

Propofol Priming for Enhancing Induction Safety in Hypertensive Patients: A Prospective Double-blind Randomised Controlled Trial

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ABSTRACT

Introduction: Propofol is popular for inducing general anaesthesia, but it has drawbacks such as hypotension and bradycardia in a dose-dependent manner, especially in hypertensive patients. Reducing the total dose of Propofol using the priming principle has proven to be effective; however, it has not been studied in high-risk hypertensive patients. Therefore, the current study was planned.

Aim: To evaluate the effects of priming on Propofol induction in controlled hypertensive patients, with objectives to assess haemodynamic parameters, total dose, and side-effects.

Materials and Methods: This prospective, comparative, double-blind randomised controlled trial was performed at the Department of Anaesthesiology, Pt. Bhagwat Dayal Sharma (B.D.) Postgraduate Institute of Medical Sciences (PGIMS) Rohtak, Haryana, India, from April 2023 to April 2024. A total of 60 patients scheduled for elective surgery under general anaesthesia with controlled hypertension were randomly assigned to either Group 1 (study group, n=30), where Propofol priming was used, or Group 2 (control group, n=30). Demographics, total Propofol dose, haemodynamic parameters

{Heart Rate (HR), Systolic Blood Pressure (SBP), Mean Arterial Pressure (MAP)}, and adverse events were evaluated using International Business Machine (IBM) Statistical Package of Social Sciences (SPSS) version 25.0. The Kolmogorov-Smirnov test was used for normalisation, and the Mann's-Whitney U test, Independent t-test, and Chi-square test were used for data analysis.

Results: In Group 1 (study), the mean age was 49.36±9.42 years, while in Group 2 (control), it was 49.36±9.19 years (p=0.91). A lesser dose of Propofol was needed in the study group (Group 1) (90.07±19.73 mg) compared to the control group (Group 2) (120.00±21.81 mg) (p=0.001). Haemodynamic alterations were more pronounced in the controls compared to the study group. After induction, a significant fall in MAP was observed in the controls compared to the study group (67.63±5.86 mmHg vs. 90.63±4.33 mmHg) (p=0.001). Adverse effects, such as pain at the injection site, were reported in one patient (n=1) in Group 2.

Conclusion: The priming principle significantly reduces the total Propofol dose and leads to more stable haemodynamics during general anaesthesia induction in controlled hypertensive patients. Priming with Propofol is advised for patients with hypertension when inducing general anaesthesia.

Keywords: Dose, Hypotension, General anaesthesia

INTRODUCTION

Daycare surgeries are on the rise, increasing the demand for daycare anaesthesia [1]. Thus, there is a need for an ideal anaesthetic agent characterised by rapid induction, prompt recovery, and minimal side-effects. Propofol has emerged as the most suitable anaesthetic induction agent, gaining popularity due to its rapid onset, rapid elimination, and clear-headed recovery [2]. However, Propofol is not devoid of adverse effects, such as hypotension, bradycardia, anaphylactic reactions, and pain on injection [3]. Hypotension is a significant cause of morbidity during anaesthesia in hypertensive patients [4]. Hypertensive patients have increased vasoreactivity, which helps regulate blood pressure. In hypertensive patients, the baroreflex is reset to a higher level to maintain blood flow to organs at a higher blood pressure, protecting perfusion even during a hypertensive crisis [5]. Despite this protective mechanism, hypertensive patients are less capable of tolerating minimal hypotension, which compromises perfusion. Propofol-induced hypotension is dose-dependent, so decreasing the total dose requirement can reduce associated morbidity [3]. Various methods have been described in the literature to reduce Propofol dose requirements, such as the simultaneous use of nitrous oxide, opioids, and barbiturates [6]. Other methods, like preloading, slow administration of Propofol, parallel use of vasoactive agents, and the priming principle, have also been found to be effective [7-9]. The priming principle involves administering a predetermined sub-anaesthetic dose of the inducing agent prior to the complete

dose [8]. Hypertensive patients face a higher risk of hypotension during the induction of general anaesthesia due to the vasodilatory effects of induction agents [10]. Previous researchers have shown that Propofol priming facilitates a gradual induction with a reduced total Propofol dose, thereby minimising the risk of hypotension and maintaining stable haemodynamic parameters during the induction of general anaesthesia [3,6,11-15]. However, the safety of priming techniques during induction in hypertensive patients has not been studied at all. To fill this gap in the literature, the current study aimed to evaluate the effects of priming on Propofol induction in controlled hypertensive patients, with the objective of observing haemodynamic parameters and total Propofol dose requirements.

