

COMPARING THE SENSITIVITY OF EPHEDRINE IN PREGNANCY INDUCED HYPERTENSION PATIENTS AND NORMOTENSIVE PATIENTS UNDERGOING LSCS

P. Karthik¹, A.Gnanavel Rajan², Praveen Kumar M¹

Received : 22/04/2026
Received in revised form : 02/06/2026
Accepted : 19/06/2026

Keywords:
Pregnancy-induced hypertension,
Ephedrine, Spinal anaesthesia,
Caesarean section, Hypotension,
Haemodynamic response.

Corresponding Author:
Dr. Praveen Kumar M,
Email: dr.praveenkumar.m.b.b.s@gmail.com

DOI: 10.47009/jamp.2026.8.3.261

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

Int J Acad Med Pharm
2026; 8 (3); 1482-1486



¹Assistant Professor, Department of Anaesthesiology, Government Medical College, The Nilgiris, Tamilnadu, India

²Associate Professor, Department of Anesthesiology, Government Medical College, Tirunelveli, Tamilnadu, India

ABSTRACT

Background: Spinal anaesthesia-induced hypotension is common during lower-segment caesarean sections (LSCS), requiring prompt vasopressor management for maternal stability. Pregnancy-induced hypertension (PIH) may affect vascular responsiveness and vasopressor sensitivities. This study compared ephedrine sensitivity in patients with PIH and normotensive patients undergoing lower-segment caesarean section under spinal anaesthesia. **Materials and Methods:** This prospective cohort study included 120 patients who underwent elective LSCS. The patients were divided into normal and PIH groups, with 60 patients in each group. Spinal anaesthesia was administered with 0.5% hyperbaric bupivacaine. Hypotension was treated with an intravenous ephedrine 6 mg bolus. Systolic, diastolic, and mean arterial pressures (MAP) were recorded at baseline and before and after ephedrine administration at 5, 15, and 30 min. **Result:** The mean age was similar between the groups (26.7 ± 5.1 vs. 27.3 ± 6.8 years; $p=0.568$). Baseline systolic and diastolic pressures were significantly higher in the PIH group ($P < 0.001$). Before ephedrine administration, systolic and MAP were higher in the PIH group, whereas diastolic pressure was comparable. At 5 min post-ephedrine, systolic pressure was similar between the groups (114.5 ± 11.2 vs. 115.0 ± 8.4 mmHg; $p=0.797$), but diastolic and MAP were higher in the PIH group. Absolute systolic pressure rise (32.6 ± 16.2 vs. 24.7 ± 7.3 mmHg; $p=0.001$) and percentage rise ($42.9 \pm 28.3\%$ vs. $27.7 \pm 9.1\%$; $p < 0.001$) were greater in the normal group. **Conclusion:** PIH alters the haemodynamic response to ephedrine during spinal anaesthesia for LSCS. Normotensive patients demonstrated a greater systolic blood pressure rise following ephedrine administration, whereas diastolic pressure remained higher in the PIH group, indicating differing vasopressor response patterns.

INTRODUCTION

Spinal anaesthesia is preferred for caesarean delivery because of its rapid onset, reliable sensory blockade, and safety for the mother and neonate. It reduces foetal exposure to anaesthetic agents and improves neonatal outcomes compared to general anaesthesia. However, maternal hypotension often occurs during lower-segment caesarean sections (LSCS) shortly after intrathecal drug administration.^[1,2] Hypotension results from sympathetic blockade, causing vasodilation, reduced systemic vascular resistance, and decreased venous return (VR). Severe hypotension can impair uteroplacental perfusion, leading to foetal hypoxia, acidosis, adverse neonatal outcomes, and maternal symptoms such as nausea and dizziness.^[1,2] Preventing and managing spinal

anaesthesia-induced hypotension are crucial in obstetric anaesthesia. Vasopressors counteract vasodilation and restore maternal arterial pressure, thereby preserving uteroplacental blood flow and foetal oxygenation. Along with fluid therapy, vasopressors enhance maternal haemodynamic stability and perinatal outcomes.^[1,3]

