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Maternal Haemodynamic Changes and Neonatal Outcomes Following Spinal Anaesthesia for Caesarean Delivery: A Prospective Observational Study.

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Abstract

Background: Spinal anaesthesia is widely used for caesarean delivery, but sympathetic blockade can cause maternal hypotension and reduce uteroplacental perfusion. Careful maternal and neonatal assessment is therefore essential. ***Objectives:*** To describe maternal haemodynamic changes after spinal anaesthesia, determine the incidence and associated factors of hypotension, and compare neonatal outcomes according to maternal hypotension status. ***Methods:*** This prospective observational study included 100 pregnant women undergoing elective or emergency caesarean delivery under spinal anaesthesia at Prathima Institute of Medical Sciences, Karimnagar, Telangana, India, from September 2025 to March 2026. Blood pressure and heart rate were recorded at baseline, serial intraoperative intervals, delivery, and completion of surgery. Maternal hypotension was defined as systolic blood pressure below 90 mmHg or a reduction of at least 20% from baseline. Maternal adverse events, vasopressor use, Apgar scores, umbilical arterial pH, resuscitation, and neonatal intensive care admission were recorded. ***Results:*** Maternal hypotension occurred in 38 women (38.0%; 95% confidence interval: 29.1–47.8%). The lowest mean systolic pressure occurred at 5 minutes, decreasing from 122.8 ± 11.6 to 101.7 ± 14.6 mmHg. Hypotension was associated with higher maternal age, body mass index, baseline heart rate, and sensory block at T4 or above. Neonates exposed to maternal hypotension had lower mean 1-minute Apgar scores (7.8 ± 0.9 versus 8.3 ± 0.7), 5-minute Apgar scores (9.0 ± 0.7 versus 9.3 ± 0.5), and umbilical arterial pH (7.27 ± 0.05 versus 7.30 ± 0.04). No maternal or early neonatal deaths occurred. ***Conclusion:*** Spinal anaesthesia produced a transient decline in maternal blood pressure. Maternal hypotension was common and was associated with modest deterioration in immediate neonatal indicators, although overall maternal and neonatal outcomes remained favourable.

Keywords: Caesarean delivery; maternal hypotension; neonatal outcomes; spinal anaesthesia; umbilical arterial pH; vasopressors.

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INTRODUCTION

Caesarean delivery is one of the most frequently performed obstetric operations, and neuraxial anaesthesia is generally preferred because it allows the mother to remain conscious, limits fetal exposure to systemic anaesthetic agents, provides effective surgical anaesthesia, and avoids many airway-related risks associated with general anaesthesia. Spinal anaesthesia is particularly attractive because of its rapid onset, technical simplicity, dense sensory and motor block, and predictable operating conditions. Nevertheless, the abrupt sympathetic blockade that follows intrathecal local anaesthetic administration reduces systemic vascular resistance and venous return. The gravid uterus further limits preload through aortocaval compression, creating a setting in which maternal arterial pressure can decline rapidly after the block.1,3

Maternal hypotension remains the most frequent clinically important complication of spinal anaesthesia for caesarean delivery. Its reported incidence varies widely because studies use different blood-pressure thresholds, baseline measurements, monitoring intervals, prophylactic strategies, and obstetric populations. A literature review identified numerous definitions, with a systolic pressure below 80% of baseline among the most commonly applied criteria.2 International guidance recommends maintaining systolic pressure close to the accurately measured pre-spinal value and avoiding reductions below 80% of baseline.1 Even brief hypotensive episodes can produce nausea, vomiting, dizziness, dyspnoea, altered consciousness, and, in extreme situations, cardiovascular collapse. Early recognition is therefore central to safe obstetric anaesthesia.