MATERIALS AND METHODS

The prospective, randomised, double-blind comparative study was conducted in the Department of Anaesthesiology, Pt. B.D.Sharma PGIMS, Rohtak, Haryana, India, from April 2023 to April 2024. The study complied with the ethical standards of the Institutional Biomedical Research Ethics Committee and received formal approval (no. BREC/22/TH/Anaesth-22). It was also registered with the Clinical Trial Registry of India (registration no. CTRI/2023/03/050586).

Sample size calculation: A total sample size of 60 patients, with 30 patients in each of the two groups, was calculated based on the study by Suganya S et al., [11]. For the sample size calculation, a mean difference of 14.25 was used, with a 95% confidence interval, 80% power, and an alpha level of 0.05.

Inclusion criteria: Patients aged 18 to 60 years, of either sex, with controlled hypertension (blood pressure <140/90 mmHg) who had been on antihypertensive treatment for atleast eight weeks and were scheduled for elective surgery under general anaesthesia were included in the study after obtaining written informed consent.

Exclusion criteria: Patients were excluded if they had a history of allergy to the study drug, an anticipated difficult airway, a Body Mass Index (BMI) over 30 kg/m², were pregnant, or were unwilling to participate in the study.

Study Procedure

Patients were randomised into two groups using computer-generated randomisation numbers. Group 1 (study group, n=30) received 25% of the calculated dose of 2 mg/kg of Propofol intravenously as a priming dose [11]. Group 2 (control group, n=30) received 5 mL of normal saline intravenously.

The priming drug was prepared in a 5 mL syringe by an anaesthesia technician not involved in the study. Blinding was achieved by wrapping the loaded syringe in opaque paper. Priming and induction were carried out by an anaesthesiologist not involved in the study. All observations and vital signs were recorded by the investigator of the study. All patients were examined the day before surgery during the preoperative visit. They were allowed to consume solids upto six hours and liquids upto two hours prior to surgery.

The anaesthesia procedure was standardised. A peripheral intravenous line was established, and monitoring was conducted, including electrocardiography, pulse oximetry, non invasive blood pressure measurements, and end-tidal carbon dioxide measurements. Patients were preoxygenated for three minutes with 100% oxygen. Prior to induction, premedication was administered with an intravenous injection of glycopyrrolate (0.004 mg/kg) and an intravenous injection of fentanyl (2 mcg/kg).

In Group 1 (study), Propofol was used for priming, with a priming dose set at 0.5 mg/kg, constituting 25% of the calculated induction dose of Propofol (2 mg/kg). Priming commenced 3 minutes following premedication and was administered over 10 seconds [11]. Conversely, in Group 2 (control), 5 mL of saline was administered over the same 10-second duration. After two minutes of the priming dose, patients in both groups were induced with an injection of Propofol, administered at a rate of 30 mg/10 seconds via a titration method until loss of vocalisation. The cumulative Propofol dosage was recorded. Following induction, a muscle relaxant, inj. Vecuronium bromide 0.1 mg/kg, was given as a loading dose to facilitate endotracheal intubation. Patients were intubated with an adequately sized endotracheal tube, and anaesthesia was maintained by intermittent muscle relaxants, oxygen {Fraction of Inspired Oxygen (FiO₂ 35%)}, nitrous oxide, and sevoflurane (dial concentration adjusted according to an age-related iso-MAC chart). Haemodynamic parameters, including HR, SBP, Diastolic Blood Pressure (DBP), and MAP, were recorded at various time points. Measurements were taken 30 minutes prior to induction in the preoperative room (T1), at baseline upon transfer to the operating table (T2), immediately after premedication administration (T3), immediately after the priming dose (T4), 2 minutes post-priming (T5), immediately after induction (T6), and three minutes post-induction (T7). If side-effects such as pain at the injection site, pruritus, or anaphylaxis were encountered, they were noted and managed with inj. lignocaine 2% i.v. 1 ml, inj. Pheniramine maleate 44.5 mg i.v. over five minutes, and inj. Adrenaline 0.1 mg/kg Subcutaneous (SC), respectively, along with supportive treatment.