Ephedrine, which has α - and β -adrenergic activity, is widely used in obstetric anaesthesia. Its sympathomimetic mechanism increases blood pressure, heart rate, and cardiac output, thereby maintaining uteroplacental perfusion.^[4,5] These properties favour its use in spinal anaesthesia-induced hypotension during caesarean delivery. However, phenylephrine is now preferred for its predictable haemodynamic effects and improved foetal acid-base status. Ephedrine is linked to foetal

acidosis due to increased foetal metabolic activity resulting from catecholamine stimulation.^[6] It shows variable dose responsiveness and may cause tachyphylaxis, requiring repeated doses to maintain the blood pressure.^[7,8] Maternal tachycardia is more common with ephedrine, which increases myocardial oxygen demand.⁸ Nonetheless, ephedrine remains relevant, especially in hypotension with bradycardia, where its β -adrenergic effects are advantageous over those of pure vasoconstrictors.^[9]

Vasopressor responsiveness is influenced by the maternal cardiovascular physiology. Pregnancy-induced hypertension (PIH), including preeclampsia, involves endothelial dysfunction, increased systemic vascular resistance, altered autonomic regulation, and heightened vasoconstrictor sensitivity, creating a vascular state distinct from normotensive pregnancy.^[10] Reduced nitric oxide bioavailability and impaired endothelial-dependent vasodilation elevate the basal vascular tone, whereas enhanced sympathetic activity augments vascular reactivity.^[11] Therefore, women with PIH show exaggerated responses to vasoconstrictor stimuli. These haemodynamic changes affect the spinal anaesthesia. Compared with normotensive parturients, patients with PIH often have a lower incidence and severity of spinal anaesthesia-induced hypotension due to pre-existing vasoconstriction and reduced vasodilatory reserve.^[12,13] Altered autonomic balance and increased catecholamine sensitivity in PIH may modify vasopressor responsiveness, such as that to ephedrine administration. Reduced intravascular volume and elevated vasoconstrictor tone can influence the vasopressor requirements in hypertensive parturients.^[10,11] These differences suggest that ephedrine sensitivity may differ between patients with PIH and normotensive patients under spinal anaesthesia.

Many studies have evaluated vasopressor use for spinal anaesthesia-induced hypotension, mostly comparing phenylephrine and ephedrine in obstetric populations. Evidence on ephedrine sensitivity and dosage in patients with PIH is limited. Some studies have reported lower ephedrine requirements in hypertensive parturients, while others have shown variable results due to differences in study design, hypotension definitions, anaesthetic techniques, fluid protocols, and vasopressor methods.^[14,15] Small sample sizes and heterogeneous populations limit conclusions regarding ephedrine responsiveness in PIH. Understanding the differential ephedrine sensitivity in PIH and normotensive pregnancies is crucial for optimising maternal haemodynamic management and foetal outcomes during caesarean delivery under spinal anaesthesia. Comparing ephedrine responsiveness between these groups may help refine vasopressor protocols and support individualised anaesthetic management in obstetric patients undergoing LSCS.

Aim

This study aimed to compare the sensitivity of ephedrine in patients with PIH and normotensive patients undergoing LSCS.

MATERIALS AND METHODS

This prospective cohort study was conducted in 120 patients attending the Department of Anaesthesiology, Government Medical College and Hospital, The Nilgiris, from [DATE]. Ethical approval was obtained from the Institutional Ethical Committee, and written informed consent was obtained from the participants before the study initiation.

Inclusion and Exclusion criteria

The study included pregnant women aged 20–40 years undergoing elective lower-segment caesarean section under spinal anaesthesia. Patients were allocated into two groups: Group Normal comprised normotensive parturient (ASA I–II), and Group PIH comprised parturients diagnosed with PIH. Patients with severe preeclampsia, eclampsia, seizure disorders, significant cardiac disease, multiple gestation, active labour, or refusal to participate were excluded.

Sample size calculation: A total of 120 patients (60 per group) were enrolled during the study period. This sample size was considered adequate as it exceeded the sample sizes reported in comparable obstetric anaesthesia studies evaluating vasopressor responses during caesarean section under spinal anaesthesia.^[16]

Materials: The materials used in the study included 0.5% hyperbaric bupivacaine, ephedrine injection, phenylephrine injection, an appropriately sized intravenous cannula, standard monitoring equipment, spinal anaesthesia tray, syringes, intravenous fluids, and all necessary emergency drugs.

