

Original Article

Factors Influencing Bradycardia during Spinal Anesthesia in Obstetric Patients Undergoing Cesarean Section

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ABSTRACT

Background: Spinal anaesthesia is widely used for caesarean delivery, but sympathetic blockade can produce hypotension and maternal bradycardia. Recognition of associated factors is important for timely intraoperative monitoring and management. **Objective:** To determine the frequency, clinical characteristics, and factors associated with bradycardia during spinal anaesthesia for caesarean section. **Methods:** A hospital-based observational study included 138 women aged 18–40 years undergoing elective or emergency caesarean delivery under spinal anaesthesia at Maqsood Medical Complex Hospital and Hayatabad Medical Complex, Peshawar. Bradycardia was defined as heart rate <60 beats/min, while hypotension was defined as a >30% reduction in systolic blood pressure from baseline. **Results:** Bradycardia occurred in 26/138 patients (18.8%; 95% CI 13.2–26.2%) and hypotension in 38/138 (27.5%). Most bradycardic episodes occurred 6–15 minutes after spinal anaesthesia. Atropine was administered in 15/26 cases (57.7%). High sensory block level was associated with bradycardia, $\chi^2=9.84$, $p=.043$, and hypotension was also associated with bradycardia, $\chi^2=5.31$, $p=.021$. In exploratory multivariable analysis, a high block level (T4–T6 versus $\geq T7$) was associated with greater odds of bradycardia (OR 2.31, 95% CI 1.08–5.21; $p=.031$), as was hypotension (OR 3.44, 95% CI 1.37–8.62; $p=.009$). **Conclusion:** Approximately one in five women developed bradycardia. Higher sensory block and concurrent hypotension were the principal factors associated with bradycardia in this cohort. **Keywords:** Bradycardia; Spinal Anaesthesia; Caesarean Section; Hypotension; Obstetric Anaesthesia; Sensory Block.

INTRODUCTION

Spinal anaesthesia is commonly used for caesarean delivery because it provides rapid and effective neuraxial blockade while allowing the mother to remain awake and avoiding many of the airway and systemic anaesthetic risks associated with general anaesthesia. However, spinal anaesthesia produces sympathetic blockade, resulting in peripheral vasodilatation, reduced systemic vascular resistance, redistribution of blood volume, and decreased venous return. Consequently, maternal hypotension remains one of the most frequent physiological disturbances after spinal anaesthesia for caesarean delivery and requires active prevention, monitoring, and treatment (1,2).

Alterations in maternal heart rate may occur concurrently with these haemodynamic changes. Bradycardia after spinal anaesthesia has been reported with varying frequency, reflecting differences in study populations, definitions, intrathecal drug regimens, block characteristics, baseline physiology, and haemodynamic-management protocols. Previous observational studies have identified associations between spinal anaesthesia-related haemodynamic disturbances and factors including sensory block height, anaesthetic dose, baseline cardiovascular characteristics, hypotension, and procedural variables (3–7). In obstetric populations specifically, both hypotension and bradycardia have been documented following spinal anaesthesia for caesarean delivery, emphasizing the importance of continuous haemodynamic surveillance during the period immediately following intrathecal injection (3–5).

The mechanisms underlying bradycardia during spinal anaesthesia are multifactorial. Extensive sympathetic blockade may reduce venous return and cardiac filling, while blockade involving higher thoracic levels can alter sympathetic cardiac acceleration. Reduced preload may also enhance vagal predominance and activate cardioinhibitory reflex pathways. These mechanisms can become clinically important when marked bradycardia accompanies hypotension or a rapidly ascending neuraxial block (5,6,8). Higher sensory block levels have therefore received particular attention as potentially modifiable or clinically recognizable correlates of haemodynamic instability during spinal anaesthesia (6,7).

Vasopressor therapy can further influence maternal heart rate. Phenylephrine is an effective α -adrenergic vasopressor for prevention and treatment of post-spinal hypotension but can produce baroreceptor-mediated reflex bradycardia. Norepinephrine combines potent α -adrenergic activity with modest β -adrenergic activity and has consequently been investigated as an alternative capable of maintaining arterial pressure with less reduction in heart rate and cardiac output (9–11). Randomized evidence nevertheless indicates that differences in bradycardia depend partly on dose and mode of administration, with intermittent bolus comparisons not invariably showing a statistically significant advantage for norepinephrine (12). Clinically important bradycardia has also been reported during phenylephrine treatment, particularly when the pretreatment heart rate is already relatively low (13). International and regional consensus guidance therefore emphasizes proactive haemodynamic management, appropriate vasopressor use, and close maternal monitoring during spinal anaesthesia for caesarean delivery (2,14).

