Original Article

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Prophylactic Intravenous Phenylephrine to Prevent Propofol-induced Hypotension during Induction of General Anaesthesia: A Randomised Clinical Trial

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ABSTRACT

Introduction: Propofol is commonly used intravenous induction agent which are frequently complicated by peri-induction hypotension. Reduced Mean Arterial Pressure (MAP) during anaesthesia contributes to significant postoperative morbidity, including renal and myocardial injury. Preventive strategies aimed at maintaining haemodynamic stability during this critical period are of clinical value. Phenylephrine offers a simple, rapid option for counteracting propofol-induced vasodilatation.

Aim: To assess effectiveness of i.v. phenylephrine in preventing propofol-induced hypotension during induction of general anaesthesia.

Materials and Methods: This randomised clinical trial was conducted from October 2024-December 2025 at Department of Anaesthesiology, Shri BM Patil Medical College, Hospital and Research Centre, BLDE DU, Vijayapura, Karnataka, India. The study included 130 American Society of Anaesthesiologists (ASA) I–II patients (18–65 years) undergoing elective general anaesthesia and were randomised (1:1) to receive i.v. phenylephrine 100 µg or saline before propofol induction. Primary outcome was hypotension (≥20% MAP fall). Secondary outcomes included Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), MAP, and Heart Rate (HR) at 5, 10, and 15 minutes. Analysed using Statistical Package for the Social Sciences (SPSS) version 20.0, normally distributed continuous variables (Shapiro-Wilk test) were expressed as mean±SD and evaluated via independent student's t-tests.

Results: The control group's average age was 37.76±15.69 years (50.8% female, 49.2% male), the study group averaged 34.38±14.30 years (49.2% female, 50.8% male). Postinduction hypotension was lower in the study group (32.3%, n=21 vs 73.8%, n=48; p-value <0.001). At 5, 10, and 15 minutes, Group A maintained higher MAP, SBP, and DBP than Group B (all p-value <0.001), Group A vs Group B MAP was 87.88±8.694 vs 79.45±9.384 (p-value <0.001), 86.51±9.045 vs 74.86±9.890 (p-value <0.001), and 87.60±9.375 vs 77.55±8.816 mmHg (p-value <0.001); SBP was 117.80±10.145 vs 107.48±11.172 (p-value <0.001), 116.46±10.827 vs 102.37±11.870 (p-value <0.001), and 117.43±11.243 vs 105.25±10.070 mmHg (p-value <0.001); DBP was 71.45±9.038 vs 65.18±9.084 (p-value <0.001), 70.20±9.00 vs 60.98±8.977 (p-value <0.001), and 71.32±9.059 vs 63.45±8.646 mmHg (p-value <0.001). Group A HR was lower at 10 minutes (88.60±11.900 vs 93.29±14.021 bpm, p-value=0.009) and 15 minutes (85.18±11.144 vs 91.75±12.668 bpm, p-value=0.001).

Conclusion: A single 100 µg bolus of phenylephrine administered immediately before induction with propofol effectively attenuated peri-induction hypotension and maintained stable haemodynamic. This simple intervention improves perioperative safety in patients at risk of hypotension.

Keywords: Acute kidney injury, Baroreflex, Blood pressure, Hypovolemia, Vasoconstrictor agents

INTRODUCTION

Propofol remains the most commonly administered intravenous agent for induction of anaesthesia due to its rapid onset, smooth transition to unconsciousness, and predictable recovery profile [1,2]. Conventional strategies such as administering fluids before induction or slowing the rate of propofol injection have yielded inconsistent protection and often fail to adequately maintain haemodynamic stability. Induction of general anaesthesia is a phase marked by significant haemodynamic instability, with hypotension being one of the most frequent and clinically important complications encountered while using propofol as an induction agent. Available literature from clinical trials is diverse and limited in scope, lacking uniformity in dosage protocols, timing, and administration technique. However, despite these benefits, propofol is well known to cause marked reduction in blood pressure, occurring in approximately 20-40% of cases even at conventional doses [3,4]. The existing literature highlights the severity of propofol-induced hypotension, demonstrating that short durations of a MAP below 55 mmHg are sufficient to precipitate renal injury, induce myocardial ischaemia, and significantly increase postoperative morbidity and mortality [5,6]. The haemodynamic impact is especially pronounced in elderly, hypovolemic, or hypertensive individuals, as well as in patients with impaired autonomic reflex regulation [7].