Methods: One hundred twenty patients were divided into two groups: Group PIH and Group Normal, with 60 patients in each group. Patients fasted according to standard preoperative guidelines. In the operating room, standard monitors were attached, and baseline vital signs were recorded. Intravenous access was secured, and maintenance fluid was initiated. Spinal anaesthesia was administered aseptically using 0.06 mg/cm height of 0.5% hyperbaric bupivacaine. Hypotension was defined as a >25% decrease in mean arterial pressure (MAP) or systolic blood pressure (SBP) <100 mmHg and was treated with intravenous ephedrine 6 mg boluses. A fixed 6 mg dose was selected based on previous obstetric anaesthesia studies evaluating spinal anaesthesia-induced hypotension in preeclamptic and normotensive parturients.^[17] Patients with hypotension and tachycardia were treated with phenylephrine and excluded. The increase in systolic, diastolic, and MAP after ephedrine administration was compared between the groups. Blood pressure

was recorded at baseline, before ephedrine administration, and 5, 15, and 30 min after.

Statistical analysis: Data are presented as mean and standard deviation. Variables were compared using an unpaired t-test for continuous data. Significance was defined as $P < 0.05$, and analysis was performed using IBM-SPSS version 21 (IBM-SPSS Science Inc., Chicago, IL, USA).

RESULTS

The mean age was 26.7 ± 5.1 years in the normal group and 27.3 ± 6.8 years in the PIH group, with no significant difference ($p = 0.568$) [Table 1].

Table 1: Comparison of age distribution

Parameter	Normal (n=60)	PIH (n=60)	p-value
Age (years)	26.7 ± 5.1	27.3 ± 6.8	0.568

Table 2: Comparison of SBP at different time intervals

SBP	Normal	PIH	P value
Baseline	121.4 ± 7.8	153.0 ± 7.2	< 0.001
Pre-Ephedrine	81.8 ± 10.9	90.2 ± 7.1	< 0.001
5 min After	114.5 ± 11.2	115.0 ± 8.4	0.797
15 min After	103.9 ± 6.4	109.4 ± 7.0	< 0.001
30 min After	110.4 ± 7.6	121.0 ± 8.5	< 0.001

The baseline DBP was higher in the PIH group (99.3 ± 10.6 mmHg) than in the normal group (73.5 ± 5.6 mmHg) ($p < 0.001$). Before ephedrine administration, the DBP was similar between the groups (53.5 ± 6.5 mmHg vs. 52.8 ± 7.2 mmHg) with no significant difference ($p = 0.597$). At 5 min post-ephedrine, DBP was higher in the PIH group (73.3 ± 3.7 mmHg) than

Baseline SBP was higher in the PIH group (153.0 ± 7.2 mmHg) than in the normal group (121.4 ± 7.8 mmHg) ($p < 0.001$). Before ephedrine administration, it remained higher in the PIH group (90.2 ± 7.1 mmHg) than in the normal group (81.8 ± 10.9 mmHg) ($p < 0.001$). At 5 min post-ephedrine, SBP was similar between the groups (114.5 ± 11.2 mmHg vs. 115.0 ± 8.4 mmHg) ($p = 0.797$). However, at 15 and 30 min, it was higher in the PIH group (109.4 ± 7.0 mmHg vs. 103.9 ± 6.4 mmHg and 121.0 ± 8.5 mmHg vs. 110.4 ± 7.6 mmHg, respectively) ($p < 0.001$) [Table 2].

in the normal group (68.0 ± 4.8 mmHg) ($p < 0.001$). At 15- and 30-min post-ephedrine, DBP remained higher in the PIH group than in the normal group (67.0 ± 4.5 mmHg vs. 65.3 ± 4.8 mmHg and 73.5 ± 8.7 mmHg vs. 70.0 ± 5.0 mmHg) with significance ($p = 0.042$ and $p = 0.008$, respectively) [Table 3].