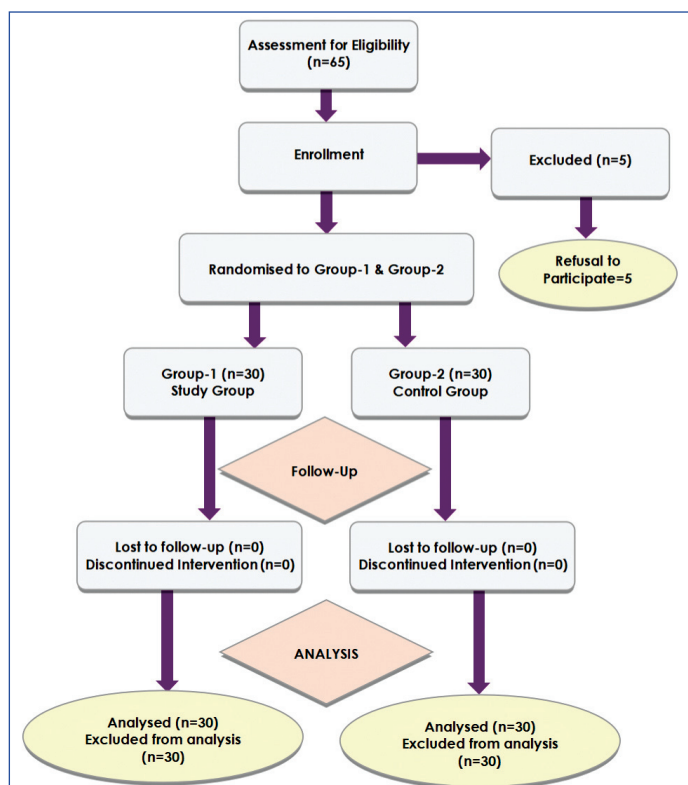
STATISTICAL ANALYSIS

The statistical analysis for the study was conducted using IBM SPSS version 25.0 (SPSS Inc., IBM Corporation, NY, USA), with the data compiled and organised in a Microsoft Excel spreadsheet. Descriptive statistics were computed to determine percentages,

means, and Standard Deviation (SD) of the collected data. Before proceeding with the statistical analysis, the dataset was subjected to normality checks using the Kolmogorov-Smirnov test. Subsequently, appropriate statistical tests, such as the Mann's-Whitney U test, Independent t-test, and Chi-square test, were employed for data analysis. The level of significance was set at p<0.05, ensuring that results with a probability of occurrence less than or equal to 5% were considered statistically significant.

RESULTS

The study initially assessed a total of 65 patients for eligibility. Among these, five patients declined to participate, resulting in 60 patients who were subsequently randomised into two groups: Group 1 (study) and Group 2 (control). The flow of participants through the study process can be visualised in the Consolidated Standards of Reporting Trails (CONSORT) flowchart [Table/Fig-1].



[Table/Fig-1]: CONSORT flowchart.

In Group 1 (study), the mean age was 49.36±9.42 years, while in Group 2 (control), it was 49.36±9.19 years (p=0.91). The female-to-male ratio in Group 1 was 23:7, whereas it was 18:12 in Group 2 (p=0.16). The mean BMI was 23.69±3.44 kg/m² in Group 1 and 24.87±3.56 kg/m² in Group 2 (p=0.19). These findings suggest that the baseline demographic characteristics, including age, gender distribution, and BMI, were comparable (p>0.05) between the study and control groups, as represented in [Table/Fig-2].

