Original Article

Assessment of Phenylephrine in Preventing Hemodynamic Responses of Oxytocin During Caesarean Section under Spinal Anaesthesia

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Abstract

Background: Spinal anaesthesia is the preferred technique for caesarean section (CS) but frequently induces hypotension. Oxytocin, administered for uterine contraction, can exacerbate hemodynamic instability, causing significant hypotension and bradycardia. Phenylephrine, a selective α1-agonist, is commonly used to counteract spinal-induced hypotension, but its efficacy specifically in mitigating oxytocin-induced hemodynamic changes remains less defined. **Material and Methods:** A prospective, randomized, double-blind, controlled trial was conducted. 120 parturients undergoing elective CS under spinal anaesthesia were randomly allocated to receive either a prophylactic phenylephrine infusion (0.5 µg/kg/min, Group P, n=60) or normal saline infusion (Group C, n=60) initiated immediately after spinal block. Standardized spinal anaesthesia (hyperbaric bupivacaine 12.5 mg + fentanyl 15 µg) and oxytocin protocol (5 IU bolus over 1 minute after delivery) were used. Hemodynamic parameters (systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR)) were recorded at baseline, after spinal block, and at 1, 3, 5, 10, 15, and 30 minutes after oxytocin bolus. Incidence of hypotension (SBP <80% baseline or <90 mmHg), bradycardia (HR <50 bpm), and vasopressor requirements (rescue ephedrine) were recorded. **Results:** The incidence of hypotension after oxytocin was significantly lower in Group P (15.0%) compared to Group C (55.0%) ($p<0.001$). Mean MAP at 1, 3, and 5 minutes' post-oxytocin was significantly higher in Group P (82.4 ± 6.8 mmHg, 80.1 ± 7.2 mmHg, 78.9 ± 6.5 mmHg) vs. Group C (68.5 ± 8.1 mmHg, 65.3 ± 7.9 mmHg, 67.2 ± 7.4 mmHg) ($p<0.001$ for all). The incidence of bradycardia was lower in Group P (8.3%) vs. Group C (18.3%), but this did not reach statistical significance ($p=0.097$). Rescue ephedrine requirement was significantly lower in Group P (10.0%) vs. Group C (48.3%) ($p<0.001$). Nausea/vomiting incidence was significantly lower in Group P (11.7%) vs. Group C (35.0%) ($p=0.002$). **Conclusion:** Prophylactic phenylephrine infusion (0.5 µg/kg/min) effectively attenuates the hypotensive response to oxytocin bolus administration during CS under spinal anaesthesia, reducing the incidence of hypotension, vasopressor requirements, and associated nausea/vomiting without significantly increasing bradycardia risk.

Keywords: Phenylephrine; Oxytocin; Haemodynamic stability; Caesarean section; Spinal anaesthesia; Hypotension.

Received: 10 June 2025 **Revised: 04 July 2025** **Accepted: 28 August 2025** **Published: 18 September 2025**

Introduction

Spinal anaesthesia is widely regarded as the anaesthetic technique of choice for elective caesarean section (CS) due to its rapid onset, dense sensory and motor blockade, and avoidance of general anaesthesia risks.[1] However, a significant and well-documented complication is spinal-induced hypotension, occurring in up to 80% of parturients if untreated.[2] This hypotension results primarily from sympathetic blockade-induced vasodilation and decreased systemic vascular resistance (SVR), compounded by aortocaval compression in the supine position.[3] Untreated maternal hypotension can lead to adverse maternal outcomes such as nausea, vomiting, and unconsciousness, and more critically, compromise uteroplacental perfusion, potentially resulting in fetal acidosis and neonatal depression.[4]

Oxytocin is the first-line uterotonic agent administered prophylactically after delivery of the infant during CS to prevent postpartum haemorrhage (PPH).[5] While effective, the rapid intravenous bolus administration of oxytocin, particularly at higher doses (e.g., 5-10 IU), is associated with significant hemodynamic disturbances. These include transient but profound hypotension, tachycardia followed by reflex bradycardia, and reductions in cardiac output and systemic vascular resistance.[6,7] The mechanisms involve direct vasodilatory effects on vascular smooth muscle, potentially mediated by nitric oxide release, and a negative inotropic effect on the heart.[8] The combination of pre-existing sympathetic blockade from spinal anaesthesia and the vasodilatory effects of oxytocin creates a potent scenario for severe hemodynamic instability, increasing the risk of maternal morbidity and

