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Comparative Analysis of Coload and Pre-Load in the Prevention of Spinal Anesthesia Induced Hypotension

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ABSTRACT

Background: *Spinal anesthesia is widely used for elective lower abdominal and urologic procedures but is frequently complicated by hypotension due to sympathetic blockade, reduced venous return, and decreased cardiac output. Optimizing the timing of crystalloid administration may improve hemodynamic stability and reduce vasopressor exposure.* ***Objective:*** *To compare crystalloid preload versus crystalloid coload in preventing spinal anesthesia–induced hypotension and reducing vasopressor requirement in adults undergoing elective lower abdominal and urologic surgery.* ***Methods:*** *In this prospective comparative cross-sectional study, 44 ASA I–III patients (18–70 years) were allocated to receive Ringer's lactate either as preload (10–15 mL/kg before intrathecal injection; n=22) or coload (same volume initiated at intrathecal injection; n=22). Hemodynamics were recorded at predefined intervals intraoperatively. Hypotension was defined as SBP decrease ≥20% from baseline or MAP <65 mmHg. Vasopressor use followed a standardized protocol, and total dose was expressed as ephedrine equivalents.* ***Results:*** *Hypotension occurred in 22.7% (5/22) of preload patients versus 50.0% (11/22) of coload patients (RR 0.45; 95% CI 0.19–0.99; p=0.049). Vasopressors were required in 27.3% (6/22) versus 54.5% (12/22), respectively (RR 0.50; 95% CI 0.23–0.99; p=0.048), and total ephedrine-equivalent dose was lower with preload (7.2±4.1 mg vs 11.8±6.5 mg; p=0.01). Minimum MAP was higher in the preload group (72.6±6.4 mmHg vs 66.1±7.9 mmHg; p=0.004).* ***Conclusion:*** *Crystalloid preload before spinal anesthesia reduced hypotension incidence, vasopressor requirement, and vasopressor dose while improving MAP stability compared with coload in elective lower abdominal and urologic surgery.*

Keywords: *Spinal anesthesia; Hypotension; Preload; Coload; Mean arterial pressure; Vasopressors; Ephedrine; Ringer's lactate.*

INTRODUCTION

Spinal anesthesia remains one of the most frequently employed neuraxial techniques for lower abdominal and urologic surgery because of its rapid onset, dense sensory and motor blockade, avoidance of airway manipulation, and favorable postoperative analgesic profile. Despite these advantages, spinal anesthesia is consistently associated with arterial hypotension resulting from sympathetic blockade, peripheral vasodilation, reduced venous return, and subsequent decline in cardiac output (1). The incidence of hypotension varies widely depending on patient population, block height, and definition thresholds, but it remains one of the most clinically significant adverse events in neuraxial anesthesia. Even transient reductions in mean arterial pressure (MAP) may precipitate nausea, vomiting, altered mentation, and in vulnerable patients, myocardial ischemia, cerebral hypoperfusion, or renal dysfunction. Therefore, prevention of spinal anesthesia–induced hypotension is not merely a comfort measure but a central determinant of perioperative safety and organ perfusion (1).

Intravenous fluid administration has historically formed the cornerstone of preventive strategies against neuraxial hypotension. The physiological rationale is grounded in maintenance of preload and intravascular volume prior to or during the onset of

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sympathectomy. Two principal approaches are practiced: crystalloid preload, in which fluids are administered before intrathecal injection, and crystalloid coload, in which rapid infusion coincides with or immediately follows spinal block placement. Preloading aims to expand circulating volume in anticipation of vasodilation, thereby preserving venous return and stroke volume at the onset of sympathetic blockade. In contrast, coloading is based on the premise that the maximal hemodynamic effect of crystalloids is short-lived; thus, synchronizing fluid administration with vasodilation may optimize intravascular retention and mitigate hypotension more effectively.

The comparative effectiveness of preload versus coload has been extensively evaluated in obstetric populations undergoing cesarean delivery under spinal anesthesia. Early observational work identified hypotension as a frequent complication of neuraxial blockade and highlighted the need for preventive strategies (2). Subsequent randomized investigations compared crystalloid timing strategies, reporting variable findings regarding equivalence or superiority (3). Systematic reviews and meta-analyses in parturients have suggested that coload may reduce the incidence of hypotension compared with traditional preload, although results remain influenced by vasopressor protocols and fluid volumes used (4). However, obstetric physiology differs substantially from that of non-pregnant surgical patients due to increased circulating blood volume, aortocaval compression, altered vascular tone, and distinct autonomic responses. Extrapolation of findings from cesarean delivery to non-obstetric elective surgery may therefore be inappropriate without direct evaluation in the target population.