Maternal haemodynamic stability also has direct fetal relevance because uteroplacental perfusion lacks effective autoregulation and depends substantially on maternal perfusion pressure. Marked or prolonged hypotension can reduce

intervillous blood flow, contribute to fetal hypoxaemia and acidaemia, and influence Apgar scores or the need for neonatal resuscitation. The clinical impact is shaped by the magnitude and duration of hypotension, the interval from spinal injection to delivery, pre-existing fetal compromise, and the speed of corrective treatment.8,9,11,12 Contemporary management combines left uterine displacement, intravenous fluid co-loading, close non-invasive blood-pressure monitoring, and prompt vasopressor administration. Phenylephrine is widely used, while norepinephrine has emerged as an alternative that better preserves maternal heart rate and cardiac output in selected settings.6,7,13,14

Although preventive strategies are well described, real-world haemodynamic patterns and their relationship with neonatal outcomes differ across institutions and patient populations. Locally generated prospective data are valuable for evaluating the burden of post-spinal hypotension, identifying readily observable risk factors, and strengthening perioperative monitoring protocols. The present study was therefore undertaken to characterise serial maternal blood-pressure and heart-rate changes following spinal anaesthesia for caesarean delivery, determine the incidence and clinical correlates of maternal hypotension, document associated maternal adverse events and treatment requirements, and compare immediate neonatal outcomes between women who developed hypotension and those who remained haemodynamically stable.

METHODOLOGY

Study design and setting: This prospective hospital-based observational study was conducted at Prathima Institute of Medical Sciences, Karimnagar, Telangana, India. Recruitment and data collection were performed from September 2025 to March 2026. Necessary Permissions were obtained before starting the study. Written informed consent was obtained from every participant. The study followed the Declaration of Helsinki and institutional obstetric-anaesthesia standards.

Participants: Pregnant women aged 18–40 years with a singleton term pregnancy, American Society of Anesthesiologists physical status I or II, and planned elective or emergency caesarean delivery under spinal anaesthesia were enrolled consecutively. Assuming an expected hypotension proportion of 50%, 95% confidence, and 10% absolute precision, the minimum calculated sample was 96; this was rounded to 100. Women with hypertensive disorders of pregnancy, significant cardiovascular disease, major fetal anomaly, active placenta previa bleeding, contraindication to spinal anaesthesia, failed spinal block, or incomplete records were excluded. Conversion to general anaesthesia before delivery also led to exclusion from final analysis.

Anaesthetic procedure and monitoring: Standard fasting and aspiration prophylaxis were followed. On arrival in the operating theatre, electrocardiography, pulse oximetry, and non-invasive blood-pressure monitoring were instituted. Baseline heart rate and blood pressure were recorded after the participant had rested in the supine position with left uterine displacement. Intravenous access was secured, and crystalloid co-loading was commenced. Under aseptic precautions, spinal anaesthesia was administered at the L3–L4 or L4–L5 interspace using a 25- or 26-gauge needle. Hyperbaric bupivacaine with an intrathecal opioid was administered according to institutional practice. The patient was immediately positioned supine with left uterine displacement, and the upper sensory level was assessed before surgery.

Outcome assessment: Systolic blood pressure, diastolic blood pressure, mean arterial pressure, and heart rate were documented at baseline; 2, 5, 10, and 15 minutes after spinal injection; at delivery; and at the end of surgery. Hypotension was defined as systolic blood pressure below 90 mmHg or a reduction of at least 20% from baseline, consistent with commonly applied obstetric definitions.1,2 Hypotension was treated with rapid crystalloid administration, optimisation of uterine displacement, and intravenous ephedrine or phenylephrine at the attending anaesthesiologist's discretion. Bradycardia was defined as heart rate below 60 beats/minute. Nausea, vomiting, shivering, dizziness, high spinal block, respiratory depression, and conversion to general anaesthesia were recorded. Neonatal outcomes included birth weight, Apgar scores at 1 and 5 minutes, umbilical arterial pH, resuscitation, neonatal intensive care unit admission, and early neonatal mortality.

Statistical analysis: Data were analysed using a standard statistical software package. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, according to distribution, and categorical variables as frequency and percentage. Repeated haemodynamic measurements were evaluated using repeated-measures analysis. Women with and without hypotension were compared using the independent-samples t-test or Mann–Whitney U test for continuous variables and the chi-square or Fisher exact test for categorical variables. Two-sided p-values below 0.05 were considered statistically significant. The incidence of hypotension was reported with a 95% confidence interval.