The mechanisms underlying propofol-induced cardiovascular depression are multifactorial. It lowers systemic vascular resistance through direct vasodilatation and inhibition of sympathetic vasoconstrictor tone [8], while also causing venodilatation that diminishes venous return and cardiac preload [9]. Additionally, propofol blunts baroreceptor reflex sensitivity, reducing the compensatory tachycardic response to hypotension [10], and exerts a mild negative inotropic effect on the myocardium [11]. Collectively, these effects make the fall in blood pressure following propofol more pronounced than with many other intravenous induction drugs [12].

Because of the adverse consequences of postinduction hypotension, multiple preventive measures have been proposed. Preinduction fluid loading may help but can be restricted by the risk of overload and reduced efficacy in high-risk or elderly individuals [13-17]. Slowing the injection rate or using target-controlled infusions can lessen the fall in pressure but are not always feasible [17]. Direct laryngoscopy elicits an acute sympathoadrenal response

with increased plasma catecholamines, producing tachycardia and hypertension that may transiently counterbalance propofol-induced vasodilatory hypotension. As a result, there is growing interest in the prophylactic use of vasopressors, specifically, agents like ephedrine, norepinephrine and phenylephrine are increasingly utilised for both the prevention and treatment of anaesthetic-induced hypotension [18,19].

Phenylephrine, a selective α1-adrenergic agonist, increases systemic vascular resistance and enhances venous return through arterial and venous constriction, without directly stimulating the heart [19]. Its quick onset and short duration make it well suited for managing peri-induction blood pressure changes. In obstetric anaesthesia, phenylephrine is widely recognised for preventing and treating spinal-induced hypotension during caesarean delivery, with both bolus and infusion regimens proving effective [20,21]. Recently, phenylephrine use has expanded to preventing propofol-related hypotension during general anaesthesia, with several studies reporting a significant reduction in hypotensive episodes and decreased rescue vasopressor requirements when administered prophylactically [18,22].

However, given that the available evidence is derived from limited and heterogeneous trials, the optimal dosage, timing, and route of administration remain to be clearly established [23]. In order to evaluate the potency of prophylactic intravenous phenylephrine in reducing hypotension during propofol induction, the current randomised clinical trial has been conducted. This study seeks to enhance patient safety and peri-induction management by methodically assessing haemodynamic parameters and possible adverse effects.

The present study aimed to assess the effectiveness of i.v. phenylephrine in preventing propofol-induced hypotension during induction of general anaesthesia. The primary objective was to assess the efficacy of i.v. phenylephrine in reducing the incidence of hypotension (defined as a drop in MAP ≥20% from baseline) following induction with propofol and the secondary objectives were to record changes in SBP, DBP, MAP and HR after induction and intubation in the phenylephrine group, to record the same haemodynamic parameters in the control group and to compare these parameters between both groups to determine statistical significance.

MATERIALS AND METHODS

This double-blinded, randomised clinical trial was conducted at the Department of Anaesthesiology, Shri BM Patil Medical College, Hospital and Research Centre, BLDE DU, Vijayapura, Karnataka, India during October 2024-December 2025. The participants were blinded to the group allocation. A total of 130 adult patients (18-65 years, ASA I–II) scheduled for elective surgery (ENT surgery, neurosurgery, abdominal surgery with duration of surgery more than 1 hour) under general anaesthesia were enrolled. The study protocol was approved by the Institutional Ethics Committee (IEC) {Protocol Number: BLDE(DU)/IEC-SBMPMC/068/2023-24} and was registered with the Clinical Trials Registry {Registration Number:- CTRI/2025/04/084484}. Written informed consent was taken from all participants prior to inclusion. The study adhered to the principles of the Declaration of Helsinki.

Inclusion criteria: Patients of either sex, aged 18 to 65 years, with an ASA physical status of I or II were included in the study.

Exclusion criteria: Patients with history of ischaemic heart disease, pheochromocytoma, conduction abnormalities, uncontrolled hypertension, a postintubation blood pressure exceeding 180/110 mmHg, an anticipated difficult airway, or any known drug hypersensitivity were excluded from the study.

Sample size: The sample size (n=130; 65/group) was calculated based on a previous study by Farhan M et al., which reported a difference in the incidence of hypotension (defined as a drop in MAP ≥20% from baseline) between groups (PS 60% vs PP 24.4%) with α =0.05 and 99% power [13].