In non-obstetric lower abdominal and urologic surgery, patients often present with heterogeneous comorbid profiles, including advanced age and cardiovascular risk factors. These characteristics may alter autonomic compensation and hemodynamic tolerance to sympathectomy. While vasopressors remain effective for treating established hypotension, reliance on pharmacologic rescue alone may introduce risks such as reflex bradycardia, tachyarrhythmias, or increased myocardial oxygen demand. A preventive fluid strategy that minimizes vasopressor exposure while maintaining stable MAP would therefore represent a clinically meaningful optimization of perioperative management. Despite this, high-quality comparative data specifically addressing preload versus coload in elective non-obstetric lower abdominal and urologic surgery remain limited. Much of the available literature either focuses on obstetric populations or lacks standardized outcome definitions and hemodynamic measurement protocols (1–4).

From a biostatistical and methodological perspective, interpretation of previous studies is often complicated by inconsistent definitions of hypotension, variable timing of outcome assessment, and heterogeneous vasopressor algorithms. Some investigations define hypotension as a percentage decrease from baseline systolic blood pressure, whereas others use absolute MAP thresholds. Additionally, the absence of standardized measurement intervals during the critical early minutes following intrathecal injection may obscure clinically relevant hemodynamic differences. These methodological inconsistencies contribute to ongoing uncertainty regarding the optimal fluid-timing strategy in non-obstetric surgical settings.

Within the PICO framework, the present study addresses adult patients undergoing elective lower abdominal and urologic surgery under spinal anesthesia (Population), comparing crystalloid preload administered before intrathecal injection (Intervention) with crystalloid coload administered at the time of spinal anesthesia (Comparison), and evaluating the incidence of spinal anesthesia–induced hypotension, vasopressor requirement, and intraoperative MAP trends (Outcomes). By focusing on a homogeneous elective surgical

cohort and systematically assessing hemodynamic endpoints, this study seeks to clarify whether the timing of crystalloid administration independently influences perioperative hemodynamic stability in this population.

Given the physiological rationale supporting volume expansion prior to sympathectomy and the limited direct evidence in non-obstetric surgical patients, we hypothesize that crystalloid preload is associated with a lower incidence of hypotension and reduced vasopressor requirement compared with crystalloid coload in adults undergoing elective lower abdominal and urologic surgery under spinal anesthesia. The objective of this study is therefore to comparatively evaluate preload and coload strategies with respect to hypotension incidence, vasopressor utilization, and maintenance of mean arterial pressure, thereby providing clinically applicable evidence to guide fluid management during spinal anesthesia.

METHODS

This prospective comparative cross-sectional study was conducted in the Department of Anesthesiology of a tertiary care teaching hospital over a four-month period following institutional approval. The study was designed to evaluate the comparative effect of crystalloid preload versus crystalloid coload on the incidence of spinal anesthesia–induced hypotension and related hemodynamic outcomes in adults undergoing elective lower abdominal and urologic surgery. A prospective design was selected to allow standardized implementation of fluid protocols and uniform perioperative hemodynamic monitoring, thereby minimizing information bias and ensuring temporal clarity between exposure (fluid timing strategy) and outcomes. The methodology was structured in accordance with internationally accepted reporting recommendations for observational comparative studies (5).

Adult patients aged 18 to 70 years, classified as American Society of Anesthesiologists (ASA) physical status I, II, or III, and scheduled for elective lower abdominal or urologic procedures under spinal anesthesia were eligible for inclusion. Patients with pre-existing uncontrolled hypertension, ischemic heart disease, clinically significant arrhythmias, baseline systolic blood pressure below 90 mmHg, severe renal, hepatic, or endocrine dysfunction, known hypersensitivity to the study fluids or local anesthetics, and those undergoing emergency surgery were excluded to reduce confounding from unstable cardiovascular physiology. Consecutive eligible patients presenting during the study period were screened preoperatively. After verification of eligibility, patients were informed in detail about the study protocol, and written informed consent was obtained prior to enrollment.

