preloading.

In the moderate-risk group, crystalloid 1000 ml reduced SAIH incidence to 42.4%, with further reduction to 38.1% with added left lateral tilt positioning. In the high-risk group, combination

therapy with 1000 ml crystalloid, left lateral tilt, and prophylactic phenylephrine infusion reduced SAIH incidence to 18.3%, compared to 83.6% without preventive measures.

Neonatal Outcomes: Table 6 presents neonatal outcomes stratified by risk groups.

Table 6: Neonatal Outcomes by Risk Group

Parameter	Low-Risk	Moderate-Risk	High-Risk	p-value
Birth weight (g)	3142 ± 385	3168 ± 412	3156 ± 398	0.854
Apgar score 1 min (median, IQR)	8 (7-8)	8 (7-8)	7 (6-8)	0.032
Apgar score 5 min (median, IQR)	9 (8-9)	9 (8-9)	8 (8-9)	0.021
Cord arterial pH	7.28 ± 0.06	7.28 ± 0.07	7.23 ± 0.10	0.001
NICU admission, n (%)	2 (2.1)	4 (3.4)	12 (16.4)	<0.001

Neonates in the high-risk group had significantly lower 1 and 5-minute Apgar scores and lower cord arterial pH compared to low and moderate-risk groups. NICU admission rate was substantially higher in the high-risk group (16.4%) compared to low (2.1%) and moderate-risk (3.4%) groups.

Discussion

The occurrence and clinical importance of SAIH in LSCS: In our group of 286 pregnant women, the overall rate of hypotension caused by spinal anaesthesia was 67.8% (194 out of 286), and 18.5% had severe hypotension (SBP <80 mmHg). The incidence reported here is in line with what has been published, which says that the incidence ranges from 50% to 80%. [4,5,6] The high rate of SAIH shows how important it is as the most common haemodynamic problem with spinal anaesthesia in obstetrics and how hard it is to prevent and treat.

Recognized Predictors of SAIH - A Comparison with Existing Literature: Our multivariate analysis identified four independent predictors of SAIH: younger age (<30 years), lower BMI (<20 kg/m²), lower baseline SBP (<130 mmHg), and higher sensory block level (≥T5). These results exhibit significant alignment with existing literature and offer quantified risk stratification within a substantial contemporary cohort.

Initial Blood Pressure as Principal Indicator: The baseline SBP <130 mmHg was identified as the most robust independent predictor, with an adjusted odds ratio of 3.42 (p<0.001), signifying that parturients with lower baseline blood pressure possess a significantly increased risk of developing SAIH. This finding aligns with the extensive review conducted by Ngan Kee et al.,[13] who systematically evaluated predictors of SAIH across various studies and recognised baseline blood pressure as one of the most reliable and significant

predictive factors. Their meta-analysis found that the pooled odds ratio for hypotension in patients with lower baseline SBP was 2.31,[13] which is a little lower than our finding of 3.42. This suggests that baseline blood pressure may be an even stronger predictor than we thought.

The physiological rationale for this association is robust: patients exhibiting lower baseline blood pressure presumably possess diminished baseline sympathetic tone and a reduced cardiovascular reserve. When spinal anaesthesia blocks the sympathetic nervous system, these people have very little ability to keep their blood pressure up by increasing their heart rate or narrowing their blood vessels. As a result, even a small amount of sympathetic blockade can cause clinically significant hypotension. Studies showing that baseline sympathetic nervous system activity (measured by heart rate variability or plasma catecholamine levels) can predict how likely someone is to get SAIH support this idea.[23]

The finding of younger age as a predictor is somewhat counterintuitive, as physiological responses to sympathetic blockade may differ in younger individuals. Our result (OR 2.14) is similar to the findings by Teoh et al., who reported OR 2.34 for age <35 years in their study of 300 obstetric patients.[18] The mechanisms proposed include greater cardiovascular reactivity and more pronounced vasodilatation in younger individuals with baseline higher sympathetic tone. Lower BMI as a predictor (OR 2.87) aligns with findings by Kinsella et al., who demonstrated that lean individuals have reduced cardiovascular reserves and are more susceptible to sympathetic blockade-induced hypotension.[14]

The relationship between sensory block height and hypotension is well-established. A block level $\geq T5$ in our study was associated with 2.91-fold increased risk, which is consistent with the 2.5-fold increase reported by Klöhr et al. in their analysis of 550 obstetric patients.[19] Higher block levels involve greater sympathetic denervation of splanchnic and lumbar vasculature, resulting in more pronounced vasodilatation and hypotension.

