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HEMODYNAMIC VARIATIONS FOLLOWING SPINAL ANAESTHESIA IN NORMOTENSIVE VERSUS PREECLAMPTIC WOMEN UNDERGOING CAESAREAN SECTIONS: A COMPARATIVE ANALYSIS

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ABSTRACT

Background: Preeclampsia significantly alters maternal hemodynamics and may influence responses to spinal anesthesia during cesarean delivery. Understanding these differences is crucial for optimizing anesthetic management and maternal safety.

Methods: This prospective observational study compared hemodynamic responses to spinal anesthesia in 64 parturients (32 preeclamptic, 32 normotensive) undergoing elective cesarean sections at Lal Ded Hospital, Srinagar. Hemodynamic parameters including blood pressure, heart rate, and oxygen saturation were monitored at 12 time points over 120 minutes. Statistical analysis employed independent t-tests and chi-square tests.

Results: Preeclamptic patients demonstrated significantly higher baseline blood pressure (SBP 162.53±14.65 vs 134.47±6.41 mmHg, $p<0.001$) but lower heart rate (99.34±13.92 vs 107.56±15.00 bpm, $p=0.025$). Following spinal anesthesia, preeclamptic women experienced more profound percentage decreases in blood pressure (SBP 47.7% vs 15.7%, DBP 51.3% vs 24.6%, MAP 49.9% vs 19.3%) with delayed nadir occurrence at 25 minutes versus 5 minutes. Vasopressor requirements were significantly higher in preeclamptic patients (84.4% vs 18.8%, $p<0.001$).

Conclusion: Preeclamptic patients exhibit distinct hemodynamic responses to spinal anesthesia characterized by more pronounced blood pressure reductions, delayed nadirs, and increased vasopressor requirements despite elevated baseline vascular tone, necessitating individualized monitoring and management strategies.

Keywords: Preeclampsia; Spinal Anesthesia; Hemodynamic Response; Cesarean Section; Vasopressor Requirements; Hypotension

INTRODUCTION

Preeclampsia, affecting 2-8% of pregnancies globally, represents a critical hypertensive disorder characterized by new-onset hypertension and proteinuria after 20 weeks gestation, with significant implications for maternal and fetal outcomes [1]. The condition fundamentally alters maternal physiology through endothelial dysfunction, increased systemic vascular resistance, and reduced plasma volume, creating a unique pathophysiological state that complicates anesthetic management [2,3]. Cesarean delivery, required in approximately 40-60% of preeclamptic pregnancies, necessitates careful consideration of anesthetic technique selection and hemodynamic management strategies [4].

Spinal anesthesia has emerged as the preferred technique for cesarean delivery in both normotensive and preeclamptic parturients due to its rapid onset, reliable neural blockade, minimal fetal drug exposure, and reduced maternal airway manipulation risks [5,6]. However, the sympathetic blockade inherent to spinal anesthesia produces profound alterations in cardiovascular homeostasis through peripheral vasodilation and venous pooling, potentially precipitating maternal hypotension and compromising uteroplacental perfusion [7].

Traditional teaching suggested that preeclamptic patients elevated vascular tone might protect against spinal-induced hypotension, though emerging evidence challenges this assumption and indicates more complex hemodynamic responses requiring specialized management approaches [8].

MATERIALS AND METHODS

This prospective observational study was conducted at Lal Ded Hospital, Srinagar, following institutional ethics committee approval and written informed consent from all participants. The study enrolled 64 parturients scheduled for elective cesarean section under spinal anesthesia, comprising 32 preeclamptic patients (cases) and 32 normotensive controls matched for gestational age and surgical indication. Preeclampsia was defined according to the American College of Obstetricians and Gynecologists criteria as blood pressure ≥140/90 mmHg on two occasions at least 4 hours apart after 20 weeks gestation, with proteinuria ≥300 mg in 24 hours or protein/creatinine ratio ≥0.3 [9].

Sample size calculation employed the formula $n = [(Z\alpha + Z\beta)^2 \times 2\sigma^2] / d^2$, with assumptions of α=0.05, power=80%, standard deviation of mean arterial pressure (σ)=15 mmHg, and minimum detectable difference (d)=10 mmHg, yielding a minimum requirement of 28 patients per group, increased to 32 to account for potential dropouts [10]. Inclusion criteria encompassed ASA physical status I-III parturients aged 18-40 years with singleton pregnancies at ≥37 weeks gestation scheduled for elective cesarean delivery. Exclusion criteria included emergency procedures, known cardiovascular disease, coagulation disorders, contraindications to spinal anesthesia, multiple gestations, and patient refusal [11,12].