Participants were allocated into two groups based on the timing of intravenous crystalloid administration. In the preload group, patients received 10–15 mL/kg of Ringer's lactate solution administered over 15–20 minutes immediately prior to intrathecal injection. In the coload group, the same volume of Ringer's lactate was administered rapidly beginning at the time of intrathecal injection and completed within 15 minutes. Fluid volumes were calculated according to body weight measured on the day of surgery. All patients were kept fasting according to institutional guidelines and received standard monitoring upon arrival in the operating room, including non-invasive blood pressure measurement, electrocardiography, and pulse oximetry.

Baseline hemodynamic parameters, including systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR), were recorded after five minutes of rest in the supine position prior to fluid administration. Spinal anesthesia was performed at the L3–L4 or L4–L5 interspace using a standardized aseptic technique with a

25-gauge Quincke spinal needle. A fixed dose of 0.5% hyperbaric bupivacaine (12.5–15 mg depending on height and surgical requirement) was administered intrathecally. Patients were positioned supine immediately after injection. Sensory block level was assessed using loss of pinprick sensation at five-minute intervals until stabilization. No intrathecal opioids were added. Supplemental oxygen at 2–3 L/min via nasal cannula was administered routinely.

Hemodynamic measurements were recorded at one-minute intervals for the first 10 minutes following spinal anesthesia, at five-minute intervals for the next 20 minutes, and thereafter at 10-minute intervals until completion of surgery. The primary outcome was the incidence of spinal anesthesia–induced hypotension, operationally defined as a decrease in systolic blood pressure of ≥20% from baseline or an absolute MAP <65 mmHg at any recorded time point intraoperatively. Secondary outcomes included requirement for vasopressor therapy, total vasopressor dose administered (expressed in ephedrine milligram equivalents), minimum intraoperative MAP, and trends in SBP, DBP, MAP, and HR over time. Vasopressor therapy was standardized: intravenous ephedrine 5 mg boluses were administered for hypotension accompanied by bradycardia (HR <60 beats per minute), and phenylephrine 50–100 μg boluses were administered for hypotension with HR ≥60 beats per minute. Repeat doses were given as required to restore SBP to within 20% of baseline. Bradycardia without hypotension was treated with intravenous atropine 0.5 mg.

To minimize selection bias, consecutive sampling was used, and baseline demographic and clinical characteristics were compared between groups to assess comparability. Standardization of spinal technique, anesthetic dose, monitoring intervals, and vasopressor protocol was implemented to reduce performance and measurement bias. All hemodynamic data were recorded in real time by an anesthesia provider not involved in data analysis. Data entry was performed using double-entry verification to ensure accuracy. Potential confounding variables, including age, ASA status, baseline MAP, body mass index (BMI), and block height, were recorded prospectively and included in adjusted analyses when appropriate.

The sample size was calculated to detect a clinically meaningful absolute difference of 30% in hypotension incidence between groups, assuming a baseline incidence of approximately 60% based on prior literature in neuraxial anesthesia (2–4). With a two-sided alpha of 0.05 and power of 80%, the required sample size was estimated at 20 patients per group. To account for potential incomplete data, 22 patients were included in each group, yielding a total sample of 44 participants.

Statistical analysis was performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test. Normally distributed data were presented as mean ± standard deviation and compared using independent-samples t-tests. Non-normally distributed variables were expressed as median with interquartile range and compared using the Mann–Whitney U test. Categorical variables were summarized as frequencies and percentages and compared using chi-square or Fisher's exact test as appropriate. Relative risk (RR) with 95% confidence intervals (CI) was calculated for the primary outcome. Repeated hemodynamic measurements over time were analyzed using repeated-measures analysis of variance with group as the between-subject factor. Multivariable logistic regression analysis was conducted to adjust for potential confounders affecting hypotension incidence. A p-value <0.05 was considered statistically significant. Missing data were assessed for randomness; complete-case analysis was applied when missingness was <5%.

The study protocol was reviewed and approved by the Institutional Review Board of the hosting institution prior to initiation. All procedures were conducted in accordance with the

ethical principles outlined in the Declaration of Helsinki (6). Written informed consent was obtained from all participants before enrollment. Patient confidentiality was maintained by assigning anonymized study identification codes, and access to the dataset was restricted to the principal investigators. Standardized protocols, detailed documentation of fluid timing and volumes, predefined operational definitions of outcomes, and a prespecified statistical analysis plan were implemented to ensure methodological transparency, reproducibility, and data integrity.