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DOI:
10.21276/amit.2025.v12.i3.57

How to cite this article: Jadhav A, Shingala S, Modiya M. Assessment of Phenylephrine in Preventing Hemodynamic Responses of Oxytocin During Caesarean Section under Spinal Anaesthesia. Acta Med Int. 2025;12(3):173-178.

potentially affecting neonatal well-being.[9]

Prophylactic administration of vasopressors is a cornerstone strategy to maintain hemodynamic stability during CS under spinal anaesthesia.[10] Ephedrine, a mixed α- and β-agonist, was traditionally the first-line agent. However, phenylephrine, a selective α1-adrenergic receptor agonist, has gained prominence as the preferred vasopressor due to its efficacy in increasing SVR without causing significant tachycardia, and evidence suggesting better preservation of umbilical artery pH compared to ephedrine.[11,12] Numerous studies have established the efficacy of phenylephrine in preventing and treating spinal-induced hypotension.[13,14] However, the specific challenge lies in the additive hemodynamic insult from oxytocin administration superimposed on the spinal block.

Recent studies have explored various strategies to mitigate oxytocin-induced hemodynamic changes, including slow infusion instead of bolus administration and lower oxytocin doses.[15,16] While slow infusion reduces hemodynamic perturbations, it may not provide the rapid uterine contraction needed for optimal haemostasis in all cases. Lower doses (e.g., 3 IU) also show reduced hemodynamic effects but may be less effective in preventing PPH compared to 5 IU in some contexts.[17] The role of prophylactic vasopressors specifically targeted at counteracting the oxytocin bolus effect, particularly when administered concurrently with spinal anaesthesia, requires further investigation. While phenylephrine is used prophylactically for spinal hypotension, its efficacy in specifically blunting the hemodynamic response triggered by the oxytocin bolus itself has not been conclusively quantified in a well-controlled trial design.

This study addresses a critical research gap: the lack of robust evidence on the effectiveness of prophylactic phenylephrine infusion in preventing the acute hemodynamic disturbances specifically attributable to the standard oxytocin bolus administered during CS under spinal anaesthesia. Understanding this interaction is vital for optimizing maternal safety and hemodynamic management protocols. Therefore, the aim of this study was to assess the efficacy of a prophylactic phenylephrine infusion in preventing hypotension, bradycardia, and associated adverse events following oxytocin bolus administration in parturients undergoing elective caesarean section under spinal anaesthesia.

Materials and Methods

Study Design: A prospective, randomized, double-blind, placebo-controlled trial was conducted at a tertiary care teaching hospital between January 2022 and December 2023.

Sample Size Calculation: Based on a pilot study and previous literature, the incidence of hypotension (SBP <80% baseline) within 10 minutes of oxytocin bolus in the control group was estimated at 60%. We hypothesized that prophylactic phenylephrine would reduce this incidence to 30%. Using a two-sided alpha error of 0.05 and a power of 90%, and accounting for a 10% dropout rate, a minimum sample size of 54 participants per group was required. We enrolled 60 participants per group to ensure adequate power.

Participants: Parturients aged 18-45 years, with American Society of Anesthesiologists (ASA) physical status I or II, scheduled for elective caesarean delivery at term gestation (≥37 weeks) under spinal anaesthesia, were eligible for inclusion.

Exclusion Criteria

Parturients were excluded if they had: pre-existing hypertension (pregnancy-induced or chronic), cardiovascular disease (ischemic heart disease, heart failure, significant arrhythmias), cerebrovascular disease, known hypersensitivity to phenylephrine, oxytocin, or local anaesthetics, contraindications to spinal anaesthesia (coagulopathy, infection at puncture site), significant fetal anomalies, multiple gestation, preterm labour, or BMI >40 kg/m².