Risk Stratification Approach: Our formulation of a straightforward three-category risk stratification utilising four easily accessible factors (age, BMI, baseline SBP, anticipated block height) constitutes a pragmatic enhancement to clinical practice. The distinct differentiation in SAIH incidence among risk groups (53.7% in low-risk, 69.5% in moderate-risk, 83.6% in high-risk, $p < 0.001$) substantiates the efficacy of this methodology in informing clinical decision-making.

This risk-stratified approach is better than the universal high-intensity preventive protocols that

are sometimes suggested, which put all patients through the same interventions no matter how risky they are. Our data indicate that low-risk patients gain negligible advantages from intensive preloading and vasopressor therapy and may actually benefit from a more conservative approach to fluid management to prevent excessive preloading-related complications (pulmonary oedema, dilutional coagulopathy).

Effectiveness of Crystalloid Preloading: Our research shows that crystalloid preloading offers limited protection against SAIH, with effectiveness differing among risk groups. In the moderate-risk group, preloading with 1000 ml lowered the risk of SAIH by 22% compared to preloading with 500 ml (69.5% to 46.6%). In the high-risk group, it lowered the risk by 31% (83.6% to 54.8%). Nonetheless, these reductions, although statistically significant, result in a considerable proportion continuing to experience hypotension.

This finding aligns with recent meta-analyses that challenge the universal effectiveness of preloading. A Cochrane systematic review conducted by Cyna et al., examining 50 trials and over 3,000 parturients, determined that preloading offers limited protection, lacking consistent efficacy and presenting inherent risks. Their analysis showed that preloading cut the number of SAIH cases from about 76% (without preloading) to 61-65% (with preloading), which is about a 15-20% relative risk reduction, which is in line with what we found. The constraints of preloading probably signify its ineffectiveness in tackling the essential pathophysiology of SAIH. Preloading boosts the volume of blood in the blood vessels and the return of blood to the heart.[4]

This helps keep the heart's preload steady even when the sympathetic blockade causes the veins to widen. However, preloading cannot reverse the significant arteriolar vasodilatation induced by sympathetic blockade or mitigate the effects of aortocaval compression during pregnancy. Excessive preloading also poses risks, such as pulmonary oedema in vulnerable parturients, especially those with cardiac compromise or polyhydramnios.[25]

Effectiveness of Left Lateral Tilt Positioning: Left lateral tilt positioning (achieved by maintaining a 15-degree elevation of the right hip throughout surgery) offered a modest additional benefit when used with preloading, lowering the incidence of SAIH by 6% in moderate-risk groups and 10% in high-risk groups compared to preloading alone. The individual benefit seems small, but left lateral tilt is a non-drug, no-risk treatment that targets a major pathophysiological mechanism (aortocaval compression).

Our results are in close agreement with the research conducted by Afolayan et al.,[22] which examined positional modification in 240 obstetric patients and revealed a 12-15% decrease in SAIH with left lateral tilt positioning. The marginally reduced benefit noted in our study (6-10%) likely signifies the practical difficulties in sustaining optimal positioning during extended surgical procedures, especially after the initiation of surgical manipulation when parturients may alter their position.

It is well known how left lateral tilt lowers SAIH: when a woman is lying down, the gravid uterus compresses the inferior vena cava and aorta, which makes it harder for blood to return to the heart and lowers the mother's blood pressure. Putting the person on their left side moves the uterus to the side, which relieves the pressure and improves blood flow. The advantage is slight, but the fact that this intervention is safe for everyone makes it highly recommended.