RESULTS

During the study period, 106 pregnant women scheduled for caesarean delivery under spinal anaesthesia were assessed for eligibility. Six women were excluded: three did not meet the eligibility criteria, two declined participation, and one required conversion to general anaesthesia before delivery. The remaining 100 participants were included in the final analysis. Complete maternal haemodynamic and neonatal outcome data were available for all participants.

The mean maternal age was 27.6 ± 4.2 years, and the mean gestational age at delivery was 38.5 ± 1.1 weeks. Forty-two women were primigravidae and 58 were multigravidae. Elective caesarean delivery accounted for 64.0% of procedures. Previous caesarean delivery was the most frequent indication, followed by fetal distress and cephalopelvic disproportion. The complete maternal, obstetric, and perioperative profile is presented in Table 1.

Table 1. Maternal, obstetric, and perioperative characteristics of the participants

Characteristic	Value
Maternal age, years	27.6 ± 4.2
Body mass index, kg/m²	27.2 ± 3.8
Gestational age, weeks	38.5 ± 1.1
Primigravida	42 (42.0%)
Multigravida	58 (58.0%)
Elective caesarean delivery	64 (64.0%)
Emergency caesarean delivery	36 (36.0%)
Previous caesarean delivery	38 (38.0%)
Fetal distress	20 (20.0%)
Cephalopelvic disproportion	15 (15.0%)
Malpresentation	12 (12.0%)
Failed induction of labour	9 (9.0%)
Other indications	6 (6.0%)
Spinal anaesthesia-to-delivery interval, minutes	12.8 ± 3.6
Duration of surgery, minutes	48.7 ± 11.5
Intravenous fluid administered, mL	1,286 ± 312

Values are presented as mean ± standard deviation or number (percentage).

A significant early reduction in maternal arterial pressure followed spinal anaesthesia. Mean systolic blood pressure decreased from 122.8 ± 11.6 mmHg at baseline to 101.7 ± 14.6 mmHg at 5 minutes, while mean arterial pressure declined from 92.2 ± 8.9 to 73.8 ± 10.7 mmHg. Maternal heart rate increased transiently, reaching 94.3 ± 13.6 beats/minute at 5 minutes, and subsequently returned towards baseline. Serial changes in systolic pressure, diastolic pressure, mean arterial pressure, and heart rate were statistically significant (Table 2).

Table 2. Maternal haemodynamic changes following spinal anaesthesia

Time point	Systolic BP, mmHg	Diastolic BP, mmHg	Mean arterial pressure, mmHg	Heart rate, beats/min
Baseline	122.8 ± 11.6	76.9 ± 8.7	92.2 ± 8.9	88.4 ± 11.2
2 minutes	110.2 ± 13.1	66.8 ± 9.6	81.3 ± 10.2	91.7 ± 12.5
5 minutes	101.7 ± 14.6	59.8 ± 10.1	73.8 ± 10.7	94.3 ± 13.6
10 minutes	106.9 ± 13.8	63.4 ± 9.8	77.9 ± 10.2	92.1 ± 12.8
15 minutes	112.4 ± 12.7	67.5 ± 9.1	82.5 ± 9.7	89.8 ± 11.9
At delivery	115.8 ± 12.0	69.9 ± 8.9	85.2 ± 9.2	88.1 ± 11.4
End of surgery	119.1 ± 11.4	73.2 ± 8.5	88.5 ± 8.8	86.9 ± 10.7
Overall p-value	<0.001	<0.001	<0.001	<0.001

Values are presented as mean ± standard deviation. Overall p-values represent within-participant changes across the observation period. BP: blood pressure.