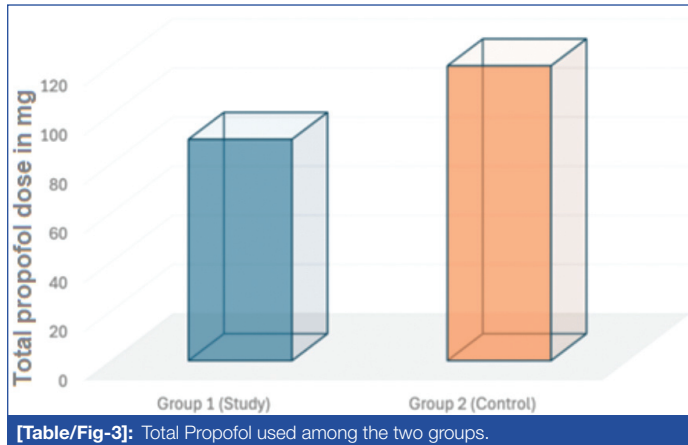
Demographic parameters	Group 1 (Study)	Group 2 (Control)	p-value
Age (mean±SD)	49.36±9.42 years	49.36±9.19 years	0.901*
Gender (n%)	Male	12 (40%)	0.165^
	Female	18 (60%)	
Weight (mean±SD)	66.66±12.28 kg	62.96±11.85 kg	0.240*
Height (mean±SD)	163.23±5.39 cm	162.40±5.46 cm	0.555*
BMI (mean±SD)	24.87±3.56 kg/m ²	23.69±3.44 kg/m ²	0.198*

[Table/Fig-2]: Demographic parameters.

*Independent t-test; ^Chi-square test used

The total dose of Propofol administered differed significantly between the two groups. In Group 1 (study), the mean total Propofol used was 90.07±19.73 mg, whereas in Group 2 (control), it was

120.00±21.81 mg (p=0.001). This indicates a significant difference in the total Propofol dosage between the study and control groups, as represented in [Table/Fig-3].



The mean HR per minute at T6 was significantly lower in Group 1 (90.57±11.04) compared to Group 2 (99.60±11.08), with a p-value of 0.002. At the time interval T7, Group 2 had a lower mean HR (78.03±8.90 per minute) compared to Group 1 (84.37±10.50 per minute), with a p-value of 0.015. The mean HRs at different time intervals is depicted in [Table/Fig-4].

Time interval	Group 1 (Study)	Group 2 (Control)	p-value
30 min prior to induction in preoperative (T1)	85.87±13.821/min	87.70±9.735/min	0.55
Baseline after shifting on table (T2)	88.07±14.142/min	89.00±8.554/min	0.75
Immediate after premedication (T3)	84.60±11.467/min	87.10±8.620/min	0.34
Immediate after priming dose (T4)	86.17±10.422/min	88.10±8.664/min	0.43
2 min after priming (T5)	88.10±10.453/min	87.07±8.777/min	0.68
Immediate after induction (T6)	90.57±11.041/min	99.60±11.085/min	0.002(S)
3 min after induction (T7)	84.37±10.509/min	78.03±8.908/min	0.015(S)

[Table/Fig-4]: Mean Heart Rate (HR) at different time intervals.
Mann's-Whitney U test

At T6, the SBP was significantly higher in Group 1 (120.13±5.37 mmHg) compared to Group 2 (95.53±4.60 mmHg), with a p-value of 0.001. Similarly, at the time interval T7, Group 1 exhibited a significantly higher mean SBP (116.20±3.85 mmHg) compared to Group 2 (90.10±4.60 mmHg), with a p-value of 0.001. The SBP at different time intervals is shown in [Table/Fig-5].

Time interval	Group 1 (Study)	Group 2 (Control)	p-value
30 min prior to induction in preoperative (T1)	139.33±2.40 mmHg	138.80±2.33 mmHg	0.38
Baseline after shifting on table (T2)	137.03±7.35 mmHg	134.40±4.91 mmHg	0.11
Immediate after premedication (T3)	131.87±6.53 mmHg	131.67±8.41 mmHg	0.91
Immediate after priming dose (T4)	128.93±6.16 mmHg	131.03±9.76 mmHg	0.32
2 min after priming (T5)	126.40±5.12 mmHg	127.57±11.33 mmHg	0.61
Immediate after induction (T6)	120.13±5.37 mmHg**	95.53±5.24 mmHg**	0.001(S)
3 min after induction (T7)	116.20±3.85 mmHg**	90.10±4.60 mmHg**	0.001(S)

[Table/Fig-5]: Systolic Blood Pressure (SBP) at different time intervals.
Mann's-Whitney U test

At T6, the DBP was significantly higher in Group 1 (78.73±7.11 mmHg) compared to Group 2 (59.87±7.44 mmHg), with a p-value of 0.001. Similarly, at the time interval T7, Group 1 exhibited a significantly higher mean DBP (77.87±5.73 mmHg) compared to

Group 2 (56.43±8.47 mmHg), also with a p-value of 0.001. The values at different time intervals are shown in [Table/Fig-6].