Randomization and Blinding: Participants were randomly assigned to either the Phenylephrine group (Group P) or the Control group (Group C) using a computer-generated randomization sequence in a 1:1 ratio. The allocation sequence was concealed in sequentially numbered, opaque, sealed envelopes. An anaesthesia nurse not involved in patient care or data collection prepared the study infusions. Group P received phenylephrine 0.5 µg/kg/min diluted in normal saline to a total volume of 50 ml. Group C received an equivalent volume of normal saline (placebo). Both infusions were prepared in identical syringes labelled only with the participant's unique study number. The participant, the attending anaesthesiologist performing the spinal block and managing the case, the obstetrician, and the data analyst were all blinded to group allocation.

Anaesthetic Procedure: All participants underwent standard preoperative fasting. Upon arrival in the operating room, standard monitoring was applied: electrocardiography (ECG), non-invasive blood pressure (NIBP) measured every 3 minutes, and pulse oximetry (SpO_2). A 16-gauge intravenous cannula was inserted in the forearm, and a preload of 10 ml/kg of warmed Ringer's lactate solution was administered over 15 minutes. Baseline hemodynamic parameters (SBP, DBP, MAP, HR) were recorded as the average of two consecutive readings taken 2 minutes apart in the supine position with left uterine displacement.

Spinal anaesthesia was performed in the sitting or lateral position at the L3-L4 or L4-L5 interspace using a 25-gauge Quincke needle. After confirming free flow of cerebrospinal fluid, participants received intrathecal hyperbaric bupivacaine 12.5 mg (2.5 ml of 0.5%) with fentanyl 15 µg. Immediately after the intrathecal injection, the participant was positioned supine with left uterine displacement, and the study infusion (phenylephrine or placebo) was commenced via a syringe pump at a rate calculated to deliver 0.5 µg/kg/min (Group P) or an equivalent volume of saline (Group C). The infusion continued throughout the procedure and for 30 minutes after oxytocin administration, unless terminated earlier due to adverse effects or completion of surgery.

Sensory block level (loss of cold sensation to alcohol swab) was assessed at 5-minute intervals until a T4-T6 level was achieved. Surgery commenced upon achieving an adequate sensory block. Following delivery of the infant and clamping of the umbilical cord, all participants received a standardized intravenous bolus of oxytocin 5 IU over 1 minute, administered by the

anaesthesiologist.

Hemodynamic Management and Data Collection: Hemodynamic parameters (SBP, DBP, MAP, HR) were recorded at the following time points: Baseline (BL), immediately after spinal block (T0), and at 1, 3, 5, 10, 15, and 30 minutes after the start of the oxytocin bolus (T1, T3, T5, T10, T15, T30). The primary outcome was the incidence of hypotension, defined as SBP <80% of the baseline value or an absolute SBP <90 mmHg, occurring within 30 minutes after oxytocin administration. Secondary outcomes included: 1) Incidence of bradycardia (HR <50 bpm), 2) Magnitude of change in MAP and HR from baseline at each time point, 3) Requirement for rescue vasopressor (ephedrine 5 mg boluses, repeated every 3 minutes as needed to treat hypotension), 4) Incidence of nausea and vomiting (assessed by direct questioning and observation), 5) Total dose of rescue ephedrine administered, 6) Neonatal outcomes (Apgar scores at 1 and 5 minutes, umbilical artery pH).

Statistical Analysis: Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Normality of distribution was assessed using the Shapiro-Wilk test. Continuous variables were expressed as mean ± standard deviation (SD) or median [interquartile range (IQR)] as appropriate. Categorical variables were expressed as frequencies and percentages. Baseline characteristics were compared between groups using the independent samples t-test (for normally distributed continuous data) or Mann-Whitney U test (for non-normally distributed data). Chi-square test or Fisher's exact test was used for categorical variables. Hemodynamic parameters (MAP, HR) over time were analyzed using two-way repeated measures ANOVA with group and time as factors, followed by Bonferroni post-hoc tests for pairwise comparisons at specific time points. The incidence of hypotension, bradycardia, nausea/vomiting, and rescue ephedrine use were compared using Chi-square or Fisher's exact test. A p-value <0.05 was considered statistically significant.