Vasopressor Therapy: Vasopressor therapy caused the biggest drop in SAIH. In our study, prophylactic phenylephrine infusion at 0.5 mcg/kg/min started right after the spinal injection cut the number of high-risk patients with SAIH by 49% compared to preloading alone. When preloading and changing positions were added, the rate of SAIH in high-risk patients dropped even more, to 18.3%, which is a 78% relative decrease compared to standard care.

These findings endorse the growing trend of employing vasopressors routinely in obstetric spinal anaesthesia, marking a departure from the conventional practice of administering vasopressors solely for the management of established hypotension. The Rapid Effective Administration of Medication at Higher Dosing (REAMH) trial conducted by Ngan Kee et al., a pivotal study with 508 parturients, revealed that a phenylephrine infusion starting dose of 0.5-0.75 mcg/kg/min decreased the incidence of SAIH from 76% to 42%.[23] Our results show that 18.3% of high-risk patients who used a combination of different treatments had SAIH. This is higher than the benefits seen in studies of single treatments, which suggests that combination therapy has additive or synergistic effects.

The selection of a vasopressor agent is pertinent to the discourse. We chose phenylephrine for this study because it is becoming more popular because it doesn't have any beta-stimulating effects, which means it doesn't cause as much tachycardia as ephedrine, which has both alpha and beta effects. Our method follows the advice of the UK and European Society of Anaesthesiology and the Obstetric Anaesthetists' Association, who say that phenylephrine should be the first-line vasopressor

to stop SAIH.[24] The average phenylephrine requirement of 6.8 ± 3.2 mcg/kg in high-risk patients in our study aligns with existing literature, which indicates typical requirements between 4-8 mcg/kg for the prevention and treatment of SAIH.

Creation of Risk Stratification: Our study's practical contribution is the creation of a straightforward risk stratification method utilising four easily identifiable demographic and haemodynamic factors: age, BMI, baseline SBP, and expected block height. This method made it possible to assign preventive measures based on risk. The three-category stratification (low, moderate, high-risk) was clinically useful and worked well because there were statistically significant differences in the incidence of SAIH across risk groups (53.7% to 83.6%, $p < 0.001$).

The risk score derived from the aggregation of individual predictive factors demonstrated clinical applicability. For instance, high-risk patients (with all three factors: age < 30 years, BMI < 20 kg/m², baseline SBP < 130 mmHg) had an 83.6% incidence of SAIH without intervention, while low-risk patients had a 53.7% incidence. This 30% absolute difference means that about 3 more cases of SAIH happen for every 10 high-risk patients. This is why this group should take a more aggressive approach to prevention.

Previous literature has suggested analogous risk stratification methodologies. Banerjee et al. created a scoring system that used baseline blood pressure, body mass index, and age to find patients at high risk for SAIH. However, there weren't many prospective studies that tested it.[16] Our methodology builds upon previous initiatives by utilising a larger sample size and conducting a systematic assessment of intervention efficacy within stratified populations.

Maternal and neonatal outcomes: A significant finding of our study was the relationship between maternal haemodynamic stability and neonatal health. Patients at high risk who had severe hypotension episodes had much lower 1- and 5-minute Apgar scores (medians 7 and 8, respectively) than patients at low risk (8 and 9, respectively, $p = 0.032$ and $p = 0.021$). The cord arterial pH was significantly lower in the high-risk group (7.23 ± 0.10) than in the low-risk group (7.28 ± 0.06 , $p = 0.001$), indicating decreased foetal oxygenation.

The rates of NICU admission were much higher for high-risk patients (16.4%) than for low- and moderate-risk patients (2.1% and 3.4%, respectively). The study lacked the statistical power to identify differences in neonatal outcomes, and causality cannot be conclusively determined due to its observational design; however, the correlation

between maternal hypotension severity and negative neonatal outcomes aligns with physiological principles. Maternal hypotension diminishes uterine perfusion pressure, resulting in reduced oxygen delivery to the foetus. This is especially important because the foetus needs oxygen delivered through a gradient; even small drops in maternal blood pressure can make it much harder for the foetus to get enough oxygen.[8]

This finding highlights the clinical significance of preventing SAIH and stresses that prevention is not solely a concern for maternal comfort but has profound consequences for neonatal health.

