Section: Anaesthesiology

Original Research Article

COMPARISON OF NOREPINEPHRINE AND PHENYLEPHRINE INFUSION FOR THE PREVENTION OF POST-SPINAL HYPOTENSION FOR CESAREAN SECTION

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Received : 22/12/2024
Received in revised form : 10/02/2025
Accepted : 26/02/2025

Keywords:
Spinal anaesthesia, Caesarean section, post-spinal hypotension, Norepinephrine, Phenylephrine infusions.

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DOI: 10.47009/jamp.2025.7.1.194

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2025; 7 (1); 993-998

Abstract

Background: Spinal anaesthesia for caesarean section frequently results in hypotension, which can compromise maternal and fetal well-being. This study aimed to compare the efficacy and safety of norepinephrine and phenylephrine infusion in preventing the post-spinal hypotension during a caesarean section. **Materials and Methods:** This randomised, double-blind observational study included 130 ASA-PS II term parturients undergoing elective caesarean section under spinal anaesthesia in K. A. P. V Government Medical College, Trichy, from January2024 to July2024. They were assigned to norepinephrine (0–5 µ/min) or phenylephrine (0–100 µ/min) infusion groups. The primary outcome was hypotension (SBP <80% of baseline or <100 mmHg). Secondary outcomes included bradycardia (HR <50 bpm), intraoperative nausea and vomiting (IONV), hypertension, infusion boluses, and APGAR scores. **Result:** The results showed No significant difference was observed in the incidence of hypotension between the groups (p=0.171). However, bradycardia was significantly more frequent in the PE group (p=0.002), whereas no cases occurred in the NE group. Similarly, IONV was more common in the PE group (p=0.042). No cases of hypertension were observed in either group of the study. The NE group had consistently higher heart rates between 3 and 7 min post-spinal anaesthesia, with statistically significant differences at each time point (p<0.05). No significant differences were observed in the neonatal APGAR scores at 1 min (p=0.07) or 5 min (p=0.559). **Conclusion:** Both norepinephrine and phenylephrine effectively prevented post-spinal hypotension during caesarean delivery. However, norepinephrine has the advantage of a lower incidence of bradycardia, reduced need for bolus doses, and improved haemodynamic stability.

INTRODUCTION

Anaesthesia for a parturient is unique and demands exceptional skill and knowledge, as the anaesthesiologist is responsible for the well-being of both the mother and the fetus. Spinal anaesthesia is the preferred technique for caesarean sections because of its ease of administration, rapid onset, and effective sensory and motor blockade. However, hypotension remains the most frequently encountered complication, occurring in approximately 85% of patients undergoing elective caesarean section under spinal anaesthesia.[1]

Hypotension during subarachnoid block (SAB) for caesarean section can have significant adverse effects on both the mother and foetus. A major concern is the reduction in placental blood flow, leading to foetal hypoxia, asphyxia stress, and foetal acidosis.[2] Maternal complications include symptoms associated with decreased cardiac output (CO), such as nausea, vomiting, dizziness, and, in severe cases, impaired consciousness. Given these risks, managing hypotension effectively is vital.[3]

Several strategies have been employed to prevent and manage this condition. One commonly used preventive measure is left uterine displacement (LUD), which helps relieve aortocaval compression and improve venous return.[4]

Additionally, preloading or co-loading with crystalloids or colloids has been explored as a means to mitigate hypotension.[5]

Despite these measures, they often prove inadequate, and vasopressors are frequently required to promptly correct hypotension.[6]

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Vasopressors, including ephedrine, phenylephrine, and methoxamine, play pivotal roles in managing maternal hypotension following spinal anaesthesia. The mechanism underlying hypotension is the sympathetic blockade induced by spinal anaesthesia, which results in decreased systemic vascular resistance (SVR).[7] Therefore, the ideal vasopressor should counteract these effects by promoting vasoconstriction and maintaining blood pressure without causing significant adverse effects.[8]

Phenylephrine, a pure α-adrenergic receptor agonist, is currently the gold standard for treating spinal anaesthesia-induced hypotension.[9] It effectively restores blood pressure by increasing SVR; however, it is associated with dose-dependent reflex bradycardia and reduced cardiac output.[10] This reflex bradycardia may be detrimental in some situations, potentially compromising maternal and foetal well-being.[11]

Norepinephrine, an older vasopressor, has recently gained attention as a promising alternative to phenylephrine. Unlike phenylephrine, norepinephrine has modest β-adrenergic effects in addition to its α-adrenergic actions. Recent studies have indicated that norepinephrine is equally effective in managing maternal hypotension, with the added advantage of preserving cardiac output and reducing the incidence of bradycardia. Current evidence suggests that norepinephrine is a safer and more physiologically favourable alternative to phenylephrine. However, further research is warranted to confirm its long-term safety and efficacy in obstetric anaesthesia.