Results

A total of 134 parturients were assessed for eligibility. Fourteen were excluded (8 had BMI >40, 4 had chronic hypertension, 2 declined consent). Thus, 120 participants were randomized: 60 to Group P (Phenylephrine) and 60 to Group C (Control). All participants completed the study protocol as per intention-to-treat analysis. Demographic and baseline characteristics were comparable between the two groups [Table 1]. There were no significant differences in age, weight, height, BMI, gestational age, baseline SBP, DBP, MAP, or HR (p>0.05 for all). The sensory block level achieved and the time from spinal block to oxytocin administration were also similar between groups.

Table 1: Demographic and Baseline Characteristics

Variable	Group P (n=60)	Group C (n=60)	p-value
Age (years)	29.4 ± 4.2	30.1 ± 4.5	0.382
Weight (kg)	72.5 ± 8.3	74.1 ± 9.1	0.312
Height (cm)	162.3 ± 5.8	161.8 ± 6.1	0.624
BMI (kg/m²)	27.5 ± 2.9	28.2 ± 3.2	0.189
Gestational Age (weeks)	38.9 ± 1.1	39.1 ± 1.0	0.291
Baseline SBP (mmHg)	118.5 ± 10.2	120.2 ± 11.5	0.401
Baseline DBP (mmHg)	72.3 ± 8.1	73.8 ± 8.7	0.367
Baseline MAP (mmHg)	87.7 ± 8.5	89.3 ± 9.2	0.345
Baseline HR (bpm)	78.4 ± 9.6	76.9 ± 10.2	0.423
Sensory Block Level (T)	T4 [T4-T5]	T4 [T4-T5]	0.851*
Time Spinal to Oxytocin (min)	12.3 ± 2.8	11.9 ± 3.1	0.512

Data presented as mean ± SD or median [IQR]. *Mann-Whitney U test. BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: Mean Arterial Pressure; HR: Heart Rate.

Hemodynamic Outcomes: The incidence of hypotension within 30 minutes after oxytocin administration was significantly lower in Group P (9/60, 15.0%) compared to Group C (33/60, 55.0%) (p<0.001). The mean MAP values were significantly higher in Group P compared to Group C at all-time points after oxytocin administration (T1, T3, T5, T10, T15, T30) (p<0.001 for all comparisons) (Figure 1). The most significant differences were observed at T1, T3, and T5 minutes (Table 2). The magnitude of MAP decrease from baseline was significantly smaller in Group P at all post-oxytocin time points (p<0.001).

The incidence of bradycardia (HR <50 bpm) was lower in Group P (5/60, 8.3%) compared to Group C (11/60, 18.3%), but this difference did not reach statistical significance (p=0.097). Mean HR was significantly lower in Group P compared to Group C at T1, T3, T5, and T10 minutes (p<0.05), but the differences were small in absolute terms (Table 2). The requirement for rescue ephedrine was significantly lower in Group P (6/60, 10.0%) compared to Group C (29/60, 48.3%) (p<0.001). The median total dose of rescue ephedrine administered was also significantly lower in Group P (0 mg [0-0 mg]) compared to Group C (10 mg [0-20 mg]) (p<0.001).

Table 2: Hemodynamic Parameters After Oxytocin Bolus

Time Point	Variable	Group P (n=60)	Group C (n=60)	p-value
T1 (1 min)	MAP (mmHg)	82.4 ± 6.8	68.5 ± 8.1	<0.001
	HR (bpm)	72.1 ± 8.9	75.8 ± 9.2	0.018
T3 (3 min)	MAP (mmHg)	80.1 ± 7.2	65.3 ± 7.9	<0.001
	HR (bpm)	70.5 ± 8.3	74.2 ± 8.7	0.012

T5 (5 min)	MAP (mmHg)	78.9 ± 6.5	67.2 ± 7.4	<0.001
	HR (bpm)	69.8 ± 7.9	73.5 ± 8.4	0.015
T10 (10 min)	MAP (mmHg)	79.5 ± 6.1	71.8 ± 6.8	<0.001
	HR (bpm)	71.2 ± 7.6	74.1 ± 8.1	0.032
T15 (15 min)	MAP (mmHg)	80.8 ± 5.9	75.1 ± 6.5	<0.001
	HR (bpm)	72.5 ± 7.4	74.8 ± 7.9	0.091
T30 (30 min)	MAP (mmHg)	83.1 ± 5.7	79.2 ± 6.2	<0.001
	HR (bpm)	73.8 ± 7.1	75.2 ± 7.6	0.287

Data presented as mean ± SD. MAP: Mean Arterial Pressure; HR: Heart Rate. p-values for between-group comparisons at each time point (Independent t-test).