The clinical significance of maternal bradycardia depends on its severity, duration, associated arterial pressure, maternal symptoms, sensory block level, and response to treatment. Identification of factors associated with bradycardia may therefore assist anaesthesia teams in recognizing patients who require particularly close surveillance and early haemodynamic intervention.

The present study evaluated the frequency and clinical characteristics of bradycardia among women receiving spinal anaesthesia for caesarean delivery and explored its associations with sensory block level, anaesthetic solution, hypotension, age, bupivacaine dose, and estimated blood loss.

MATERIALS AND METHODS

A hospital-based observational analytic study was conducted over four months at Maqsood Medical Complex Hospital and Hayatabad Medical Complex, Peshawar. A total of 138 obstetric patients undergoing elective or emergency caesarean section under spinal anaesthesia were recruited using convenience sampling.

Women aged 18–40 years with American Society of Anesthesiologists physical status I or II who provided consent were eligible for inclusion. Patients with known cardiac disease, use of medication affecting heart rate, contraindications to spinal anaesthesia, combined spinal-epidural or general anaesthesia, and incomplete records were excluded.

Data were recorded using a structured proforma. Recorded variables included type of caesarean section, spinal anaesthetic preparation, highest sensory block level, bupivacaine dose, heart rate, blood pressure, onset and severity of bradycardia, associated hypotension, estimated blood loss, intravenous-fluid administration, treatment of bradycardia, maternal symptoms, 5-minute Apgar score, and neonatal intensive-care unit admission.

Bradycardia was defined as a heart rate <60 beats/min. Hypotension was defined as a reduction in systolic blood pressure of >30% from baseline. These thresholds are consistent with definitions used in previous investigations of spinal anaesthesia-associated haemodynamic complications and facilitate comparison with earlier studies (3,6).

Data were analyzed using SPSS version 29. Categorical associations were evaluated using chi-square testing, continuous variables using independent-samples t tests, and exploratory multivariable associations using logistic regression. Statistical significance was defined as $p < 0.05$.

Ethical approval was obtained from the Institutional Review Board of Superior University, Lahore, Pakistan. Participant consent was obtained.

RESULTS

Procedural Characteristics

A total of 138 women undergoing caesarean delivery under spinal anaesthesia were included. Ninety caesarean sections were elective and 48 were emergency procedures. Hyperbaric bupivacaine was used in 81 patients and isobaric bupivacaine in 57. The highest sensory block ranged from T4 to T8, with T6 being the most frequently recorded level.

Table 1. Obstetric and Spinal-Anaesthesia Characteristics

Variable	Category	n (%)
Caesarean section	Elective	90 (65.2)
	Emergency	48 (34.8)
Bupivacaine preparation	Hyperbaric	81 (58.7)
	Isobaric	57 (41.3)
Highest sensory block	T4	28 (20.3)
	T5	31 (22.5)
	T6	42 (30.4)
	T7	25 (18.1)
	T8	12 (8.7)
Estimated blood loss	<400 mL	17 (12.3)
	400–599 mL	43 (31.2)
	600–799 mL	52 (37.7)
	≥800 mL	26 (18.8)
IV fluid	<1000 mL	21 (15.2)
	1000–1249 mL	35 (25.4)
	1250–1499 mL	42 (30.4)
	≥1500 mL	40 (29.0)

Twenty-six women developed bradycardia, corresponding to 18.8% of the cohort. The Wilson 95% confidence interval was approximately 13.2–26.2%. Three of the 26 patients with bradycardia had a minimum heart rate of 35–39 beats/min, while most episodes began between 6 and 15 minutes after spinal anaesthesia.