Maternal hypotension occurred in 38 participants, corresponding to an incidence of 38.0% (95% confidence interval: 29.1–47.8%). The median time to the first episode was 5 minutes after spinal injection. Twenty-six women had a single episode and 12 experienced recurrent hypotension. Vasopressor treatment was required in 32 women; ephedrine was administered to 24 and phenylephrine to eight. Women with hypotension were older, had a higher body mass index and baseline heart rate, and more frequently had a sensory block at T4 or above. Nausea or vomiting and dizziness were also more common in the hypotension group (Table 3).

No participant developed high spinal block, respiratory depression, severe arrhythmia, cardiac arrest, or loss of consciousness. There were no maternal deaths, and no participant required conversion to general anaesthesia after inclusion in the analysis.

Table 3. Maternal adverse events and factors associated with hypotension

Variable	Hypotension (n=38)	No hypotension (n=62)	p-value
Maternal age, years	29.3 ± 4.5	26.6 ± 3.7	0.002
Body mass index, kg/m²	28.6 ± 3.9	26.3 ± 3.5	0.003
Baseline systolic blood pressure, mmHg	121.9 ± 12.1	123.3 ± 11.3	0.557
Baseline heart rate, beats/minute	91.8 ± 11.4	86.3 ± 10.6	0.017
Sensory block at T4 or above	24 (63.2%)	20 (32.3%)	0.003
Emergency caesarean delivery	18 (47.4%)	18 (29.0%)	0.063
Recurrent hypotension	12 (31.6%)	0	Not applicable
Vasopressor requirement	32 (84.2%)	0	Not applicable
Bradycardia	8 (21.1%)	0	Not applicable
Nausea or vomiting	12 (31.6%)	2 (3.2%)	<0.001
Shivering	9 (23.7%)	9 (14.5%)	0.245
Dizziness	8 (21.1%)	1 (1.6%)	0.001

Values are presented as mean ± standard deviation or number (percentage). A dash indicates that a comparative p-value was not applicable.

The mean neonatal birth weight was 3.02 ± 0.42 kg. Mean Apgar scores were 8.1 ± 0.8 at 1 minute and 9.2 ± 0.6 at 5 minutes. Six neonates had an Apgar score below 7 at 1 minute, and one remained below 7 at 5 minutes. The mean umbilical arterial pH was 7.29 ± 0.05, while seven neonates required neonatal intensive care admission.

Neonates born to women who developed hypotension had significantly lower mean Apgar scores at 1 and 5 minutes and a lower mean umbilical arterial pH than neonates born to women without hypotension. An Apgar score below 7 at 1 minute was more frequent in the hypotension group. Differences in birth weight, umbilical arterial pH below 7.20, neonatal resuscitation, and neonatal intensive care admission were not statistically significant (Table 4). No early neonatal deaths occurred.

Table 4. Neonatal outcomes according to maternal hypotension status

Neonatal outcome	Overall (n=100)	Maternal hypotension (n=38)	No maternal hypotension (n=62)	p-value
Birth weight, kg	3.02 ± 0.42	2.97 ± 0.44	3.05 ± 0.41	0.357
Apgar score at 1 minute	8.1 ± 0.8	7.8 ± 0.9	8.3 ± 0.7	0.004
Apgar score at 5 minutes	9.2 ± 0.6	9.0 ± 0.7	9.3 ± 0.5	0.018
Apgar score <7 at 1 minute	6 (6.0%)	5 (13.2%)	1 (1.6%)	0.027
Apgar score <7 at 5 minutes	1 (1.0%)	1 (2.6%)	0	0.380
Umbilical arterial pH	7.29 ± 0.05	7.27 ± 0.05	7.30 ± 0.04	0.003
Umbilical arterial pH <7.20	4 (4.0%)	3 (7.9%)	1 (1.6%)	0.148
Neonatal resuscitation required	8 (8.0%)	5 (13.2%)	3 (4.8%)	0.151
Neonatal intensive care unit admission	7 (7.0%)	5 (13.2%)	2 (3.2%)	0.071
Early neonatal mortality	0	0	0	Not applicable

Values are presented as mean ± standard deviation or number (percentage). A dash indicates that a comparative p-value was not applicable.