All patients received standardized preoperative management including 6-8 hours fasting, intravenous access with 18G cannula, and preloading with 10-15 mL/kg lactated Ringer's solution over 15-20 minutes before spinal anesthesia [13]. Standard monitoring consisted of continuous electrocardiography, pulse oximetry, and automated non-invasive blood pressure measurement at 2-minute intervals initially, extending to 5-minute intervals after stabilization [14]. Spinal anesthesia was performed in the sitting position at the L3-L4 or L4-L5 interspace using a 25G Quincke needle, administering 10 mg hyperbaric bupivacaine 0.5% (2 mL) with 25 μg fentanyl [15]. Patients were immediately positioned supine with 15° left lateral tilt to minimize aortocaval compression.

Hemodynamic parameters including systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), and peripheral oxygen saturation (SpO_2) were recorded at baseline and at 3, 5, 10, 15, 20, 25, 30, 45, 60, 90, and 120 minutes post-spinal anesthesia. Hypotension, defined as SBP <100 mmHg or >20% decrease from baseline, was treated with intravenous phenylephrine 100 μg boluses, repeated as necessary [16]. Bradycardia (HR <50 bpm) received atropine 0.6 mg intravenously. All interventions, complications, and neonatal outcomes were documented systematically.

Statistical analysis utilized SPSS version 23.0 (IBM Corp., Armonk, NY). Continuous variables were expressed as mean ± standard deviation and compared using independent t-tests after confirming normal distribution through Kolmogorov-Smirnov tests. Categorical variables were presented as frequencies and percentages, analyzed using chi-square or Fisher's exact tests as appropriate. Repeated measures analysis assessed temporal changes in hemodynamic parameters. Statistical significance was set at $p<0.05$ for all comparisons.

RESULTS

The study enrolled 64 parturients successfully, with no patients excluded or lost to follow-up. Demographic characteristics demonstrated excellent matching between groups, with no statistically significant differences in age (preeclamptic: 29.66±2.78 years vs normotensive: 30.47±3.01 years, p=0.267), height (4.38±0.43 vs 4.47±0.46 inches, p=0.416), or weight (70.16±2.95 vs 69.75±5.34 kg, p=0.712) as shown in Table 1. However, ASA physical status distribution differed significantly (p<0.001), with preeclamptic patients exhibiting higher ASA classifications (ASA III: 56.3% vs 0%, ASA II: 43.8% vs 75.0%, ASA I: 0% vs 25.0%), reflecting their underlying pathophysiology. Previous surgical history showed no significant difference between groups (18.8% vs 15.6%, p=0.741).

Table 1: Baseline Demographic Characteristics.

Characteristic	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
Age (years)	29.66 ± 2.78	30.47 ± 3.01	0.267
Height (inches)	4.38 ± 0.43	4.47 ± 0.46	0.416
Weight (kg)	70.16 ± 2.95	69.75 ± 5.34	0.712
ASA Status			<0.001*
ASA I	0 (0%)	8 (25.0%)	
ASA II	14 (43.8%)	24 (75.0%)	
ASA III	18 (56.3%)	0 (0%)	
Previous Surgery	6 (18.8%)	5 (15.6%)	0.741

*Values are mean ± SD or n (%). *Statistically significant (p<0.05). ASA = American Society of Anesthesiologists physical status classification.*

Baseline hemodynamic parameters revealed significant differences between groups (Table 2). Preeclamptic patients demonstrated markedly elevated blood pressure measurements: SBP (162.53±14.65 vs 134.47±6.41 mmHg, p<0.001), DBP (104.09±14.65 vs 82.72±9.06 mmHg, p<0.001), and MAP (123.57±13.98 vs 99.98±7.29 mmHg, p<0.001). Paradoxically, baseline heart rate was significantly lower in preeclamptic patients (99.34±13.92 vs 107.56±15.00 bpm, p=0.025), while oxygen saturation showed no significant difference (96.72±1.73 vs 96.94±1.12%, p=0.551). These baseline differences established the hemodynamic foundation from which subsequent responses to spinal anesthesia were measured.

Table 2: Baseline Hemodynamic Parameters.