Formula used is:

$$n=\frac{(z_\alpha+z_\beta)^2\ 2\ p^*q}{MD^2}$$

Where Z= Z statistic at a level of significance

MD= Anticipated difference between two proportions

P=Common Proportion

q= 100-p

The exact variables:

P_1 (Group PS)=0.60, so $_{q1}$=0.40

P_2(Group PP)=0.244, so q_2=0.756

$\bar{P}$ (Average proportion)=0.422, so $\bar{q}$=0.578

z_α(0.05 significance)=1.96

z_β (99% power)=2.326

Numerical Calculation:

Numerator Part 1: $1.96\times\sqrt{2 * 0.422 * 0.578}=1.369$

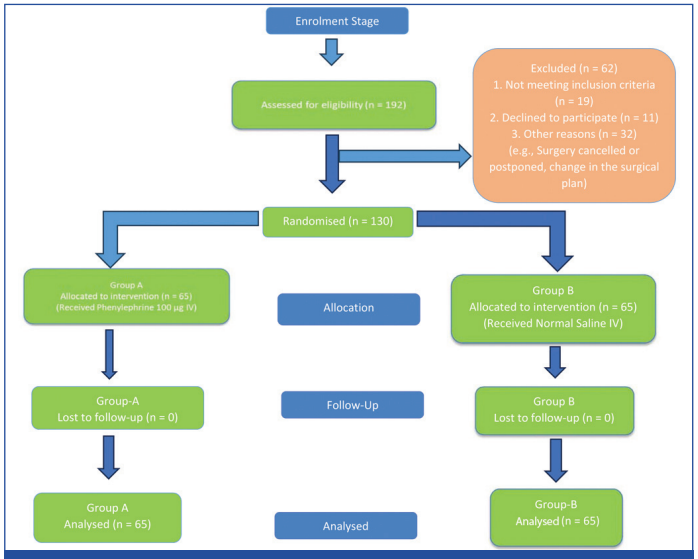
Numerator Part 2: $2.326\times\sqrt{(0.60 * 0.40) + (0.244 * 0.756)}=1.515$

Total Numerator: $(1.369+1.515)^2=(2.884)^2=8.317$

Denominator (MD^2): $(0.356)^2=0.1267$

$n=\frac{8.317}{0.1267}=65.6$

Randomisation and intervention: Participants were allocated to study groups using a computer-generated randomisation sequence prepared by an independent statistician prior to study initiation [Table/Fig-1]. Allocation concealment was ensured using sequentially numbered, opaque, sealed envelopes, which were opened only after the participant had been enrolled in the study. Participant enrolment was performed by a study investigator who was not involved in generating the random sequence.



[Table/Fig-1]: CONSORT flow diagram showing participant enrolment, allocation, follow-up, and analysis in the study and control groups.

- **Group A (Intervention):** Inj. phenylephrine 100 µg (10 mL) administered intravenously before induction of anaesthesia [24] (before i.v. propofol).
- **Group B (Control):** Equal volume of normal saline (10 mL) administered at the same time point.

The study drug solutions were prepared by an anaesthesiologist who was not involved in patient monitoring or data collection to maintain allocation concealment and ensure double blinding. Both the patient and the anaesthesiologist (double blinding) administering the drug were blinded to group assignment, with study medications presented in identical syringes with respect to volume, appearance and labelling, thereby minimising performance bias.

Anaesthetic technique: All patients underwent standard preanaesthetic evaluation and fluid preloading (5-7 mL/kg of Ringer's lactate/ 0.9% Normal Saline 15-20 minutes before induction). Patients were premedicated with midazolam 1 mg and ondansetron 4 mg i.v. induction was started with phenylephrine 100 µg (10 mL) for study group, and normal saline (10 mL) for control group (30 seconds before propofol), followed by propofol 2 mg/kg i.v., succinylcholine 1.5 mg/kg for intubation. Anaesthesia was maintained with isoflurane in Oxygen/Nitrous Oxide and atracurium. Reversal was with neostigmine and glycopyrrolate at the end of surgery.