Aim

This study aimed to compare norepinephrine and phenylephrine infusions for the prevention of post-spinal hypotension during caesarean sections.

MATERIALS AND METHODS

This randomized, double-blinded observational study included 130 ASA-PS II full-term singleton parturients scheduled for lower segment caesarean section under subarachnoid block at K. A. P. V Government Medical College, Trichy, between January 2024 and July 2024. The study was conducted after receiving approval from the Institutional Ethics Committee, and informed consent was obtained from all women.

Inclusion Criteria

Patients classified as ASA Physical Status II, with singleton term pregnancy (≥37 weeks of gestation), and scheduled for elective caesarean delivery were included.

Exclusion Criteria

Patients with the onset of labour, known foetal abnormality, hypertension, cardiovascular or cerebrovascular disease, renal impairment, allergy to any study medication, maternal weight less than 50 kg or >100 kg, height < 140 cm or > 180 cm, and age < 18 years were excluded. Patients receiving monoamine oxidase (MAO) inhibitors or tricyclic antidepressants were excluded.

Methods

Patients meeting the inclusion criteria were randomly assigned to two groups (n=65 each) using a sealed envelope technique. Group NE received norepinephrine infusion (0–5 µ/min) with bolus doses as needed, whereas Group PE received phenylephrine infusion (0–100 µ/min) with boluses for hypotension.

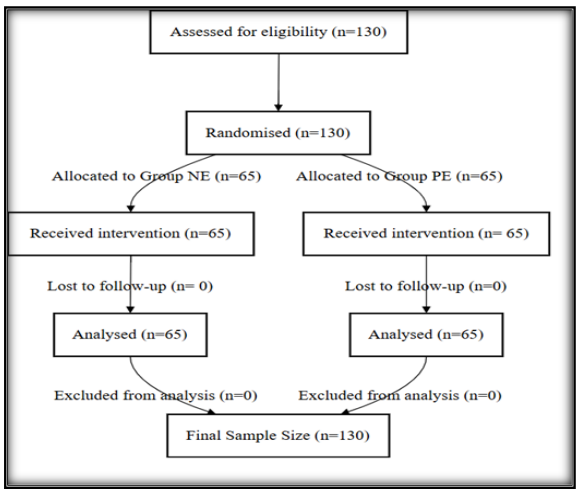
Preoperative investigations included Hb%, CBC, blood grouping, BT, CT, platelet count, blood sugar, blood urea, serum creatinine, and urine analyses. Patients received oral ranitidine (150 mg) and metoclopramide (10 mg) with sips of water two hours before surgery and were kept NPO for six hours for solids and two hours for clear liquids.

In the operating room, airway equipment and emergency drugs were prepared. Patients were positioned supine with a pillow under the head, and monitoring (NIBP, ECG, and pulse oximetry) was performed. Baseline SBP, DBP, and HR were recorded as the average of three consecutive measurements. Intravenous access was secured using an 18G cannula.

Spinal anaesthesia was administered in the right lateral position using a 25G Quincke's needle at L3–L4 or L4–L5, injecting 1.8 mL of 0.5% hyperbaric bupivacaine and 0.2 mL fentanyl intrathecally. Patients were positioned supine with a 15°–30° left tilt and received 4 L/min oxygen. Crystalloids were rapidly co-loaded up to 2 L and then reduced to maintenance.

The infusion (diluted in 50 mL of 5% dextrose) was started immediately after spinal anaesthesia at a rate of 30 mL/h. NIBP was monitored every minute until delivery and then every five minutes. Hypotension (SBP <80% of baseline or <100 mmHg) was managed by doubling the infusion rate (60 mL/h) and administering 1 mL rescue boluses if required. Hypertension (>120% baseline SBP) was managed by pausing the infusion and restarting it at 30 mL/h when the SBP normalised. Bradycardia (HR <50 bpm) with SBP ≥ baseline led to stopping the infusion; if SBP was < baseline, atropine (0.6 mg IV) was administered.