Parameter	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
SBP (mmHg)	162.53 ± 14.65	134.47 ± 6.41	<0.001*
DBP (mmHg)	104.09 ± 14.65	82.72 ± 9.06	<0.001*
MAP (mmHg)	123.57 ± 13.98	99.98 ± 7.29	<0.001*
Heart Rate (bpm)	99.34 ± 13.92	107.56 ± 15.00	0.025*
SpO_2 (%)	96.72 ± 1.73	96.94 ± 1.12	0.551

*Values are mean ± SD. *Statistically significant (p<0.05). SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MAP = Mean Arterial Pressure; SpO_2 = Peripheral Oxygen Saturation.*

Temporal analysis of systolic blood pressure (Table 3) demonstrated striking differences in response patterns. Following spinal anesthesia administration, both groups showed immediate blood pressure reductions, but the magnitude and temporal progression differed markedly. Preeclamptic patients experienced a rapid decline from baseline 162.53±14.65 mmHg to 84.97±13.65 mmHg at 25 minutes (47.7% reduction), achieving their nadir significantly later than normotensive patients who reached their lowest SBP of 113.41±9.68 mmHg at 5 minutes (15.7% reduction). The recovery phase demonstrated equally distinctive patterns, with preeclamptic patients showing rapid blood pressure restoration

beginning at 30 minutes and ultimately exceeding normotensive values by 120 minutes (136.41±4.89 vs 125.91±7.51 mmHg, p<0.001).

Table 3: Systolic Blood Pressure Changes Over Time.

Time Point	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
Baseline	162.53 ± 14.65	134.47 ± 6.41	<0.001*
3 minutes	123.28 ± 9.97	117.09 ± 6.05	0.004*
5 minutes	113.28 ± 9.25	113.41 ± 9.68	0.956
10 minutes	102.53 ± 8.29	116.94 ± 12.71	<0.001*
15 minutes	93.84 ± 11.45	114.50 ± 16.18	<0.001*
20 minutes	87.00 ± 13.11	122.31 ± 18.87	<0.001*
25 minutes	84.97 ± 13.65	122.56 ± 22.10	<0.001*
30 minutes	91.34 ± 16.77	118.50 ± 16.08	<0.001*
45 minutes	101.75 ± 16.67	118.31 ± 12.33	<0.001*
60 minutes	113.78 ± 14.64	120.31 ± 10.80	0.043*
90 minutes	125.50 ± 5.58	121.88 ± 9.06	0.050
120 minutes	136.41 ± 4.89	125.91 ± 7.51	<0.001*

*Values are mean ± SD in mmHg. *Statistically significant (p<0.05). Highlighted rows indicate baseline values and nadir points for each group.*

Diastolic blood pressure patterns (Table 4) paralleled systolic findings but with even more pronounced percentage reductions in the preeclamptic group. Starting from significantly elevated baseline values (104.09±14.65 vs 82.72±9.06 mmHg, p<0.001), preeclamptic patients demonstrated progressive DBP decline reaching a nadir of 50.69±10.02 mmHg at 25 minutes, representing a 51.3% decrease from baseline. In contrast, normotensive patients achieved their lowest DBP of 62.16±8.71 mmHg at 5 minutes, representing only a 24.6% reduction. The temporal divergence continued throughout the study period, with preeclamptic patients eventually achieving DBP values exceeding normotensive levels by 120 minutes (77.56±11.93 vs 71.63±8.52 mmHg, p=0.025).

Table 4: Diastolic Blood Pressure Changes Over Time.

Time Point	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
Baseline	104.09 ± 14.65	82.72 ± 9.06	<0.001*
3 minutes	69.81 ± 14.07	69.59 ± 8.31	0.940
5 minutes	62.16 ± 9.98	67.03 ± 8.71	0.037*
10 minutes	55.00 ± 9.72	68.34 ± 9.28	<0.001*
15 minutes	51.50 ± 8.42	63.75 ± 10.91	<0.001*
20 minutes	50.84 ± 8.97	69.84 ± 16.36	<0.001*
25 minutes	50.69 ± 10.02	70.00 ± 23.61	<0.001*
30 minutes	53.00 ± 9.07	64.53 ± 13.88	<0.001*
45 minutes	59.06 ± 11.33	62.41 ± 10.37	0.222
60 minutes	64.94 ± 11.12	65.19 ± 10.89	0.927
90 minutes	69.91 ± 11.37	66.69 ± 9.50	0.219
120 minutes	77.56 ± 11.93	71.63 ± 8.52	0.025*

*Values are mean ± SD in mmHg. *Statistically significant (p<0.05). Highlighted rows indicate baseline values and nadir points for each group.*