Adverse Events and Neonatal Outcomes: The incidence of nausea and vomiting was significantly lower in Group P (7/60, 11.7%) compared to Group C (21/60, 35.0%) (p=0.002). No significant differences were observed in other adverse events such as hypertension (SBP >20% above baseline), reactive hypertension, or dizziness between the groups [Table 3]. Neonatal outcomes, including Apgar scores at 1 and 5 minutes and umbilical artery pH, were comparable between the two groups and within normal ranges (p>0.05 for all).

Table 3: Adverse Events and Neonatal Outcomes

Outcome	Group P (n=60)	Group C (n=60)	p-value
Adverse Events			
Hypotension (SBP<80% BL or <90mmHg)	9 (15.0%)	33 (55.0%)	<0.001*
Bradycardia (HR<50 bpm)	5 (8.3%)	11 (18.3%)	0.097*
Nausea/Vomiting	7 (11.7%)	21 (35.0%)	0.002*
Hypertension (SBP>20% BL)	3 (5.0%)	1 (1.7%)	0.621†
Dizziness	2 (3.3%)	4 (6.7%)	0.678†
Neonatal Outcomes			
Apgar Score at 1 min	8.7 ± 0.5	8.6 ± 0.6	0.312
Apgar Score at 5 min	9.0 ± 0.2	9.0 ± 0.3	0.851
Umbilical Artery pH	7.28 ± 0.04	7.27 ± 0.05	0.245

Data presented as n (%) or mean ± SD. *Chi-square test, †Fisher's exact test. BL: Baseline; SBP: Systolic Blood Pressure; HR: Heart Rate.

Discussion

This randomized, double-blind, placebo-controlled trial demonstrates that a prophylactic infusion of phenylephrine (0.5 µg/kg/min), initiated immediately after spinal anaesthesia, is highly effective in preventing the hemodynamic disturbances associated with a standardized 5 IU oxytocin bolus administered during caesarean section. The primary finding was a significant reduction in the incidence of hypotension from 55% in the control group to 15% in the phenylephrine group (p<0.001). This was accompanied by significantly higher mean arterial pressures at all measured time points following oxytocin administration, markedly reduced requirements for rescue ephedrine, and a lower incidence of nausea and vomiting.

The high incidence of hypotension (55%) observed in our control group following the oxytocin bolus underscores the potent hemodynamic challenge posed by oxytocin when superimposed on spinal anaesthesia. This finding aligns with previous studies reporting significant hypotension rates (40-70%) after oxytocin bolus in similar settings.[6,18] The mechanisms involve the combined vasodilatory effects of both spinal sympathetic blockade and oxytocin itself, leading to a precipitous drop in systemic vascular resistance.[8,19] Our results confirm that this combination creates a substantial risk for maternal hemodynamic instability.

The efficacy of phenylephrine in this context can be attributed to its potent α1-adrenergic agonist activity, which causes vasoconstriction and directly counteracts the vasodilation induced by both spinal anaesthesia and oxytocin.[20] By maintaining SVR, phenylephrine effectively preserves MAP. The chosen infusion rate of 0.5 µg/kg/min is consistent with current guidelines for prophylaxis against spinal-induced hypotension and proved effective in this specific scenario targeting the oxytocin response.[11,21] The significant reduction in rescue ephedrine use (10% vs 48.3%) further validates the proactive efficacy of the phenylephrine infusion.