ISSN (O): 2687-5365; ISSN (P): 2753-6556



CONSORT flow diagram

Intraoperative nausea and vomiting (IONV) were recorded using a nausea-vomiting score (0: none, 1: nausea, 2: vomiting) and treated with ondansetron (4 mg IV) for scores ≥1. A blinded paediatrician assessed the Apgar scores at 1 and 5 min.

Statistical analysis: Data are presented as mean, standard deviation, frequency, and percentage. Continuous variables were compared using the independent sample t-test, and categorical variables were analysed using the Pearson chi-square test. Statistical significance was defined as a P-value <0.05 using a two-tailed test. Data analysis was performed using IBM SPSS version 21.0.

RESULTS

No significant differences were observed between the groups in terms of age (p=0.464), height (p=0.219), weight (p=0.423), BMI (p=0.986), or spinal-to-delivery interval (p=0.197) [Table 1].

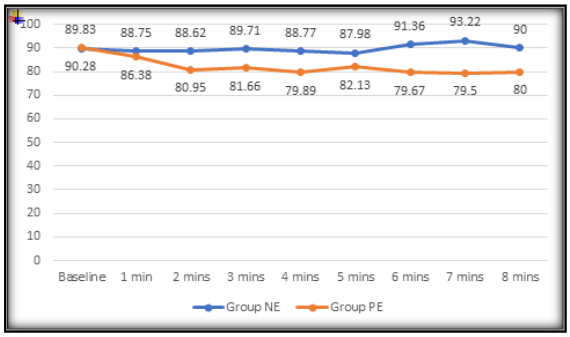


Figure 1: Comparison of heart rate between groups

There was no significant difference in the systolic blood pressure between the groups from baseline to 1 min. However, at 2–5 min, group NE had significantly higher systolic blood pressure than group PE, with the most notable differences at 2, 3, and 5 min (p<0.0001). No significant difference was observed between 6 and 8 min [Figure 2].

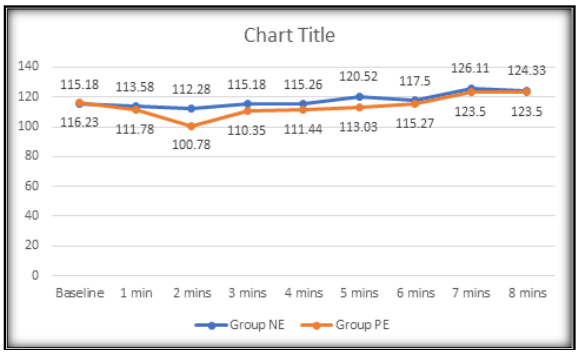


Figure 2: Comparison of SBP between groups

The comparison of DBP between groups showed no significant difference in diastolic blood pressure between groups at baseline to 8 min [Figure 3].

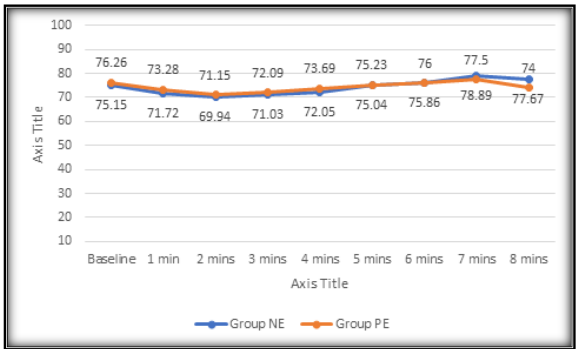


Figure 3: Comparison of DBP between groups

Table 1: Comparison of demographic and clinical characteristics between groups.

	Group (Mean±SD)		P value
	NE	PE	
Age (years)	25.45±4.33	25.00±2.29	0.464
Height (cm)	156.23±6.22	157.49±5.39	0.219
Weight (kg)	63.23±5.31	61.88±5.25	0.423
BMI	25.93±1.98	25.94±1.71	0.986
Spinal-to-Delivery Interval (min)	5.02±1.17	4.75±1.13	0.197

No significant difference in hypotension was observed between the groups (p=0.171). However, bradycardia and IONV were significantly more frequent in the group PE (p=0.002 and p=0.042, respectively), whereas no cases occurred in the group NE. Hypertension was not observed in either group [Table 2].