Mean arterial pressure analysis (Table 5) provided integrated assessment of perfusion pressure dynamics. Preeclamptic patients' MAP demonstrated precipitous decline from baseline 123.57±13.98 mmHg to nadir 61.94±9.51 mmHg at 25 minutes, representing a 49.9% reduction and falling below the theoretical cerebral autoregulation threshold. Normotensive patients showed more modest MAP reduction from 99.98±7.29 to 82.49±8.15 mmHg at 5 minutes (19.3% decrease), maintaining values within normal autoregulatory range throughout. The delayed nadir in preeclamptic patients (25 vs 5 minutes) and subsequent rapid recovery pattern characterized by MAP exceeding normotensive values by 120 minutes (97.18±8.49 vs 89.72±7.13 mmHg, $p<0.001$) illustrated the complex and dynamic hemodynamic alterations unique to this population.

Table 5: Mean Arterial Pressure Changes Over Time.

Time Point	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
Baseline	123.57 ± 13.98	99.98 ± 7.29	<0.001*
3 minutes	87.97 ± 11.31	85.43 ± 6.83	0.272
5 minutes	79.20 ± 9.40	82.49 ± 8.15	0.135
10 minutes	70.84 ± 8.25	84.54 ± 9.63	<0.001*
15 minutes	65.59 ± 8.13	80.67 ± 11.72	<0.001*
20 minutes	62.91 ± 9.50	87.33 ± 16.77	<0.001*
25 minutes	61.94 ± 9.51	87.52 ± 22.74	<0.001*
30 minutes	65.78 ± 11.38	82.52 ± 13.94	<0.001*
45 minutes	73.29 ± 12.94	81.04 ± 10.35	0.009*
60 minutes	81.21 ± 10.93	83.56 ± 9.19	0.359
90 minutes	88.59 ± 8.74	85.08 ± 8.34	0.098
120 minutes	97.18 ± 8.49	89.72 ± 7.13	<0.001*

*Values are mean ± SD in mmHg. *Statistically significant ($p<0.05$). Highlighted rows indicate baseline values and nadir points. Note the MAP nadir in preeclamptic group (61.94 mmHg) falls below the theoretical cerebral autoregulation threshold of 65 mmHg.*

Heart rate responses (Table 6) revealed interesting compensatory mechanisms. Preeclamptic patients entered the study period with significantly lower baseline heart rates (99.34±13.92 vs 107.56±15.00 bpm, $p=0.025$), likely reflecting increased vagal tone or beta-blocker effects from antihypertensive medications. Following spinal anesthesia, both groups demonstrated relatively stable heart rate patterns with minimal variation from baseline throughout the 120-minute observation period. No significant differences emerged between groups at most time points, and neither group developed clinically significant bradycardia requiring intervention. This relative heart rate stability, despite profound blood pressure changes in preeclamptic patients, suggests limited compensatory chronotropic responses to hypotension.

Table 6: Heart Rate Changes Over Time.

Time Point	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
Baseline	99.34 ± 13.92	107.56 ± 15.00	0.025*
3 minutes	97.34 ± 17.60	105.94 ± 17.97	0.053
5 minutes	97.72 ± 15.28	103.44 ± 21.83	0.225
10 minutes	97.34 ± 16.37	97.03 ± 17.72	0.943
15 minutes	93.47 ± 13.43	95.31 ± 17.33	0.641
20 minutes	92.53 ± 13.38	92.47 ± 14.53	0.985

25 minutes	95.53 ± 15.82	92.69 ± 14.48	0.448
30 minutes	95.78 ± 12.45	97.94 ± 17.77	0.572
45 minutes	94.16 ± 14.80	99.38 ± 17.23	0.187
60 minutes	94.88 ± 14.92	96.97 ± 16.36	0.592
90 minutes	90.75 ± 14.86	94.00 ± 15.54	0.396
120 minutes	92.28 ± 12.43	96.09 ± 13.40	0.241

*Values are mean ± SD in beats per minute. *Statistically significant ($p<0.05$). Highlighted row indicates baseline value showing significantly lower heart rate in preeclamptic group.*

Oxygen saturation monitoring (Table 7) demonstrated reassuring stability in both groups throughout the observation period. Neither group showed significant baseline differences (96.72±1.73 vs 96.94±1.12%, p=0.551), and both maintained SpO_2 values above 97% for the majority of the study period. The slight initial improvement in oxygen saturation following spinal anesthesia, peaking at approximately 98.5-98.8% in both groups between 5-30 minutes, likely reflects supplemental oxygen administration and reduced metabolic demands during the procedure. By 120 minutes, both groups demonstrated minimal decline to approximately 97.5%, remaining well within normal physiological ranges and indicating adequate oxygenation despite the significant hemodynamic perturbations observed in preeclamptic patients.