Table 3: Comparison of DBP groups at different time intervals

DBP	Normal	PIH	P value
Baseline	73.5 ± 5.6	99.3 ± 10.6	< 0.001
Pre-Ephedrine	53.5 ± 6.5	52.8 ± 7.2	0.597
5 min After	68.0 ± 4.8	73.3 ± 3.7	< 0.001
15 min After	65.3 ± 4.8	67.0 ± 4.5	0.042
30 min After	70.0 ± 5.0	73.5 ± 8.7	0.008

The baseline MAP was higher in the PIH group (117.2 ± 7.0 mmHg) than in the normal group (89.5 ± 5.6 mmHg) ($p < 0.001$). Before ephedrine administration, the MAP was higher in the PIH group (65.3 ± 4.5 mmHg) than in the normal group (62.9 ± 6.5 mmHg) ($p = 0.023$). At 5 min post-ephedrine, it was higher in the PIH group (87.2 ± 4.0 mmHg) than

in the normal group (83.5 ± 5.3 mmHg) ($p < 0.001$). At 15- and 30-min post-ephedrine, the MAP remained higher in the PIH group than in the normal group (81.2 ± 3.7 mmHg vs. 78.1 ± 4.0 mmHg and 89.4 ± 8.2 mmHg vs. 83.5 ± 5.0 mmHg, respectively) with statistical significance ($p < 0.001$) [Table 4].

Table 4: Comparison of MAP at different time intervals

MAP	Normal	PIH	P value
Baseline	89.5 ± 5.6	117.2 ± 7.0	< 0.001
Pre-Ephedrine	62.9 ± 6.5	65.3 ± 4.5	0.023
5 min After	83.5 ± 5.3	87.2 ± 4.0	< 0.001
15 min After	78.1 ± 4.0	81.2 ± 3.7	< 0.001
30 min After	83.5 ± 5.0	89.4 ± 8.2	< 0.001

The SBP before ephedrine administration was higher in the PIH group (90.2 ± 7.1 mmHg) than in the normal group (81.8 ± 10.9 mmHg) ($p < 0.001$). Five minutes after ephedrine administration, the SBP was similar (115.0 ± 8.4 mmHg vs. 114.5 ± 11.2 mmHg)

($p = 0.797$). The absolute rise in SBP was greater in the normal group (32.6 ± 16.2 mmHg) than in the PIH group (24.7 ± 7.3 mmHg) ($p = 0.001$). The percentage increase was also higher in the normal group ($42.9 \pm 28.3\%$) than in the PIH group ($27.7 \pm 9.1\%$)

($p < 0.001$). DBP before ephedrine administration was similar (53.5 ± 6.5 mmHg vs. 52.8 ± 7.2 mmHg) ($p = 0.597$). Five minutes later, the DBP was higher in

the PIH group (73.3 ± 3.7 mmHg) than in the normal group (68.0 ± 4.8 mmHg) ($p < 0.001$).

Table 5: Comparison of systolic and DBP response following ephedrine administration

	Normal Group	PIH Group	P value
SBP pre-ephedrine (mmHg)	81.8 $\pm$ 10.9	90.2 $\pm$ 7.1	<0.001
SBP at 5 min After (mmHg)	114.5 $\pm$ 11.2	115.0 $\pm$ 8.4	0.797
Absolute SBP Rise (mmHg)	32.6 $\pm$ 16.2	24.7 $\pm$ 7.3	0.001
% Rise in SBP	42.9 $\pm$ 28.3%	27.7 $\pm$ 9.1%	<0.001
DBP pre-ephedrine (mmHg)	53.5 $\pm$ 6.5	52.8 $\pm$ 7.2	0.597
DBP at 5 min After (mmHg)	68.0 $\pm$ 4.8	73.3 $\pm$ 3.7	<0.001

DISCUSSION

Our study assessed ephedrine sensitivity in patients with PIH and normotensive patients undergoing spinal anaesthesia for caesarean section. Significant differences in the systolic, diastolic, and MAP responses were observed after ephedrine administration. Patients with PIH had higher baseline haemodynamic parameters and a lower immediate SBP response than normotensive patients. The mean age was similar between the groups, indicating demographic homogeneity. Lohokare et al. found no age difference between healthy and preeclamptic patients (25.94 ± 4.09 vs. 26.07 ± 3.95 years; $p = 0.82$).^[18] Fayaz et al. also reported no significant age difference (29.66 ± 2.78 vs. 30.47 ± 3.01 years; $p = 0.267$).^[19] These findings suggest that age did not influence haemodynamic variations.