Data collection: Haemodynamic variables (HR, SBP, DBP, MAP) were recorded as single reading using a multiparameter monitor (Manufacturer - GE Medical Systems Information Technologies Inc., Model: B125M Patient Monitor) at:

- **T0:** baseline (before induction),
- **T1:** 5 min postintubation,
- **T2:** 10 min postintubation,
- **T3:** 15 min postintubation.

The primary outcome was hypotension, defined as a ≥20% fall in MAP after induction and secondary outcome was changes in SBP, DBP and HR after induction.

STATISTICAL ANALYSIS

The data was entered into Microsoft Excel sheet and statistical analysis was conducted using SPSS version 20.0 (IBM Corp, Armonk, NY, USA). The results were presented as mean±Standard Deviation (SD), percentages and bar graphs. An independent student's t-test was utilised for continuous variables. The Chi-square test was used to evaluate categorical data (incidence of hypotension) that were expressed as frequency and percentages. Statistical significance was defined as a p-value <0.05. All tests that are used for statistical analysis are mentioned in the footnotes of tables/figures.

RESULTS

Age, gender, and ASA grading distribution: The study and control groups were well-matched with no statistically significant differences in age, gender distribution, or ASA physical status, indicating successful randomisation and minimal baseline confounding [Table/Fig-2,3]. The control group's average age was 37.76±15.69 years (50.8% female, 49.2% male), the study group averaged 34.38±14.30 years (49.2% female, 50.8% male).

Incidence of postinduction hypotension: The prophylactic phenylephrine group experienced a significantly lower incidence of postinduction hypotension compared to the control group (32.3% vs 73.8%; p-value <0.001), which translated to a substantially reduced requirement for rescue mephentermine boluses [Table/Fig-4].

Changes in Mean Arterial Pressure (MAP): Prophylactic phenylephrine effectively attenuated the postinduction fall in MAP, maintaining significantly higher pressures than the control group [Table/Fig-5].

Changes in Systolic Blood Pressure (SBP): Following propofol administration, the intervention group preserved significantly higher systolic pressures throughout the observation period compared to the control group [Table/Fig-6].

Parameters		Study group (n=65)	Control group (n=65)	Chi-square (χ^2)	p-value
Gender	Male	33 (50.8%)	32 (49.2%)	0.031	0.861
	Female	32 (49.2%)	33 (50.8%)		
ASA	I	49	47	0.1593	0.689
	II	16	18		

[Table/Fig-2]: Gender and ASA grading wise distribution between the study and control groups.
[%]: Percentage. Data is presented as frequency (percentage). P-value was calculated using the Chi-square test.

Age (years)	Study group	Control group	Total	Chi-square (χ^2)	p-value
< 20	11	7	18	11.903	0.219
	16.9%	10.8%	13.8%		
20 - 24	10	10	20		
	15.4%	15.4%	15.4%		
25 - 29	6	9	15		
	9.2%	13.8%	11.5%		
30 - 34	12	7	19		
	18.5%	10.8%	14.6%		
35 - 39	8	3	11		
	12.3%	4.6%	8.5%		
40 - 44	2	4	6		
	3.1%	6.2%	4.6%		
45 - 49	2	6	8		
	3.1%	9.2%	6.2%		
50 - 54	5	8	13		
	7.7%	12.3%	10.0%		
55 - 59	4	1	5		
	6.2%	1.5%	3.8%		
60 - 65	5	10	15		
	7.7%	15.4%	11.5%		
Total	65	65	130		
	100.0%	100.0%	100.0%		

[Table/Fig-3]: Age wise distribution between the study and control groups.
[%]: Percentage. Data is presented as frequency (percentage). p-value was calculated using the Chi-square test.

Variable	Study group (n=65)	Control group (n=65)	p-value
Hypotension present	21 (32.3%)	48 (73.8%)	<0.001*
Hypotension absent	44 (67.7%)	17 (26.2%)	

[Table/Fig-4]: The incidence of postinduction hypotension between the study and control groups.
[n]: Number of patients; [%]: Percentage. Data is presented as number (percentage). p-value was calculated using the Chi-square test. *indicates a statistically significant difference.