DISCUSSION

This prospective study demonstrated a characteristic early decline in maternal blood pressure after spinal anaesthesia for caesarean delivery. Hypotension occurred in 38.0% of participants, and the lowest average systolic and mean arterial pressures were recorded 5 minutes after spinal injection. The timing is physiologically plausible because sympathetic blockade develops rapidly, producing arterial and venous vasodilatation before compensatory mechanisms and corrective treatment fully restore vascular tone. The observed incidence was lower than the 70–80% reported in some studies without effective prophylaxis, but it remained clinically substantial.3,6 Differences between studies reflect variable definitions, patient selection, intrathecal doses, fluid strategies, monitoring frequency, and vasopressor practice.2

Women who developed hypotension were older and had higher body mass index and baseline heart rate. A sensory block at T4 or above was also strongly associated with hypotension. These observations agree with Fakherpour et al., who

identified maternal age, elevated body mass index, baseline heart rate, and higher sensory block as relevant predictors.4 The prospective Siriraj study likewise reported increased risk with advanced maternal age, obesity, and a high sensory level.5 Higher block height intensifies sympathetic denervation, while increased body mass and pregnancy-related abdominal pressure can alter cerebrospinal fluid volume and intrathecal drug spread. A faster baseline heart rate can represent reduced cardiovascular reserve or greater sympathetic activation before the block.

Nausea or vomiting and dizziness were concentrated among women with hypotension, supporting the close relationship between maternal symptoms and reduced cerebral or gastrointestinal perfusion. Most hypotensive events responded to fluid administration and vasopressors, and no severe maternal complication occurred. Current evidence favours proactive haemodynamic management rather than delayed rescue therapy. Systematic reviews show that vasopressors reduce hypotension more reliably than crystalloid administration alone.6,7 Phenylephrine provides effective blood-pressure control and is associated with more favourable fetal acid-base status than ephedrine in several low-risk elective populations.8-10 Norepinephrine also preserves neonatal umbilical pH and produces less maternal bradycardia than phenylephrine in contemporary trials.13,14

Maternal hypotension was associated with modestly lower 1- and 5-minute Apgar scores and lower umbilical arterial pH. These findings support the biological link between maternal perfusion pressure and uteroplacental blood flow. However, severe neonatal compromise was uncommon, and differences in resuscitation and neonatal intensive care admission were not statistically significant. Prompt treatment and the relatively short spinal-to-delivery interval probably limited fetal exposure to reduced perfusion. Previous studies have shown that longer neuraxial-to-delivery intervals and greater blood-pressure reductions correlate with lower umbilical arterial pH.11,12 Overall, the findings reinforce the importance of accurate baseline measurements, frequent blood-pressure assessment during the first 10 minutes, immediate correction of hypotension, and coordinated delivery without avoidable delay.

Limitations

This study was conducted at a single tertiary-care institution with a moderate sample size, which limits wider generalisation. Anaesthetic drug doses and vasopressor selection followed routine clinical judgement rather than a fixed interventional protocol. Cardiac output and uteroplacental blood flow were not measured. Neonatal assessment was restricted to immediate outcomes, without extended neurological or developmental follow-up. Residual confounding from emergency indications and operative timing remains possible.

CONCLUSION

Spinal anaesthesia for caesarean delivery produced a rapid reversible decline in maternal arterial pressure, with the greatest haemodynamic change occurring within 5 minutes of intrathecal injection. Maternal hypotension affected 38.0% of participants and was associated with higher maternal age, body mass index, baseline heart rate, and a sensory block at T4 or above. Hypotension was accompanied by more nausea, vomiting, and dizziness and by modest reductions in early Apgar scores and umbilical arterial pH. Nevertheless, serious maternal and neonatal complications were absent overall. Early monitoring, left uterine displacement, appropriate fluid co-loading, prompt vasopressor treatment, and avoidance of unnecessary spinal-to-delivery delay remain essential for preserving maternal stability and neonatal wellbeing.

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