Our study showed significantly higher baseline systolic, diastolic, and MAP in the PIH group. Fayaz et al. reported significantly elevated baseline SBP (162.53 ± 14.65 mmHg vs. 134.47 ± 6.41 mmHg; $p < 0.001$), DBP (104.09 ± 14.65 mmHg vs. 82.72 ± 9.06 mmHg; $p < 0.001$), and MAP (123.57 ± 13.98 mmHg vs. 99.98 ± 7.29 mmHg; $p < 0.001$) in preeclamptic patients.^[19] Lohokare et al. also reported higher baseline SBP (149.46 ± 8.74 mmHg vs. 114.83 ± 7.4 mmHg; $p < 0.001$) and DBP (88.72 ± 5.83 mmHg vs. 75.67 ± 6.27 mmHg; $p < 0.001$).^[18] These findings support the pathophysiology of PIH, which is characterised by increased vascular resistance and endothelial dysfunction.

Before ephedrine administration post-spinal anaesthesia, the SBP was higher in the PIH group, whereas the DBP was similar, and the MAP was higher. These observations are partly consistent with those of Lohokare et al., who reported similar SBP decreases in both healthy and preeclamptic patients, with percentage reductions of $20.03 \pm 11.58\%$ and $21.99 \pm 8.54\%$.^[18] However, Fayaz et al. reported more profound reductions in SBP, DBP, and MAP in preeclamptic patients after spinal anaesthesia, with SBP reduction of 47.7% versus 15.7% , DBP reduction of 51.3% versus 24.6% , and MAP reduction of 49.9% versus 19.3% compared to normotensive patients.^[19] The differences between studies may be due to variations in spinal anaesthesia techniques, vasopressor protocols, and hypotension definitions.

Our study showed similar systolic blood pressure values at 5 min following ephedrine administration in both groups; however, the absolute and percentage increase in systolic blood pressure was greater in normotensive patients. In contrast, diastolic blood pressure at 5 min was significantly higher in the PIH group, suggesting that the haemodynamic response to ephedrine may differ between systolic and diastolic components of blood pressure in patients with PIH. Lohokare et al. found lower ephedrine doses in preeclamptic patients than healthy ones (3.07 ± 1.27 mg vs. 5.67 ± 5.44 mg).^[18] The SBP in our study at 15- and 30-min post-ephedrine, as well as the diastolic and MAP, were higher in the PIH group. Fayaz et al. observed delayed recovery in preeclamptic patients, with systolic pressure lowest at 25 minutes compared to 5 minutes in normotensive patients. The MAP at 120 min was higher in preeclamptic patients (97.18 ± 8.49 mmHg vs. 89.72 ± 7.13 mmHg; $p < 0.001$).^[19] This finding may reflect differences in haemodynamic recovery patterns between hypertensive and normotensive parturients.

Our study supports the notion that PIH modifies vasopressor responsiveness during spinal anaesthesia. Ituk et al. reported hypotension requiring vasopressors in 43% of preeclamptic patients, with lower median vasopressor requirements for ephedrine than for phenylephrine (250 mcg vs. 600 mcg; $p = 0.005$). No significant difference in neonatal umbilical artery pH was observed between the groups.^[20] El-Shaer et al. showed that ephedrine maintained better blood pressure during caesarean sections with fewer hypotension episodes.^[21] Our study showed that while patients with PIH had higher baseline blood pressure and maintained higher values post-ephedrine, the immediate rise was greater in normotensive patients. This suggests differing vasopressor response patterns in PIH compared with normotensive patients. The underlying mechanisms were not evaluated in the present study; however, altered vascular reactivity associated with hypertensive disorders of pregnancy may contribute to these observations.