Time interval	MAP		p-value
	Study group (Mean±SD)	Control group (Mean±SD)	
Baseline	97.66±9.166	96.58±7.084	0.548
5 minutes	87.88±8.694	79.45±9.384	<0.001*
10 minutes	86.51±9.045	74.86±9.890	<0.001*
15 minutes	87.60±9.375	77.55±8.816	<0.001*

[Table/Fig-5]: Comparison of Mean Arterial Pressure (MAP) between the study and control groups at various time intervals.
SD: Standard deviation; MAP: Mean arterial pressure. Group A: Phenylephrine group; Group B: Control group. Data is presented as Mean±SD. p-value was calculated using the independent Student's t-test. * indicates a statistically significant difference (p<0.05).

Time Interval	Systolic Blood Pressure (SBP)		p-value
	Study Group (Mean±SD)	Control Group (Mean±SD)	
Baseline	130.45±10.053	129.66±9.089	0.636
5 minutes	117.80±10.145	107.48±11.172	<0.001*
10 minutes	116.46±10.827	102.37±11.870	<0.001*
15 minutes	117.43±11.243	105.25±10.070	<0.001*

[Table/Fig-6]: Comparison of mean Systolic Blood Pressure (SBP) between the study and control groups at various time intervals.
SD: Standard Deviation; Data is presented as Mean±SD. p-value was calculated using the independent Student's t-test. * indicates a statistically significant difference (p<0.05).

Changes in Diastolic Blood Pressure (DBP): Prophylactic administration also successfully mitigated the fall in DBP, yielding significantly higher diastolic readings in the study group [Table/Fig-7].

Time Interval	Diastolic Blood Pressure (DBP)		p-value
	Study group (Mean±SD)	Control group (Mean±SD)	
Baseline	80.68±7.987	79.95±7.294	0.694
5 minutes	71.45±9.038	65.18±9.084	<0.001*
10 minutes	70.20±9.000	60.98±8.977	<0.001*
15 minutes	71.32±9.059	63.45±8.646	<0.001*

[Table/Fig-7]: Comparison of mean Diastolic Blood Pressure (DBP) between the study and control groups at various time intervals.
SD: Standard deviation; Data is presented as Mean±SD. p-value was calculated using the independent Student's t-test. * indicates a statistically significant difference (p<0.05).

Changes in Heart Rate (HR): While no significant difference was observed at five minutes, the phenylephrine bolus effectively decreased reflex tachycardia at subsequent intervals, resulting in significantly lower HRs in the study group [Table/Fig-8].

No significant adverse effects or complications were observed in either group during the study period.

Time Interval	Heart Rate (HR)		p-value
	Study group (Mean±SD)	Control group (Mean±SD)	
Baseline	84.34±12.094	85.72±11.093	0.467
5 minutes	87.52±11.141	90.68±15.712	0.095
10 minutes	88.60±11.900	93.29±14.021	0.009*
15 minutes	85.18±11.144	91.75±12.668	0.001*

[Table/Fig-8]: Comparison of mean Heart Rate (HR) between the study and control groups at various time intervals.
SD: Standard deviation; Data is presented as Mean±SD. p-value was calculated using the independent Student's t-test. * indicates a statistically significant difference (p<0.05).

DISCUSSION

Propofol is a preferred induction agent [1-3], but frequently causes postinduction hypotension through direct arteriolar vasodilatation, venodilatation reducing preload and impaired baroreflexes [8-11]. These effects sharply lower Systemic Vascular Resistance (SVR), making MAP highly vulnerable [12]. Even brief MAP drops below 55 mmHg are linked to Acute Kidney Injury (AKI), myocardial injury, and postoperative morbidity [5,6]. The primary objective of this study was to determine how well the prophylactic phenylephrine bolus prevented postinduction hypotension. The study group exhibited a considerably lower postinduction hypotension incidence than controls (32.3%, 21 patients vs 73.8%, 48 patients; p-value <0.001). Furthermore, the study group maintained significantly higher, stable secondary parameters (MAP, SBP, and DBP) at all intervals, while controlling HR without reflex tachycardia.

These hypotension and MAP preservation findings correlate with Farhan M et al., [13]. They evaluated prophylactic vasopressors, reporting a 38.5% (52/135) overall hypotension incidence. Hypotension was significantly higher in their saline control group (group PS) compared to their prophylactic phenylephrine group (Group PP) (60% vs 24.4%; p-value=0.001) and ephedrine group (Group PE) (60% vs 31.1%; p-value=0.005) [13]. In the present study pre-emptive phenylephrine provided substantial protection against early MAP decline.