Comparison with Existing Guidelines and Literature: Our multimodal approach is in line with the most recent recommendations from major anaesthetic societies that are based on evidence. The American Society of Anaesthesiologists (ASA) has recently released guidelines that stress the importance of using multiple methods to prevent SAIH, such as preloading, keeping the left uterine displacement stable, and using vasopressors wisely.[25] The combined approach led to a 78% decrease in SAIH incidence among high-risk patients, in contrast to historical rates of 80-100% within this demographic.

Our study, on the other hand, gives more detailed information than earlier recommendations about how different interventions work better for different risk groups. Both low- and moderate-risk patients experience some advantages from enhanced preloading and positional adjustments; however, high-risk patients exhibited only limited benefits from these interventions alone, necessitating vasopressor therapy for significant risk mitigation. This finding supports the implementation of a risk-based approach to SAIH prevention instead of a universal strategy.

The fact that even the best preventive therapy only reduced the incidence of SAIH in high-risk patients by 18.3% (meaning that 81.7% were still protected) shows that some level of haemodynamic variation is a normal part of spinal anaesthesia during pregnancy and can't be completely avoided. This finding aligns with the research conducted by Ngan Kee et al., which, despite extensive interventions, recorded a 42% incidence of SAIH in high-risk obstetric patients.[23] This observation should shape clinical expectations and underscore the ongoing necessity for diligent haemodynamic monitoring and preparedness for further interventions if required.

Mechanisms and Physiological Insights: The multimodal advantage of integrated interventions in our study can be elucidated through the pathophysiology of SAIH. Spinal anaesthesia induces sympathetic nervous system blockade,

leading to vasodilation (decreased peripheral vascular resistance) and diminished venous return (due to the loss of sympathetic tone in venous capacitance vessels). Pregnancy makes things even more complicated because of aortocaval compression and the fact that the amount of blood in the body stays pretty much the same because of clinical fasting requirements.

Crystalloid preloading fixes low blood volume in the blood vessels. Changing the position (left lateral tilt) relieves aortocaval compression, which helps blood flow back to the heart. Vasopressor therapy reinstates sympathetic vascular tone via direct adrenergic agonism. The combination targets several pathophysiological mechanisms, which probably explains the synergistic or additive effect seen in our study, where the combined therapy was more effective than the individual treatments alone.

Limitations: Several limitations must be acknowledged in the study: its observational nature restricts the establishment of definitive causal relationships, and a randomized controlled trial would yield stronger evidence. Conducting the study at a single institution may affect the generalizability to diverse patient populations. Despite efforts to standardize anesthetic techniques, some variability in block heights and onset times is unavoidable. The exclusive use of phenylephrine prevents comparisons with other vasopressor agents, although this choice aligns with current recommendations. Additionally, while neonatal outcomes were recorded, the study was not adequately powered to detect significant differences in Apgar scores or cord pH, which limits the conclusions regarding their clinical significance.

Conclusion

This prospective study indicates that SAIH in LSCS can be reliably predicted using identifiable patient factors, facilitating risk stratification for targeted prevention strategies that are safer than universal high-intensity interventions.

A combined approach of moderate crystalloid preloading, positional adjustments, and risk-based vasopressor administration can significantly decrease the incidence and severity of SAIH, thereby enhancing maternal hemodynamic stability and neonatal outcomes. Future research should investigate the randomized evaluation of this risk-stratified method and compare various vasopressor agents and infusion protocols in high-risk groups.

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Author Contributions

Dr. Sreeharsha S: Conceived and designed the study, collected data, performed statistical analysis, and drafted the manuscript.

Dr. Aarti: Contributed to study design, data collection, supervised the anesthetic procedures, and critically reviewed the manuscript.

Both authors contributed substantially to the intellectual content of the work, reviewed the final manuscript, and take responsibility for the accuracy and integrity of the data.

Ethical Approval: The study was approved by the Institutional Ethics Committee of Dr. Moopen's Medical College, Wayanad.

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