Table 2. Characteristics and Management of Bradycardia

Variable	Category	n (%) among bradycardia cases
Minimum heart rate	35–39 bpm	3 (11.5)
	40–44 bpm	5 (19.2)
	45–49 bpm	7 (26.9)
	50–54 bpm	6 (23.1)
	55–59 bpm	5 (19.2)
Time to onset	≤5 min	2 (7.7)
	6–10 min	10 (38.5)
	11–15 min	9 (34.6)
	>15 min	5 (19.2)
Treatment	Atropine	15 (57.7)
	Ephedrine	7 (26.9)
	Epinephrine	2 (7.7)
	None required	2 (7.7)
Associated symptom	Dizziness	10 (38.5)
	Nausea	8 (30.8)
	Shortness of breath	5 (19.2)
	Chest discomfort	2 (7.7)
	No symptom	1 (3.8)

Hypotension occurred in 38/138 women, corresponding to 27.5% of the cohort (95% CI approximately 20.8–35.5%).

Neonatal Outcomes

Overall neonatal outcomes were generally favourable. A 5-minute Apgar score of 9–10 was recorded in 107 neonates, while five neonates were admitted to the neonatal intensive-care unit.

Table 3. Five-Minute Apgar and NICU Admission

Outcome	Category	n (%)
Apgar score	<7	2 (1.4)
	7–8	29 (21.0)
	9–10	107 (77.5)
NICU admission	Yes	5 (3.6)
	No	133 (96.4)

Neonatal outcomes describe the overall cohort and were not compared according to maternal bradycardia status.

Factors Associated With Bradycardia

Sensory-block level was significantly associated with bradycardia, whereas anaesthetic solution was not. Concurrent hypotension was also significantly associated with bradycardia. Age, bupivacaine dose, and estimated blood loss did not demonstrate statistically significant differences between patients with and without bradycardia.

Table 4. Bivariate Associations With Bradycardia

Variable	Test Statistic	p
Sensory block level T4–T8	$\chi^2=9.84$	.043
Hyperbaric vs isobaric bupivacaine	$\chi^2=1.27$	.259
Associated hypotension	$\chi^2=5.31$	.021
Age	t=0.78	.437
Bupivacaine dose	t=1.12	.264
Estimated blood loss	t=0.94	.352

Mean age was 27.9 ± 6.8 years in women with bradycardia and 28.5 ± 6.5 years in those without bradycardia. Mean bupivacaine dose was 10.1 ± 1.2 mg versus 9.9 ± 1.3 mg, respectively, while estimated blood loss was 565 ± 135 mL versus 548 ± 120 mL.

Exploratory Logistic Regression

In exploratory multivariable analysis, a high sensory block defined as T4–T6 compared with $\geq T7$ was associated with greater odds of bradycardia. Concurrent hypotension was also associated with increased odds of bradycardia. Anaesthetic solution, age, and bupivacaine dose were not statistically significant in the model.

Table 5. Multivariable Associations With Bradycardia

Variable	OR	95% CI	p
High sensory block, T4–T6 vs $\geq T7$	2.31	1.08–5.21	.031
Associated hypotension	3.44	1.37–8.62	.009
Isobaric vs hyperbaric bupivacaine	1.26	0.58–2.72	.552
Age, per year	0.97	0.91–1.04	.423
Bupivacaine dose	1.18	0.79–1.76	.391

The model was considered exploratory because only 26 bradycardia events were available for five modeled predictors.

DISCUSSION

Bradycardia occurred in 18.8% of women undergoing caesarean delivery under spinal anaesthesia in the present study. Most episodes occurred within the first 15 minutes after spinal anaesthesia, and atropine was the most frequently administered treatment. Higher sensory block and concurrent hypotension were the principal factors associated with bradycardia. In contrast, anaesthetic solution, age, bupivacaine dose, and estimated blood loss were not significantly associated with the outcome.

The observed frequency should be interpreted in the context of substantial variation across previous obstetric studies. Somboonviboon et al. reported bradycardia in 2.5% of 722 parturients undergoing caesarean section under spinal anaesthesia, whereas a more recent retrospective cohort reported bradycardia in 15.1% of women undergoing elective caesarean delivery (3,4). Dev et al. also found sinus bradycardia to be the most common arrhythmia observed during caesarean section under spinal

anaesthesia and reported associations between intraoperative arrhythmia, hypotension, and higher block levels (5). Differences among studies are plausibly related to patient selection, intrathecal regimens, definitions of bradycardia, block characteristics, vasopressor use, and haemodynamic-management protocols rather than representing a single universal incidence.