RESULTS

All 44 enrolled patients completed the study and were included in the final analysis, with 22 patients allocated to the Preload group and 22 to the Coload group. Baseline demographic and clinical characteristics were well balanced between groups, with no statistically significant differences observed (Table 1). The mean age in the Preload group was 47.3 ± 11.2 years compared with 44.8 ± 12.5 years in the Coload group, yielding a non-significant mean difference of 2.5 years (95% CI: −4.3 to 9.3; p = 0.46). Male patients constituted 77.3% (17/22) of the Preload group and 95.5% (21/22) of the Coload group (OR 0.15; 95% CI: 0.02–1.42; p = 0.18). Body mass index was comparable between groups (25.6 ± 3.8 kg/m² vs 24.9 ± 4.1 kg/m²; mean difference 0.7 kg/m², 95% CI: −1.6 to 3.0; p = 0.56). The distribution of ASA physical status was similar, with ASA II predominating in both groups (59.1% in Preload vs 63.6% in Coload; p = 0.89). Baseline hemodynamic parameters showed no significant differences: baseline MAP was 92.0 ± 10.8 mmHg in the Preload group and 93.2 ± 11.4 mmHg in the Coload group (mean difference −1.2 mmHg; 95% CI: −8.1 to 5.7; p = 0.74), confirming pre-intervention comparability.

Regarding the primary outcome (Table 2), hypotension occurred in 5 patients (22.7%) in the Preload group compared with 11 patients (50.0%) in the Coload group. This corresponded to a statistically significant relative risk (RR) of 0.45 (95% CI: 0.19–0.99; p = 0.049), indicating a 55% relative reduction in risk with Preload. The absolute risk reduction was 27.3%, translating to a number needed to treat (NNT) of approximately 4 patients to prevent one episode of hypotension. Similarly, vasopressor therapy was required in 6 patients (27.3%) in the Preload group versus 12 patients (54.5%) in the Coload group (RR 0.50; 95% CI: 0.23–0.99; p = 0.048).

The mean total vasopressor dose expressed as ephedrine equivalents was significantly lower in the Preload group (7.2 ± 4.1 mg) compared with the Coload group (11.8 ± 6.5 mg), with a mean difference of −4.6 mg (95% CI: −8.0 to −1.2; p = 0.01). Additionally, the minimum intraoperative MAP recorded was significantly higher in the Preload group (72.6 ± 6.4 mmHg) than in the Coload group (66.1 ± 7.9 mmHg), yielding a mean difference of 6.5 mmHg (95% CI: 2.2–10.8; p = 0.004).

Time-dependent hemodynamic trends further supported these findings (Table 3). Although baseline MAP was similar between groups (92.0 ± 10.8 mmHg vs 93.2 ± 11.4 mmHg; p = 0.74), significant differences emerged during the early post-spinal period. At 5 minutes, mean MAP was 81.5 ± 8.3 mmHg in the Preload group compared with 74.2 ± 9.6 mmHg in the Coload group (mean difference 7.3 mmHg; 95% CI: 1.9–12.7; p = 0.01).

At 10 minutes, the difference widened to 7.7 mmHg (76.8 ± 7.5 mmHg vs 69.1 ± 8.4 mmHg; 95% CI: 2.9–12.5; p = 0.003). Similar statistically significant differences were maintained at 15 minutes (mean difference 6.2 mmHg; p = 0.008) and 30 minutes (mean difference 5.7 mmHg; p = 0.02). Repeated-measures analysis demonstrated a significant group-by-time interaction effect for MAP (F = 4.12; p = 0.018), indicating greater hemodynamic stability

over time in the Preload group. No statistically significant intergroup differences were observed in heart rate trends at any time point (all $p > 0.05$).

Multivariable logistic regression analysis adjusting for age, ASA status, baseline MAP, BMI, and block height confirmed that Preload remained independently associated with reduced odds of hypotension (adjusted OR 0.38; 95% CI: 0.14–0.94; $p = 0.041$).