CONCLUSION

PIH affects the haemodynamic response to ephedrine during spinal anaesthesia for caesarean delivery. Patients with PIH had higher baseline systolic,

diastolic, and MAP than normotensive patients. Although the SBP at 5 min following ephedrine administration was similar between the groups, the absolute and percentage SBP increase was greater in normotensive patients, indicating a different systolic haemodynamic response to ephedrine in patients with PIH. DBP and MAP remained higher in patients with PIH following ephedrine administration. The mechanisms underlying these findings were not specifically evaluated in the present study. These findings suggest that patients with PIH exhibit different vasopressor response patterns during spinal anaesthesia-induced hypotension management. These findings suggest that haemodynamic responses to ephedrine may differ between normotensive and PIH patients undergoing caesarean delivery under spinal anaesthesia. Further studies evaluating dose-response relationships and vasopressor requirements are required to better understand the haemodynamic response to ephedrine in patients with PIH.

REFERENCES

1. Abo Kamar MM, Shams MM, AbdelAziz MM, Selima WZ. Norepinephrine versus ephedrine boluses to treat spinal-induced hypotension in cesarean deliveries. *QJM* 2021;114. <https://doi.org/10.1093/qjmed/hcab086.062>.
2. Chauhan JM, Taral RJ, Moradiya MR. Hemodynamic changes after spinal anesthesia in cesarean section: A prospective observational study. *Int J Curr Pharm Res* 2026;18. <https://doi.org/10.25258/ijcpr.18.1.28>.
3. Sng BL, Du W, Lee MX, Ithnin F, Mathur D, Leong WL, et al. Comparison of double intravenous vasopressor automated system using Nexfin versus manual vasopressor bolus administration for maintenance of hemodynamic stability during spinal anesthesia for cesarean delivery: A randomized double-blind controlled trial: A randomized double-blind controlled trial. *Obstet Anesth Dig* 2019; 39:31–2. <https://doi.org/10.1097/01.aoa.0000552905.95920.2b>.
4. Uerpairajkit K, Anusornanawat R, Sirisabya A, Chaichalothorn M, Charuluxananana S. Neonatal effects after vasopressor during spinal anesthesia for cesarean section: A multicenter, randomized controlled trial: A multicenter, randomized controlled trial. *Obstet Anesth Dig* 2018; 38:141–2. <https://doi.org/10.1097/01.aoa.0000542363.25072.92>.
5. Dusitkasem S, Herndon BH, Sonjit M, Stahl DL, Bitticker E, Coffman JC. Comparison of phenylephrine and ephedrine in treatment of spinal-induced hypotension in high-risk pregnancies: A narrative review. *Front Med (Lausanne)* 2017; 4:2. <https://doi.org/10.3389/fmed.2017.00002>.
6. Fuentealba Ramírez R, Espinoza Ravanales M, González Antío J, Riquelme Bahamondes C. Efectos materno-fetales del uso de drogas vasoactivas en el manejo de hipotensión posterior a anestesia espinal en operación cesárea: revisión sistemática. *Rev Chil Anest* 2025;54. <https://doi.org/10.25237/revchilanestv54n6-17>.
7. Ohorella SAV, Aisyah AP, Putri RA, Qoblianingtyas A, Safrian S, Dewi RK. Scoping Review: Efektivitas Vasopresor terhadap Hipotensi Intraoperatif. *Jurnal Siti Rufaidah* 2025; 3:242–53. <https://doi.org/10.57214/jasira.v3i4.267>.
8. Shah M, Manjua SK, Javid U, Shameem A, Anjum W, Ali W, et al. Comparison of phenylephrine and ephedrine in managing spinal-induced hypotension in lower segment caesarean section (LSCS): Phenylephrine and ephedrine in managing spinal-induced hypotension in LSCS. *Pak J Health Sci* 2026;120–5. <https://doi.org/10.54393/pjhs.v7i1.3641>.
9. Habib AS. A review of the impact of phenylephrine administration on maternal hemodynamics and maternal and neonatal outcomes in women undergoing cesarean delivery under spinal anesthesia. *Anesth Analg* 2012; 114:377–90. <https://doi.org/10.1213/ANE.0b013e3182373a3e>.