High sensory block was independently associated with bradycardia in the present exploratory model. This finding is physiologically and clinically plausible because more extensive sympathetic blockade can reduce vascular tone and venous return while altering sympathetic cardiac acceleration. Previous spinal-anaesthesia studies have similarly identified high sensory levels as correlates of haemodynamic instability, including bradycardia and hypotension (5–7). Reduced cardiac filling may further increase vagal predominance and contribute to cardioinhibitory reflex responses, particularly when the reduction in preload is abrupt or accompanied by marked hypotension (8). The present findings therefore support careful monitoring of block progression rather than implying that block height alone directly causes every episode of bradycardia.

Concurrent hypotension was associated with approximately 3.4-fold greater odds of bradycardia. Both phenomena may originate from the same underlying neuraxial haemodynamic disturbance: sympathetic blockade reduces systemic vascular resistance and venous return, while sufficiently marked reductions in preload may alter heart-rate responses and favour bradycardia (1,2,8). Hypotension should consequently be interpreted in this observational study as an important concurrent clinical correlate rather than as a proven independent upstream cause of bradycardia.

The temporal distribution of events is also clinically relevant. Approximately three-quarters of bradycardic episodes occurred between 6 and 15 minutes after spinal anaesthesia. This corresponds to the period during which the neuraxial block is becoming established and maternal haemodynamic changes can evolve rapidly. Only three women had a recorded minimum heart rate below 40 beats/min, but the occurrence of severe bradycardia in even a small proportion reinforces the importance of continuous heart-rate and blood-pressure monitoring during the early post-spinal period. When bradycardia occurs with hypotension or evidence of reduced preload, correction of the associated haemodynamic disturbance and prompt treatment of clinically important bradycardia are central management principles (2,8).

Atropine was administered in 57.7% of bradycardic cases, while ephedrine and epinephrine were used less frequently. Interpretation of these treatment frequencies is limited because treatment decisions depend on heart rate, blood pressure, symptoms, vasopressor exposure, clinician judgement, and the severity and duration of the event. Phenylephrine deserves particular consideration because it can increase arterial pressure while simultaneously producing baroreceptor-mediated reflex slowing of the heart rate. In a randomized comparison, norepinephrine maintained maternal blood pressure with higher heart rate and cardiac output and less bradycardia than phenylephrine (9). A subsequent randomized trial using fixed-rate infusions similarly reported less bradycardia and less atropine requirement with norepinephrine (10), while systematic-review evidence has generally demonstrated a lower incidence of bradycardia with norepinephrine than with phenylephrine (11).

The relationship between vasopressor choice and maternal bradycardia is nevertheless dependent on administration strategy and dose. In a randomized trial evaluating intermittent treatment boluses for established post-spinal hypotension, de Queiroz et al. observed no statistically significant difference in bradycardia between norepinephrine and phenylephrine groups (12). Phenylephrine-associated clinically important bradycardia has also been related to a lower heart rate immediately before vasopressor administration (13). Additional systematic reviews and network meta-analyses generally support norepinephrine as an effective alternative to phenylephrine with a more favourable maternal heart-rate profile, although haemodynamic outcomes vary according to dose, bolus versus infusion administration, and study protocol (15–18). Contemporary evidence therefore supports individualized haemodynamic management rather than interpreting vasopressor identity independently of dose and method of administration.

Current consensus guidance emphasizes proactive prevention and treatment of maternal hypotension during spinal anaesthesia, with vasopressor therapy forming the principal component of haemodynamic management (2,14). Fluid administration is another relevant determinant of venous return, but the evidence indicates that fluid loading should generally complement rather than replace vasopressor-based prevention. Network meta-analysis has demonstrated differences among crystalloid and colloid preload and coload strategies while also emphasizing that many historical studies did not incorporate contemporary prophylactic vasopressor practice (19). Earlier meta-analytic evidence found little

difference between crystalloid preload and coload in hypotension incidence (20), while comparisons of colloid and crystalloid strategies have suggested lower hypotension rates with some colloid regimens (21). The present study recorded intravenous-fluid administration but did not provide sufficiently detailed fluid timing or strategy for evaluating its relationship with bradycardia.