Time Interval	Group 1 (Study)	Group 2 (Control)	p-value
30 min prior to induction in preoperative (T1)	86.33±5.84 mmHg	87.73±3.70 mmHg	0.27
Baseline after shifting on table (T2)	86.73±8.64 mmHg	86.03±5.95 mmHg	0.71
Immediate after premedication (T3)	84.97±7.99 mmHg	82.03±6.45 mmHg	0.12
Immediate after priming dose (T4)	79.37±7.72 mmHg	81.13±5.41 mmHg	0.30
2 min after priming (T5)	79.53±7.60 mmHg	79.70±4.42 mmHg	0.91
Immediate after induction (T6)	78.73±7.11 mmHg**	59.87±7.44 mmHg**	0.001(S)
3 min after induction (T7)	77.87±5.73 mmHg**	56.43±8.47 mmHg**	0.001(S)

[Table/Fig-6]: Diastolic Blood Pressure (DBP) at different time intervals.
Mann's-Whitney U test

At T6, the mean blood pressure was significantly higher in Group 1 (92.60±5.47 mmHg) compared to Group 2 (71.77±5.28 mmHg), with a p-value of 0.001. Similarly, at the T7 time interval (3 minutes after induction), Group 1 exhibited a significantly higher mean arterial blood pressure (90.63±4.33 mmHg) compared to Group 2 (67.63±5.86 mmHg), with a p-value of 0.001. Mean arterial blood pressure at different time intervals is shown in [Table/Fig-7].

Time interval	Group 1 (Study)	Group 2 (Control)	p-value
30 min prior to induction in preoperative (T1)	104.03±4.05 mmHg	104.80±2.73 mmHg	0.39
Baseline after shifting on table (T2)	103.47±7.26 mmHg	102.20±4.43 mmHg	0.41
Immediate after premedication (T3)	100.67±6.66 mmHg	98.57±6.27 mmHg	0.21
Immediate after priming dose (T4)	95.87±6.07 mmHg	97.73±5.55 mmHg	0.21
2 min after priming (T5)	94.87±5.65 mmHg	95.60±5.06 mmHg	0.59
Immediate after induction (T6)	92.60±5.47 mmHg**	71.77±5.28 mmHg**	0.001(S)
3 min after induction (T7)	90.63±4.33 mmHg**	67.63±5.86 mmHg**	0.001(S)

[Table/Fig-7]: Mean Arterial Pressure (MAP) at different time intervals.
Mann's-Whitney U test

Pain at the injection site was not reported in any patients in Group 1 (study), whereas it was reported in one patient (n=1) in Group 2 (control). Pruritus and anaphylaxis were not reported in either group.

DISCUSSION

In the current study, the Propofol priming principle was assessed in controlled hypertensive patients in terms of total dose, haemodynamic stability, and side-effects. The present study observed a significant decrease in the dose of Propofol when the Propofol priming principle was applied for the induction of general anaesthesia. Consequently, steady haemodynamic parameters were observed compared to the non priming control group. The demographic characteristics, such as age, gender, and BMI, of both groups were found to be comparable, with no significant demographic differences between the study and control groups, indicating a balanced representation of participants across both groups.

In the present study, it was noted that the mean HR at various time points- 30 minutes prior to induction, baseline after shifting to the table, immediately after premedication, and two minutes after priming- did not exhibit significant alterations between both groups. However, there was a notable increase in HR after administering the induction dose of Propofol immediately after induction in the controls, where priming was not used. This increase was significantly higher in the control group immediately after induction (p=0.002) and at three minutes after induction (p=0.01).

The current findings align with those reported by Kumar AA et al., and Dhanapalan SS and Vyas BM [6,12]. Kumar AA et al., observed an increased HR one minute after the administration of the induction dose, which is consistent with the current observation of an immediate rise in HR following Propofol induction [6]. Similarly, Dhanapalan SS et al., reported an increased HR at one and three minutes after induction, with a more pronounced effect observed in the control group compared to the study group [12].