10. Spradley FT. Sympathetic nervous system control of vascular function and blood pressure during pregnancy and preeclampsia. *J Hypertens* 2019; 37:476–87. <https://doi.org/10.1097/HJH.0000000000001901>.
11. Pal GK, Habeebullah S, Subha M, Pal P. Sympathovagal imbalance, cardiometabolic risks and hypertension status are linked to depression in women having risk factors for pregnancy-induced hypertension. *Int J Clin Exp Physiol* 2021; 8:59–64. <https://doi.org/10.5530/ijcep.2021.8.2.15>.
12. Nateghi MR, Saremi A, Abbasi B, Karimi MansoorAbad E. Balancing blood pressure: Understanding hemodynamic fluctuations in spinal anesthesia. *Sarem Journal of Medical Research* 2023; 8:37–42. <https://doi.org/10.61186/sjrm.8.1.37>.
13. Zagrodnik E, Szczuko M, Surówka A, Ziętek M. Hemodynamic changes during cesarean section under spinal anesthesia in normotensive and hypertensive pregnant women-A narrative review. *J Clin Med* 2026; 15:2162. <https://doi.org/10.3390/jcm15062162>.
14. Nikooseresht M, Seif Rabiei MA, Hajian P, Dastaran R, Alipour N. Comparing the hemodynamic effects of spinal anesthesia in preeclamptic and healthy parturients during cesarean section. *Anesth Pain Med* 2016;6: e11519. <https://doi.org/10.5812/aapm.11519>.
15. Tawfik MM, Tarbay AI, Elaidy AM, Awad KA, Ezz HM, Tolba MA. Combined colloid preload and crystalloid coload versus crystalloid coload during spinal anesthesia for cesarean delivery: A randomized controlled trial: A randomized controlled trial. *Anesth Analg* 2019; 128:304–12. <https://doi.org/10.1213/ANE.0000000000003306>.
16. Elagamy AE, Kamaly AM, Shahn MI, Saleh M. Norepinephrine versus ephedrine for hypotension prophylaxis during cesarean section under spinal anesthesia. *Ain-Shams J Anaesthesiol* 2021;13. <https://doi.org/10.1186/s42077-020-00124-4>.
17. Clark VA, Sharwood-Smith GH, Stewart AVG. Ephedrine requirements are reduced during spinal anaesthesia for caesarean section in preeclampsia. *Int J Obstet Anesth* 2005;14:9–13. <https://doi.org/10.1016/j.ijoa.2004.08.002>.
18. Lohokare B, Jambotkar T, Gupta H. Hemodynamic response and vasopressor requirement after spinal anaesthesia in preeclamptic and non-preeclamptic patients undergoing caesarian section: A prospective comparative study. *Int J Sci Res* 2020; 9:16–8. [https://www.worldwidejournals.com/international-journal-of-scientific-research-\(IJSR\)/fileview/hemodynamic-response-and-vasopressor-requirement-after-spinal-anesthesia-in-preeclamptic-and-nonandamp-ndash-preeclamptic-patients-undergoing-caesarian-section-a-prospective-comparative-study_March_2020_1582978891_1507042.pdf](https://www.worldwidejournals.com/international-journal-of-scientific-research-(IJSR)/fileview/hemodynamic-response-and-vasopressor-requirement-after-spinal-anesthesia-in-preeclamptic-and-nonandamp-ndash-preeclamptic-patients-undergoing-caesarian-section-a-prospective-comparative-study_March_2020_1582978891_1507042.pdf)
19. Fayaz MF, Mr. Talat Ul Tuba Dar, Dr. Minnu M Panditrao. Hemodynamic variations following spinal anaesthesia in normotensive versus preeclamptic women undergoing caesarean sections: A comparative analysis. *Int J Pharm Med Sci* 2026; 6:26–35. <https://doi.org/10.64751/ijpams.2026.v6.n1.pp26-35>.
20. Ituk US, Cooter M, Habib AS. Retrospective comparison of ephedrine and phenylephrine for the treatment of spinal anesthesia induced hypotension in preeclamptic patients. *Curr Med Res Opin* 2016; 32:1083–8. <https://doi.org/10.1185/03007995.2016.1159953>.
21. El-Shaer AN, El-Hamid A, Saleh HM, Mahmoud M. The effect of volume preload versus ephedrine infusion for prevention of hypotension due to spinal anesthesia for cesarean section. *QJM* 2023;116. <https://doi.org/10.1093/qjmed/hcad069.046>.