ISSN (O): 2687-5365; ISSN (P): 2753-6556

Table 2: Comparison of adverse events between groups

		Group		P value
		NE	PE	
Hypotension	No	64 (96.9%)	61 (86.2%)	0.171
	Yes	1 (3.1%)	4 (13.8%)	
Bradycardia	No	65 (100%)	56 (93.8%)	0.002
	Yes	0	9 (6.2%)	
IONV	No	65 (100%)	61 (93.8%)	0.042
	Yes	0	4 (6.2%)	
Hypertension	No	65 (100%)	65 (100%)	-

No significant difference was observed in the number of infusion boluses between the groups (p=0.06). Similarly, APGAR scores at 1 and 5 min showed no significant differences (p=0.07 and p=0.559, respectively). Most neonates in both groups had scores of 9 or 10 at both time points [Table 3].

Table 3: Comparison of infusion boluses and APGAR scores between groups

		Group		P value
		NE	PE	
No. of Infusion boluses	0	65 (96.9%)	58 (86.2%)	0.06
	1	0 (1.5%)	4 (10.8%)	
	2	0 (1.5%)	2 (1.5%)	
	3	0	1 (1.5%)	
APGAR - 1st min	7	0	0	0.07
	8	0	2 (1.5%)	
	9	63 (96.9%)	63 (96.9%)	
	10	2 (1.5%)	0	
APGAR - 5th min	9	1 (1.5%)	2 (4.6%)	0.559
	10	64 (98.5%)	63 (95.4%)	

The present study showed no significant difference in heart rate between the groups from baseline to 2 min. However, at 3 to 7 min, group NE consistently had a higher mean heart rate than group PE, with significant differences at each time point (p<0.05). The differences were most pronounced at 3, 4, and 6 min (p<0.0001). No significant difference was observed at 8 min (p=0.192) [Figure 1].

DISCUSSION

In our study, both NE and PE effectively reduced post-spinal maternal hypotension (p=0.171), with comparable efficacy, despite more patients requiring additional boluses of PE (p=0.06). SBP was lower in the PE group from 2 to 6 min post spinal anaesthesia, likely due to the lower initial infusion dose (50 µg/min). The low IONV in both groups showed stable blood pressure, further minimised by routine metoclopramide and ranitidine use. Bradycardia was more frequent with PE (p<0.005, 3rd–7th minute) due to its α-adrenergic effect, while NE's weak β-adrenergic activity maintained a higher HR and SBP, which is beneficial in high-risk cases. Our dosing followed PE:NE equipotency (20:1, 100 µg PE ≈ 5 µg NE). Neonatal outcomes were favourable, with no significant APGAR differences at 1 min (p=0.07) or 5 min (p=0.559).

The meta-analysis by Jianli et al. provides robust evidence to support our findings. Their analysis of 26 RCTs involving 2984 participants showed no significant differences between norepinephrine and phenylephrine in neonatal umbilical artery pH and Apgar scores. However, norepinephrine was associated with a lower incidence of maternal bradycardia (RR 0.44; 95% CI 0.37 to 0.51; p < 0.001) and reactive hypertension (RR 0.53; 95% CI 0.39 to 0.72; p < 0.001).[12] These findings are consistent with our results, emphasising the potential benefits of norepinephrine in maintaining maternal haemodynamic stability without compromising neonatal outcomes.

Similarly, Rai et al. compared norepinephrine and phenylephrine in elective caesarean deliveries and reported that neurobehavioral scale scores were significantly higher in the norepinephrine group at 24 and 48 h (p = 0.007 and 0.002, respectively). The incidence of bradycardia (p = 0.009), reactive hypertension (p = 0.003), and atropine requirement (p = 0.005) were significantly higher in the phenylephrine group,[13] supporting our observations that norepinephrine provides better heart rate stability.