Anticholinergic prophylaxis has also been investigated as a means of modifying haemodynamic responses. In a randomized double-blind trial of non-elective caesarean sections, prophylactic glycopyrrolate did not significantly reduce vasopressor requirement and did not produce a statistically significant reduction in bradycardia or atropine requirement, although tachycardia was more frequent (22). These findings illustrate that prevention of bradycardia cannot be reduced to routine anticholinergic administration and remains closely linked to overall control of maternal haemodynamics.

Anaesthetic solution type was not significantly associated with bradycardia in the present study, and mean bupivacaine dose also did not differ significantly between groups. Although some previous studies have linked spinal anaesthetic dose or higher block level with haemodynamic complications, these relationships are not uniform across populations and protocols (4,5,7). The current results therefore do not provide evidence that hyperbaric or isobaric bupivacaine independently altered bradycardia risk in this cohort.

Age and estimated blood loss were similarly unassociated with bradycardia in the present analysis. These null findings should not be interpreted as evidence that these variables can never influence haemodynamic stability. Previous large spinal-anaesthesia cohorts have identified age among factors associated with bradycardia or hypotension in broader surgical populations (6), while blood loss can contribute to reduced circulating volume and hypotension in obstetric surgery (3). The absence of statistically significant associations in the current sample may reflect the characteristics of the relatively young obstetric population, the observed range of exposure, and limited statistical power.

Overall neonatal outcomes were favourable, with 77.5% of neonates having a 5-minute Apgar score of 9–10 and 3.6% requiring NICU admission. These findings cannot establish that maternal bradycardia was unrelated to neonatal status because neonatal outcomes were not stratified or modeled according to maternal bradycardia exposure. Contemporary vasopressor trials and meta-analyses generally report similar short-term neonatal outcomes between phenylephrine and norepinephrine when maternal blood pressure is appropriately maintained, although maternal heart-rate profiles may differ (9–12,15–18). Direct assessment of maternal bradycardia in relation to neonatal outcomes would require an analysis specifically designed for that purpose.

The exploratory multivariable model requires cautious interpretation. With 26 bradycardia events and five modeled predictors, the analysis contained approximately five outcome events per predictor. Simulation studies have demonstrated that sparse logistic-regression models can produce biased regression coefficients, unstable variance estimates, and poorly calibrated confidence intervals when the number of events is limited, although the traditional threshold of 10 events per predictor should not be regarded as an absolute rule applicable to every model (23,24). The reported odds ratios are therefore most appropriately interpreted as exploratory associations rather than precise estimates suitable for clinical prediction.

Larger studies with more outcome events would permit more stable multivariable adjustment for baseline heart rate, baseline blood pressure, emergency status, sensory block height, vasopressor type and dose, intravenous-fluid strategy, maternal position, intrathecal medication, bupivacaine dose, and blood loss. Prospective collection of these haemodynamic and treatment variables would also help distinguish bradycardia arising primarily in association with high neuraxial block from episodes associated with hypotension, pharmacological treatment, or other intraoperative stimuli.

CONCLUSION

Bradycardia occurred in 18.8% of obstetric patients undergoing caesarean delivery under spinal anaesthesia, with most episodes occurring during the first 15 minutes after spinal administration. Higher sensory block and concurrent hypotension were the principal factors associated with bradycardia in the exploratory analysis. Anaesthetic solution, age, bupivacaine dose, and estimated blood loss were not significantly associated with the outcome.

These findings reinforce the importance of close maternal heart-rate and blood-pressure monitoring during the early period following spinal anaesthesia, particularly when a high sensory block or hypotension develops. Because the multivariable analysis was based on a limited number of bradycardia

events, the observed associations should be confirmed in larger, prospectively characterized obstetric populations.

LIMITATIONS

The study used convenience sampling from two hospitals, which may limit generalizability. Only 26 bradycardia events were available for multivariable analysis, restricting the stability and complexity of adjusted modeling. Baseline heart rate and blood pressure were not incorporated into the reported risk model. Detailed information regarding vasopressor prophylaxis and treatment, intrathecal technique, patient positioning, fluid timing and strategy, and other haemodynamic interventions was incomplete, preventing assessment of their potential contribution to bradycardia.

The observational design establishes associations but cannot determine causality. Furthermore, neonatal outcomes were reported for the cohort as a whole and were not analyzed according to maternal bradycardia status, preventing conclusions regarding the relationship between maternal bradycardia and neonatal outcome.

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