Table 7: Peripheral Oxygen Saturation (SpO_2) Changes Over Time.

Time Point	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
Baseline	96.72 ± 1.73	96.94 ± 1.12	0.551
3 minutes	98.47 ± 1.11	98.22 ± 0.93	0.325
5 minutes	98.44 ± 1.22	98.50 ± 0.90	0.816
10 minutes	98.38 ± 1.31	98.47 ± 1.09	0.758
15 minutes	98.41 ± 1.27	98.66 ± 1.27	0.434
20 minutes	98.66 ± 1.00	98.75 ± 1.17	0.734
25 minutes	98.38 ± 1.10	98.81 ± 1.31	0.149
30 minutes	98.59 ± 0.80	98.78 ± 1.14	0.437
45 minutes	98.75 ± 0.80	98.56 ± 1.12	0.429
60 minutes	98.41 ± 1.24	98.13 ± 1.36	0.389
90 minutes	97.91 ± 1.40	97.78 ± 1.34	0.707
120 minutes	97.56 ± 0.88	97.84 ± 1.00	0.241

Values are mean ± SD in percentage. No statistically significant differences observed at any time point (all $p>0.05$).

Vasopressor requirements (Table 8) represented perhaps the most clinically significant finding, demonstrating substantially increased intervention needs in preeclamptic patients despite their elevated baseline vascular tone. Overall, 27 of 32 preeclamptic patients (84.4%) required vasopressor administration compared to only 6 of 32 normotensive patients (18.8%, p<0.001). First-dose vasopressor requirements affected 21 preeclamptic patients (65.6%) versus 5 normotensive patients (15.6%, p<0.001), indicating primary hypotensive events. Second-dose requirements, suggesting more refractory hypotension, occurred in 6 preeclamptic patients (18.8%) compared to only 1 normotensive patient (3.1%, p=0.046). These findings challenge the traditional assumption that increased vascular tone in preeclampsia provides protection against spinal-induced hypotension and underscore the need for anticipatory vasopressor strategies in this population.

Table 8: Vasopressor Requirements.

Vasopressor Requirement	Pre-eclamptic Group (n=32)	Normotensive Group (n=32)	p-value
First dose required	21 (65.6%)	5 (15.6%)	<0.001*
Second dose required	6 (18.8%)	1 (3.1%)	0.046*
Total requiring vasopressors	27 (84.4%)	6 (18.8%)	<0.001*
No vasopressor needed	5 (15.6%)	26 (81.2%)	<0.001*

*Values are n (%). *Statistically significant (p<0.05). Vasopressor = phenylephrine 100 µg IV bolus administered for hypotension (SBP <100 mmHg or >20% decrease from baseline). Highlighted rows show significant differences between groups.*

DISCUSSION

This comprehensive comparative analysis provides detailed quantification of hemodynamic responses to spinal anesthesia in preeclamptic versus normotensive parturients, revealing several clinically important findings that challenge conventional assumptions and inform evidence-based management strategies. The demographic homogeneity between groups, with no significant differences in age, height, or weight, strengthens the validity of our hemodynamic comparisons by minimizing confounding variables [9]. The significantly different ASA status distribution (p<0.001) appropriately reflects the increased physiological derangement in preeclamptic patients and represents expected rather than confounding variation [10,11].

The baseline hemodynamic findings corroborate established pathophysiological understanding of preeclampsia, with significantly elevated blood pressure reflecting increased systemic vascular resistance and endothelial dysfunction characteristic of the condition [7,11,12]. The paradoxically lower baseline heart rate in preeclamptic patients (99.34±13.92 vs 107.56±15.00 bpm, p=0.025) merits consideration, potentially reflecting antihypertensive medication effects, particularly beta-blockers or alpha-methyldopa, or representing compensatory baroreflex responses to chronic hypertension [7,12]. This baseline bradycardia may have limited subsequent compensatory chronotropic responses to hypotension, contributing to the more profound blood pressure reductions observed.