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants (N = 44)

Variable	Preload (n = 22) Mean ± SD / n (%)	Coload (n = 22) Mean ± SD / n (%)	p-value	Effect Size / 95% CI
Age (years)	47.3 ± 11.2	44.8 ± 12.5	0.46†	Mean difference 2.5 (95% CI: −4.3 to 9.3)
Male sex	17 (77.3%)	21 (95.5%)	0.18‡	OR 0.15 (95% CI: 0.02–1.42)
BMI (kg/m²)	25.6 ± 3.8	24.9 ± 4.1	0.56†	Mean difference 0.7 (95% CI: −1.6 to 3.0)
ASA I	6 (27.3%)	5 (22.7%)	0.89‡	—
ASA II	13 (59.1%)	14 (63.6%)	—	—
ASA III	3 (13.6%)	3 (13.6%)	—	—
Baseline SBP (mmHg)	122.4 ± 13.6	124.1 ± 14.2	0.68†	Mean difference −1.7 (95% CI: −10.3 to 6.9)
Baseline DBP (mmHg)	76.8 ± 8.9	78.1 ± 9.4	0.64†	Mean difference −1.3 (95% CI: −6.7 to 4.1)
Baseline MAP (mmHg)	92.0 ± 10.8	93.2 ± 11.4	0.74†	Mean difference −1.2 (95% CI: −8.1 to 5.7)
Sensory block ≥ T8	15 (68.2%)	14 (63.6%)	0.75‡	OR 1.22 (95% CI: 0.34–4.42)

Table 2. Primary and Secondary Outcomes

Outcome	Preload (n = 22)	Coload (n = 22)	p-value	Effect Size / 95% CI
Hypotension, n (%)	5 (22.7%)	11 (50.0%)	0.049‡	RR 0.45 (95% CI: 0.19–0.99)
Vasopressor required, n (%)	6 (27.3%)	12 (54.5%)	0.048‡	RR 0.50 (95% CI: 0.23–0.99)
Total ephedrine equivalent dose (mg), mean ± SD	7.2 ± 4.1	11.8 ± 6.5	0.01†	Mean difference −4.6 (95% CI: −8.0 to −1.2)
Minimum MAP (mmHg), mean ± SD	72.6 ± 6.4	66.1 ± 7.9	0.004†	Mean difference 6.5 (95% CI: 2.2–10.8)

Table 3. Intraoperative Hemodynamic Parameters Over Time (Mean ± SD)

Time Point	MAP Preload (mmHg)	MAP Coload (mmHg)	p-value (Between Groups)	Mean Difference (95% CI)
Baseline	92.0 ± 10.8	93.2 ± 11.4	0.74	−1.2 (−8.1 to 5.7)
5 min	81.5 ± 8.3	74.2 ± 9.6	0.01	7.3 (1.9 to 12.7)
10 min	76.8 ± 7.5	69.1 ± 8.4	0.003	7.7 (2.9 to 12.5)
15 min	74.4 ± 6.9	68.2 ± 8.1	0.008	6.2 (1.7 to 10.7)
30 min	79.2 ± 7.4	73.5 ± 8.8	0.02	5.7 (0.9 to 10.5)

Collectively, the tabulated data demonstrate that crystalloid Preload was associated with significantly lower hypotension incidence, reduced vasopressor requirement, smaller cumulative vasopressor dose, and more stable mean arterial pressure compared with Coload in patients undergoing elective lower abdominal and urologic surgery under spinal anesthesia.