The observed initial rise in HR immediately after administering the induction dose of Propofol, followed by a subsequent decrease at three minutes, can be attributed to the biphasic response associated with Propofol injection. Immediately after induction, there is a decrease in systemic vascular resistance, leading to reflexive increased sympathetic activity. This response is mediated through baroreceptors situated at the carotid sinus and aortic arch, consequently triggering tachycardia. Thereafter, at three minutes after induction, despite the continued decrease in systemic vascular resistance, the HR begins to decrease below baseline levels. This phenomenon occurs due to the resetting of baroreceptor reflexes, where the cardiovascular system adapts to the changes induced by Propofol administration [16].

The SBP exhibited more pronounced decreases in the non priming group compared to the priming group in this study. These findings are similar to the results of Kumar AA et al., who noted that in the control group, SBP dropped more significantly one minute post-induction, immediately after intubation, and five minutes post-intubation compared to the priming group [6]. The mean SBP immediately after induction and at three minutes post-induction with priming exhibited stability when compared to non priming. Significantly, there was an exaggerated drop in SBP without priming compared to with priming, with a p-value of 0.001. The observed fall in blood pressure, particularly in SBP, can indeed be attributed to a decrease in systemic vascular resistance. This mechanism is supported by a study conducted by Pensado A et al., Karlo R et al., also found a significant fall in SBP in the control group. Both studies provide valuable insights into the dose-dependent nature of blood pressure changes induced by Propofol administration [13,17]. In another study conducted by Colson P et al., on hypertensive patients induced with Propofol, it was confirmed that Propofol drastically reduces the response to norepinephrine, angiotensin II, and vasopressin, which is amplified in hypertensive individuals. Therefore, patients on antihypertensive medications should be considered at high risk for haemodynamic instability. The present study population consisted of controlled hypertensive patients, and a similar response was observed [18].

The observed decrease in DBP was significantly less pronounced when the Propofol priming principle was employed, compared to the non priming method, both immediately after induction and three minutes post-induction ($p=0.001$). Similar results were reported by Kumar AA, who found that DBP in the priming group was higher one minute after induction compared to the control group. Likewise, Malinowska-Zaprzalka M et al., also reported findings consistent with the present study in their investigation of hypertensive patients administered Propofol [6,19]. They observed a notable decrease in DBP three minutes after induction, similar to the current study.

In conjunction with the changes in systolic and DBP, the current study revealed a significantly lesser decline in mean blood pressure immediately post-induction and at three minutes post-induction in the priming group compared to the non priming group, with a p-value of 0.001 at both time points. In a study by Larsen JR et al., on the effects of Propofol on myocardial function, it was found that the decreased mean arterial blood pressure induced by Propofol could be attributed to reduced cardiac filling or the direct negative inotropic effects of the drug.

In the current study, despite the more pronounced haemodynamic effects observed in Group 2 (control) compared to Group 1 (study),

it is noteworthy that all patients received concurrent crystalloid infusions and did not require any additional drug interventions to manage their blood pressure [20]. The total drug requirement for Propofol was notably lower when Propofol priming was employed compared to the control group, where priming was not used ($p=0.001$). This significant reduction in drug usage following priming highlights the effectiveness of this technique in minimising drug requirements compared to the control group. Moreover, this finding helps elucidate the more pronounced haemodynamic side-effects observed in the non priming group, as the side-effects associated with Propofol are known to be dose-dependent.

These results align with those of Kumar AA et al., who reported a 27.48% reduction in the induction dose with the application of priming [6]. They suggested that the reduction in the total Propofol dose after priming could be due to the anxiolytic properties at sub-hypnotic doses. Similarly, Karlo R et al., utilised 25% of the calculated dose for priming in their study and found a 10.23% reduction in the total dose of Propofol in the study group, which was significantly less than in the control group [13]. Hamid AH et al., observed a 25% reduction in the total Propofol dose using priming when compared to controls [14]. Likewise, Gvalani SK et al., and Kataria R et al., also observed a significant reduction in the total Propofol dose compared to controls where the Propofol priming principle was not used [15,21]. In line with these findings, a significant reduction of 24.94% in the total dose of Propofol was observed, reinforcing the efficacy of priming in minimising drug requirements during induction.