Present study hypotensive reductions and SBP/DBP stabilisation align with Smithamol PB [14]. They demonstrated a sharp contrast: 82.14% hypotension in Group A and 79.31% in group B, compared to a highly significant 0% in the phenylephrine group (Group-C) [14]. By averting initial vasodilatory shock, phenylephrine inherently protected systolic and diastolic parameters. Additionally, while vasopressors like ephedrine are effective, they usually cause tachycardia [14]. The present study findings confirm phenylephrine avoids this, offering optimal HR control alongside pressure stabilisation.

Regarding immediate haemodynamic stabilisation and counteracting dropping SVR, the present study results agree with Kwok FY et al., [4]. They demonstrated marked induction-related hypotension reductions in elderly patients using prophylactic phenylephrine. Their research highlighted that phenylephrine 200 µg outperforms 100 µg in attenuating hypotension for patients over 55, especially during the first two minutes [4]. Although the present study successfully utilised a 100 µg dose in a broader demographic (18-65 years) to maintain all parameters, the pre-emptive alpha-agonist principle remains highly consistent.

Ultimately, intervention timing is crucial. Prophylactic phenylephrine prevents crossing haemodynamic thresholds, minimising organ hypoperfusion better than standard responsive treatments. While multiple vasopressors exist, each has limitations. Ephedrine typically causes tachycardia [14], and norepinephrine generally requires impractical infusion pumps for routine cases [19]. Therefore, phenylephrine offers an optimal middle ground, delivering stable haemodynamic support and minimal HR disturbance via a simple bolus dose.

A primary strength of this study was its randomised, double-blind design, which effectively reduced selection and observation bias. The present study also strictly standardised the anaesthetic induction protocol and defined hypotension based on a relative percentage drop (<20% MAP from baseline) rather than a fixed absolute value; this offers a more physiological assessment of haemodynamic instability tailored to each patient. The laryngoscopy stimulation can cause a sympathetic stimulus which confounds the effects of phenylephrine, this confounding factor was reduced by standardising intubation technique, duration, and excluding difficult airways. Additionally, the present study findings align with both international and regional Indian data, reinforcing the external validity of the results.

Since this study recognised the efficacy of a fixed 100 µg phenylephrine prophylactic bolus in healthy patients, future research should focus on refining dosing strategies, such as weight-based dosing or target-controlled infusions, to further minimise haemodynamic variability. Comparative trials are also needed to evaluate phenylephrine against norepinephrine which may be more suitable for patients with compromised cardiac function who cannot tolerate increased afterload.

Additionally, expanding this protocol to high-risk populations (ASA III-IV, elderly, and cardiac patients) is critical, as these groups are most vulnerable to the adverse effects of hypotension. Lastly, future studies should incorporate non invasive cardiac output monitoring and assess long-term outcomes, such as postoperative AKI and myocardial injury, to validate the clinical benefit of preventing transient induction hypotension [25,26].

Limitation(s)

First, the study was restricted to ASA I and II patients undergoing elective surgery; therefore, findings may not be generalisable to geriatric, septic, or cardiac-compromised patients who may respond differently to alpha-agonists. Second, the present study relied on non invasive blood pressure monitoring as a surrogate for haemodynamic stability; the present study did not measure Cardiac Output (CO) or stroke volume. Since phenylephrine increases afterload, it can theoretically reduce cardiac output despite improving blood pressure- a variable this study could not assess. Finally, the present study utilised a fixed bolus dose (100 µg); a weight-based dosing regimen or a target-controlled infusion might offer more precise haemodynamic control.

CONCLUSION(S)

This randomised, double-blind clinical trial demonstrated that prophylactic intravenous phenylephrine administered immediately prior to propofol induction effectively prevents peri-induction hypotension and ensures superior overall haemodynamic stability. Specifically, patients receiving this pre-emptive bolus successfully maintained stable MAP and SBP without experiencing adverse

reflex tachycardia or bradycardia. These findings highlight that pre-emptive haemodynamic intervention is clinically superior to responsive treatment, as averting the initial blood pressure drop mitigates the risk of occult organ hypoperfusion. Furthermore, this approach provides distinct clinical advantages by being cost-effective, simple to administer, and independent of complex infusion equipment. Therefore, while future studies should evaluate higher-risk ASA III-IV populations, the present study results strongly support the routine prophylactic uses of phenylephrine in ASA I and II patients to facilitate a safer, more stable induction of general anaesthesia.

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