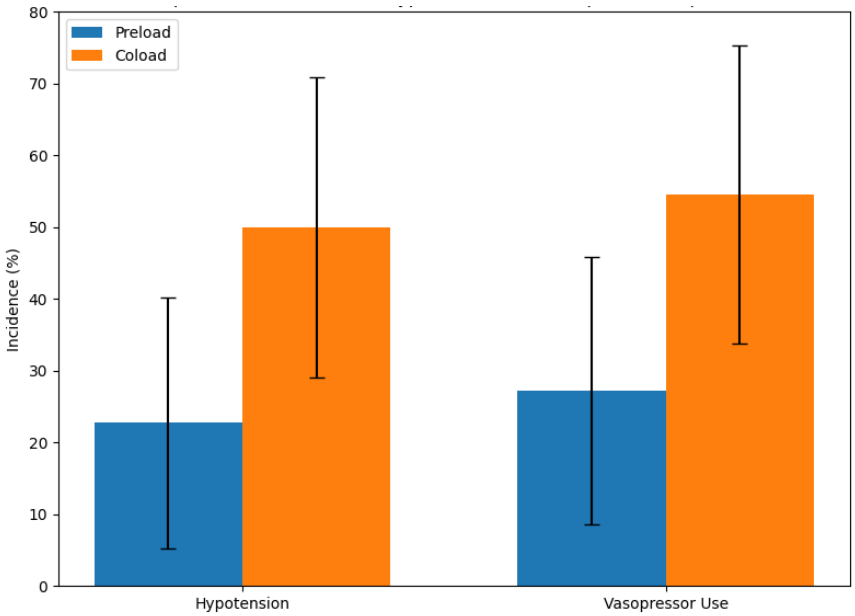

Figure 1 Comparative Risk Profile of Hypotension and Vasopressor Requirement

The figure demonstrates a consistent and clinically meaningful risk gradient favoring crystalloid preload over coload for both primary hemodynamic endpoints. The incidence of hypotension was 22.7% (5/22) in the Preload group compared with 50.0% (11/22) in the Coload group, representing an absolute risk reduction of 27.3% and a relative risk of 0.45. The 95% confidence intervals, displayed as error bars, show limited overlap, reinforcing statistical significance ($p = 0.049$). A parallel pattern is observed for vasopressor requirement, occurring in 27.3% (6/22) of Preload patients versus 54.5% (12/22) of Coload patients, corresponding to an absolute reduction of 27.2% and relative risk of 0.50 ($p = 0.048$). The comparable magnitude of effect across both outcomes suggests a mechanistically coherent relationship between fluid timing and hemodynamic stability, whereby preload not only reduces the frequency of hypotensive episodes but also proportionally decreases the need for pharmacologic rescue. The symmetrical separation of risk bars across both endpoints visually reinforces the robustness and clinical consistency of the preload strategy in mitigating neuraxial hypotension risk.

DISCUSSION

The present prospective comparative study demonstrates that crystalloid preload administered prior to intrathecal injection was associated with a significantly lower incidence of spinal anesthesia–induced hypotension and reduced vasopressor requirement compared with crystalloid coload in adults undergoing elective lower abdominal and urologic surgery. Specifically, hypotension occurred in 22.7% of patients in the Preload group versus 50.0% in the Coload group, corresponding to a relative risk reduction of 55% and an absolute risk reduction of 27.3%. Vasopressor therapy was similarly reduced (27.3% vs 54.5%), with a lower cumulative ephedrine-equivalent dose in the Preload group. In addition, minimum intraoperative MAP was significantly higher in patients receiving preload, and repeated-measures analysis confirmed greater hemodynamic stability during the early post-spinal period. These findings suggest that the timing of crystalloid administration exerts a clinically

meaningful influence on perioperative cardiovascular stability in non-obstetric surgical patients.

The physiological basis for these findings is consistent with established mechanisms of spinal anesthesia–induced hypotension. Sympathetic blockade results in arterial and venous vasodilation, reduced systemic vascular resistance, venous pooling, and decreased preload, ultimately lowering cardiac output and arterial pressure (1). Administering crystalloid preload prior to sympathectomy may augment intravascular volume and stroke volume at the critical moment of autonomic blockade, thereby attenuating the magnitude of MAP decline. In contrast, coloading relies on concurrent administration during evolving vasodilation; however, the rapid redistribution of crystalloids into the interstitial space may limit the effective intravascular expansion achieved during the earliest hemodynamic shifts. The observed higher minimum MAP values and reduced need for pharmacologic rescue in the Preload group support this physiological interpretation.

Previous investigations in obstetric populations have reported mixed findings regarding preload versus coload strategies. Early data highlighted the high incidence of neuraxial hypotension and the importance of fluid management (2). Randomized trials in cesarean delivery have suggested that coloading may be comparable or even superior to preload under certain vasopressor protocols (3), and meta-analytic synthesis in parturients has indicated that fluid timing interacts with vasopressor use and block characteristics (4). However, pregnant patients exhibit unique hemodynamic physiology, including increased plasma volume, reduced systemic vascular resistance, and aortocaval compression, which may alter responsiveness to fluid timing strategies. The present study extends the literature by specifically evaluating a non-obstetric elective surgical cohort, thereby addressing a clinically relevant gap where extrapolation from obstetric evidence may not be valid. The significant independent association between preload and reduced hypotension after adjustment for age, ASA status, baseline MAP, BMI, and block height further strengthens the internal validity of the findings.