Biricik et al. examined the effects of epinephrine, norepinephrine, and phenylephrine on maternal hypotension. They found that while the incidence of hypotension was comparable across the groups, norepinephrine and phenylephrine were superior to saline in reducing ephedrine consumption.[14] Similarly, Eskandr et al. reported that MAP was higher in the ephedrine group. However, maternal tachycardia was significantly more common with ephedrine, while bradycardia was more frequent in the phenylephrine group.[15] Their findings align with ours, suggesting that norepinephrine offers a better haemodynamic profile than phenylephrine while avoiding the tachycardic effects of ephedrine.

Mohta et al. specifically studied patients with preeclampsia and found that umbilical artery pH was comparable between norepinephrine and

ISSN (O): 2687-5365; ISSN (P): 2753-6556

phenylephrine (7.26 ± 0.06 vs. 7.27 ± 0.06; p = 0.903). However, the median number of hypotensive episodes was significantly higher in the norepinephrine group (p = 0.014), although the total vasopressor bolus requirements and systolic blood pressure trends were comparable.[16] This is consistent with our study, in which the incidence of hypotension was similar between groups, possibly due to differences in patient populations and norepinephrine dosing strategies.

Puthenveettil et al. and Singh et al. also found that norepinephrine required fewer boluses than phenylephrine, reducing the overall vasopressor requirement. This finding is consistent with our findings that norepinephrine may provide a more sustained haemodynamic response. Additionally, Singh et al. found that umbilical artery base excess was significantly higher in the norepinephrine group (-5.4 vs. -6.95; p = 0.014), suggesting better acid-base balance with norepinephrine.[17,18] Tiwari et al. confirmed that norepinephrine required fewer bolus doses (p = 0.02) and was associated with a lower incidence of bradycardia (p = 0.03).[19] Wang et al. observed similar trends, with norepinephrine resulting in fewer episodes of bradycardia than phenylephrine (3.6% vs. 21.8%; p = 0.004) and tachycardia than ephedrine (16.1% vs. 36.4%; p = 0.02).[20] These results collectively support the advantages of norepinephrine in maintaining stable maternal heart rates. Xu et al. conducted a systematic review and found no significant differences between norepinephrine and phenylephrine in the treatment of maternal hypotension (OR 0.64; p = 0.11) or hypertension (OR 0.74; p = 0.45). However, norepinephrine was associated with a significantly lower risk of bradycardia (OR 0.29; p = 0.005) and intraoperative nausea and vomiting (OR 0.54; p = 0.04),[21] which is consistent with our results. Wu et al. reported that cerebral tissue oxygen saturation (SctO2) reduction was significantly lower in the norepinephrine group than in the phenylephrine group (p = 0.02).[22] This suggests that norepinephrine may offer advantages in preserving cerebral perfusion, which could have long-term implications for maternal and neonatal outcomes.

Limitations

One concern is the administration of norepinephrine via the peripheral veins. However, none of the patients in our study experienced extravasation or paleness at the infusion site of diluted norepinephrine or phenylephrine. To ensure safety, we used a wide-bore peripheral intravenous cannula for all vasoactive drug infusions in this study. The potential for investigator bias with manually controlled infusions, as they are labour-intensive. To minimise this, we blinded the study solutions, maintained equal volumes for both drug solutions, and assigned an independent, blinded observer to manage and record the infusions during the caesarean delivery.

The inability to measure cardiac output and umbilical cord blood gases due to logistical constraints. Additionally, blood pressure recordings were obtained noninvasively, which may have introduced artefacts and imprecise timing, particularly during infusion rate adjustments or rescue bolus administration. This could be mitigated by performing error calculations. However, invasive arterial monitoring is not routinely used for uncomplicated caesarean deliveries. Future research involving parturients with severe pre-eclampsia compromised uteroplacental flow, or significant maternal cardiovascular conditions may warrant the use of invasive monitoring to enhance the applicability of these findings to high-risk obstetric populations.

CONCLUSION

Our findings show that norepinephrine and phenylephrine infusions are equally effective in preventing post-spinal hypotension. However, norepinephrine infusion has a significant advantage over phenylephrine infusion in terms of decreased risk of bradycardia, reduction in the amount of medication needed, and better neonatal outcomes. Further studies are needed to investigate the use of norepinephrine infusions in routine clinical practice.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556

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ISSN (O): 2687-5365; ISSN (P): 2753-6556