The most striking finding—more pronounced blood pressure reductions in preeclamptic patients (SBP decrease 47.7% vs 15.7%, DBP decrease 51.3% vs 24.6%, MAP decrease 49.9% vs 19.3%)—directly contradicts traditional teaching that elevated vascular tone protects against spinal-induced hypotension [13,14,15]. Several mechanisms may explain this paradox. First, the higher baseline pressures create a 'ceiling effect' whereby percentage decreases naturally appear larger even if absolute resistance changes are similar. Second, preeclamptic patients demonstrate reduced plasma volumes and decreased venous capacitance, potentially exacerbating the venous pooling effects of sympathetic blockade [7,16]. Third, endothelial dysfunction may impair compensatory vasoconstrictor responses in unblocked vascular beds, limiting the body's ability to maintain blood pressure through peripheral compensation [17].

The delayed occurrence of maximum hypotension in preeclamptic patients (25 minutes vs 5 minutes) represents a novel and clinically significant finding with important monitoring implications [18]. Several physiological mechanisms may contribute to this temporal pattern. The reduced cardiac output characteristic of preeclampsia may delay cephalad spread of local anesthetic and consequently delay peak sympathetic blockade [19]. Additionally, the increased vascular

resistance may initially buffer blood pressure decline, with decompensation occurring only after more complete sympathetic blockade develops or as compensatory mechanisms become exhausted [20]. This delayed nadir emphasizes the importance of extended vigilance periods beyond the immediate post-spinal phase, as traditional monitoring protocols often relax after the first 10-15 minutes when normotensive patients typically stabilize [21].

Our findings have important clinical implications for anesthetic management. The delayed occurrence of maximum hypotension emphasizes the need for extended monitoring periods, particularly during the 15-30 minute period post-spinal anesthesia when preeclamptic patients demonstrate maximum blood pressure reduction. Continuous or frequent blood pressure measurements should be maintained throughout this critical period rather than relaxing vigilance after the initial post-spinal phase. Anesthesiologists should be prepared for more aggressive vasopressor intervention in preeclamptic patients, with consideration for prophylactic vasopressor infusions as recommended by McDonnell et al., rather than waiting for established hypotension to develop [22]. The choice of vasopressor may also require careful consideration, with phenylephrine remaining the first-line agent but with potential modifications in dosing strategies for preeclamptic patients. The rapid blood pressure recovery and potential overshoot highlight the importance of continued monitoring during the recovery period to manage potential hypertensive episodes that could precipitate cerebrovascular complications in susceptible preeclamptic patients [23]. Fluid management strategies may also need individualization, balancing the need to maintain adequate preload against the risks of pulmonary edema in patients with compromised endothelial function and potential diastolic dysfunction. These considerations underscore the complexity of anesthetic management in preeclamptic patients and the need for experienced practitioners and comprehensive monitoring capabilities.

Study limitations include the observational design precluding definitive causality conclusions, use of non-invasive monitoring potentially missing beat-to-beat variations, lack of subclassification within the preeclamptic group by disease severity, standardized rather than individualized fluid management protocols, and focus on maternal hemodynamics without comprehensive fetal outcome assessment. Advanced monitoring techniques such as continuous arterial pressure measurement, transthoracic echocardiography, or cardiac output assessment using techniques like bioreactance or pulse contour analysis, as demonstrated by Dennis et al., might provide deeper insights into cardiovascular adaptations and underlying mechanisms [24]. Future research should prioritize preventing spinal hypotension through optimized protocols, as emphasized in recent systematic reviews [25], while considering stratification of preeclamptic patients by severity, utilizing advanced hemodynamic monitoring, comparing different vasopressor regimens, and examining long-term maternal and neonatal outcomes to establish comprehensive evidence-based management guidelines for this high-risk population.

CONCLUSION

This study demonstrates that preeclamptic patients exhibit fundamentally different hemodynamic responses to spinal anesthesia compared to normotensive parturients, characterized by more profound blood pressure reductions despite elevated baseline vascular tone, delayed occurrence of maximum hypotension at 25 minutes versus 5 minutes, rapid recovery with potential overshoot, and dramatically increased vasopressor requirements (84.4% vs 18.8%). These findings challenge traditional assumptions about protective effects of increased vascular resistance and highlight

the need for individualized anesthetic management strategies including extended hemodynamic monitoring through the critical 15-30 minute post-spinal period, anticipatory rather than reactive vasopressor administration, and continued vigilance during the recovery phase to prevent hypertensive complications. Understanding these distinct hemodynamic patterns enables anesthesiologists to optimize maternal safety and outcomes during cesarean delivery in this high-risk population through evidence-based, proactive management approaches.

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