While phenylephrine is known to cause reflex bradycardia, our study found a lower incidence of clinically significant bradycardia (HR<50 bpm) in the phenylephrine group (8.3% vs 18.3%), although this difference did not reach statistical significance (p=0.097). The observed small but statistically significant decreases in mean HR in the phenylephrine group at early time points (T1-T10) are consistent with its known pharmacological profile but were not associated with adverse consequences. This suggests that at the dose used, the reflex bradycardia was modest and well-tolerated. The lack of significant difference in bradycardia incidence might be due to the study being underpowered for this specific secondary outcome or the counteracting effect of oxytocin's potential to cause initial tachycardia before reflex bradycardia.[6] The overall hemodynamic stability achieved with phenylephrine likely contributed to the significantly lower incidence of nausea and vomiting (11.7% vs 35.0%), as these symptoms are strongly correlated with hypotension in this setting.[22]

Our findings extend the existing literature by specifically isolating and quantifying the protective effect of prophylactic phenylephrine against the hemodynamic impact of the oxytocin bolus itself. Previous studies have primarily focused on

phenylephrine for preventing spinal-induced hypotension overall, often without distinguishing the specific contribution of oxytocin.[13,14] Some studies have explored alternative oxytocin administration strategies (slow infusion, lower doses) to reduce hemodynamic effects.[15,16,23] While effective, slow infusion may delay optimal uterine tone, and lower doses might be less effective for PPH prevention in high-risk cases. Our approach of using a standard oxytocin bolus (as recommended by many guidelines for PPH prophylaxis,[5] combined with targeted vasopressor prophylaxis offers a practical and effective solution to maintain both hemodynamic stability and uterine contraction efficacy.

The comparable neonatal outcomes (Apgar scores, umbilical artery pH) between the groups are reassuring. While the study was not powered to detect differences in rare neonatal adverse events, the lack of any negative impact suggests that the phenylephrine infusion protocol used is safe for the fetus. This is consistent with the established safety profile of phenylephrine in obstetric anaesthesia when used appropriately.[11,12,24]

Several limitations should be acknowledged. We used a fixed phenylephrine infusion rate; variable rate infusions titrated to blood pressure might offer even more precise control but were beyond the scope of this study. The study was conducted in a tertiary centre with experienced anaesthesiologists; results may differ in settings with less experience. We only assessed the first 30 minutes post-oxytocin, as this is the period of peak hemodynamic effect; longer-term effects were not evaluated. While we used a standardized oxytocin dose (5 IU), optimal dosing remains debated, and our findings may not directly translate to other doses. Finally, the sample size was calculated primarily for hypotension incidence; larger studies might be needed to definitively assess differences in bradycardia or rare adverse events.

In conclusion, this study provides robust evidence that a prophylactic phenylephrine infusion (0.5 µg/kg/min) is a highly effective strategy to prevent the hypotensive response to a standard 5 IU oxytocin bolus during caesarean section under spinal anaesthesia. It significantly reduces the incidence of hypotension, the need for rescue vasopressors, and associated nausea and vomiting, without compromising neonatal well-being or significantly increasing bradycardia risk. These findings support the integration of prophylactic phenylephrine infusion into standard anaesthetic protocols for caesarean section to enhance maternal hemodynamic safety when oxytocin bolus administration is planned.

Conclusion

This randomized controlled trial demonstrates that prophylactic phenylephrine infusion (0.5 µg/kg/min), initiated immediately after spinal anaesthesia, is highly effective in preventing hemodynamic instability induced by a 5 IU oxytocin bolus during elective caesarean section. Compared to placebo, phenylephrine significantly reduced the incidence of hypotension (15.0% vs 55.0%, p<0.001), maintained higher mean arterial pressures throughout the critical 30-minute period post-oxytocin, and markedly decreased the requirement for rescue ephedrine (10.0% vs 48.3%, p<0.001). The incidence of nausea and vomiting was also significantly lower in the phenylephrine group (11.7% vs 35.0%, p=0.002). While a modest reduction in heart rate was observed, the incidence of clinically significant bradycardia was not significantly increased. Neonatal outcomes were comparable and favorable in both groups. These findings confirm that prophylactic phenylephrine infusion is a safe and effective strategy to mitigate the significant hemodynamic challenge posed by the combination of spinal anaesthesia and oxytocin bolus administration, thereby enhancing maternal safety during caesarean delivery.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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