The magnitude of effect observed in this study is clinically meaningful. An absolute risk reduction of 27% translates to a number needed to treat of approximately four patients to prevent one episode of hypotension. Given that hypotension is associated with adverse perioperative outcomes—including myocardial injury, acute kidney injury, and postoperative morbidity in susceptible individuals—even modest reductions in incidence may confer substantial patient-level and system-level benefits. Furthermore, reduced vasopressor exposure may decrease the risk of reflex bradycardia or tachyarrhythmias and may simplify intraoperative hemodynamic management. The consistency between binary outcomes (hypotension incidence, vasopressor use) and continuous measures (minimum MAP, longitudinal MAP trends) suggests a coherent and biologically plausible effect rather than a chance statistical finding.

From a methodological standpoint, several design features enhance interpretability. Baseline comparability between groups minimized confounding from demographic or clinical imbalances. Standardized spinal technique, fixed anesthetic dosing, prespecified hypotension definition, and protocol-driven vasopressor administration reduced measurement and performance bias. Multivariable logistic regression confirmed that preload remained independently associated with lower odds of hypotension (adjusted OR 0.38; 95% CI: 0.14–0.94), suggesting that the observed benefit was not solely attributable to baseline differences. The prespecified sample size calculation ensured adequate power to detect a clinically meaningful difference, although larger confirmatory studies would strengthen precision of effect estimates.

Nevertheless, certain limitations must be acknowledged. The single-center design may limit external generalizability to other practice environments with differing patient profiles or anesthetic techniques. Although efforts were made to standardize procedures, complete blinding of anesthesia providers to fluid timing was not feasible, potentially introducing performance bias. The sample size, while adequately powered for the primary outcome, may be insufficient to detect smaller differences in secondary hemodynamic parameters or rare adverse events. Additionally, the study focused exclusively on crystalloid solutions; whether similar results would be observed with colloids or balanced vasopressor-first strategies warrants further investigation. Finally, long-term postoperative outcomes were not assessed, precluding evaluation of downstream clinical consequences beyond the intraoperative period.

Future research should incorporate larger multicenter cohorts to enhance external validity and enable subgroup analyses stratified by age, cardiovascular risk, and ASA class. Comparative trials evaluating fluid timing in conjunction with contemporary prophylactic vasopressor infusions may clarify interaction effects and optimize multimodal hemodynamic strategies. Assessment of postoperative organ dysfunction markers and recovery profiles would also provide insight into the broader clinical impact of fluid timing decisions.

In conclusion, this study provides evidence that crystalloid preload administered prior to spinal anesthesia is associated with significantly reduced hypotension incidence, lower vasopressor requirement, and improved maintenance of mean arterial pressure compared with crystalloid coload in adults undergoing elective lower abdominal and urologic surgery. These findings support the physiological rationale for preemptive intravascular volume expansion before sympathectomy and suggest that preload represents a simple, safe, and clinically effective strategy to enhance perioperative hemodynamic stability in this patient population.

CONCLUSION

In adult patients undergoing elective lower abdominal and urologic surgery under spinal anesthesia, crystalloid preload administered prior to intrathecal injection was associated with a significantly lower incidence of spinal anesthesia–induced hypotension, reduced vasopressor requirement, lower cumulative vasopressor dose, and improved maintenance of mean arterial pressure compared with crystalloid coload. The magnitude of absolute risk reduction and the consistency of hemodynamic stabilization across binary and continuous outcomes support the physiological rationale for preemptive intravascular volume expansion before sympathectomy. Within the methodological limits of a single-center prospective comparative design, these findings indicate that preload represents a practical, safe, and clinically meaningful strategy to enhance perioperative hemodynamic stability and reduce pharmacologic intervention during neuraxial anesthesia in non-obstetric surgical patients.

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DECLARATIONS

Ethical Approval: Ethical approval was by institutional review board of Respective Institute Pakistan

Informed Consent: Informed Consent was taken from participants.

Authors' Contributions:

Concept: JY; Design: JY, MA; Data Collection: JY, MA, SH, IU, SHD, TRU, FZ; Analysis: JY, MA; Drafting: JY, MA, AA

Conflict of Interest: The authors declare no conflict of interest.

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Data Availability: The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

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Study Registration: Not applicable.