Adverse effects, such as pain at the injection site, were absent in the priming group. One patient in the control group experienced pain during induction. The overall lower incidence of pain at the injection site (3.33%) can be attributed to the prior administration of injection fentanyl (2 mcg per kg) and the use of a larger vein. These results were similar to those of the study conducted by Kumar AA et al., which also reported a lesser incidence of pain (3%). Notably, neither pruritus nor anaphylaxis was observed in either group [6].

Limitation(s)

The present study was conducted at a single-centre on controlled hypertensive patients; hence, further trials on a larger population with uncontrolled hypertensive patients are required.

CONCLUSION(S)

Recent research has demonstrated that the Propofol priming principle, when used during induction with a controlled rate of administration, significantly reduces the total dose requirement of Propofol. Furthermore, it helps to decrease haemodynamic alterations and the complications associated with Propofol injection. This approach effectively mitigates the dramatic effects of hypotension in hypertensive individuals. Therefore, Propofol priming is strongly advised for hypertensive patients during the induction of general anaesthesia.

REFERENCES

- [1] Cukierman DS, Cata JP, Gan TJ. Enhanced recovery protocols for ambulatory surgery. *Best Pract Res Clin Anaesthesiol.* 2023;37(3):285-303. <http://doi.org/10.1016/j.bpa.2023.04.007>.
- [2] Kulkarni S, Harsoor SS, Chandrasekar M, Bhaskar SB, Bapat J, Ramdas EK, et al. Consensus statement on anaesthesia for day care surgeries. *Indian J Anaesth.* 2017;61(2):110-24. http://doi.org/10.4103/ija.ija_659_16.
- [3] Bailarsing LA, Mhamane RA. A prospective randomized control trial to study effect of priming principle on the induction dose requirements of propofol. *International Journal of Contemporary Medical Research.* 2019;6(10):J15-J20. <http://dx.doi.org/10.21276/ijcmr.2019.6.10.49>.
- [4] Mangano DT. Perioperative cardiac morbidity. *Anesthesiology.* 1990;72(1):153-84. <http://doi.org/10.1097/0000542-199001000-00025>.
- [5] Colson P, Ryckwaert F, Coriat P. Renin angiotensin system antagonists and anaesthesia. *Anesth Analg.* 1999;89(5):1143-55. <http://doi.org/10.1213/0000539-199911000-00012>.

- [6] Kumar AA, Sanikop CS, Kotur PF. Effect of priming principle on the induction dose requirements of propofol- A randomized clinical trial. *Indian J Anaesth.* 2006;50(4):283-87.
- [7] Imran M, Khan FH, Khan MA. Attenuation of hypotension using phenylephrine during induction of anaesthesia with propofol. *J Pak Med Assoc.* 2007;57(11):543-47.
- [8] Amatya A, Marhatta MN, Shrestha GS, Shrestha A, Amatya A. A comparison of midazolam co-induction with propofol priming in propofol induced anaesthesia. *J Nepal Health Res Counc.* 2014;12(26):44-48.
- [9] Djaiani G, Ribes-Pastor MP. Propofol auto-co-induction as an alternative to midazolam co-induction for ambulatory surgery. *Anaesthesia.* 1999;54(1):63-67. <https://doi.org/10.1046/j.1365-2044.1999.00658.x>.
- [10] Saugel B, Bebert EJ, Briesenick L, Hoppe P, Greiwe G, Yang D et al. Mechanisms contributing to hypotension after anesthetic induction with sufentanil, propofol, and rocuronium: A prospective observational study. *J Clin Monit Comput.* 2022;36(2):341-47. <https://doi.org/10.1007/s10877-021-00653-9>.
- [11] Suganya S, Kumar PR, Mohanhariraj A, Jothianand P, Shankar R. Use of priming principle in the induction dose requirement of propofol and its hemodynamic stability- A cross sectional study. *Indian Journal of Clinical Anaesthesia.* 2021;8(3):367-72. <https://doi.org/10.18231/j.ijca.2021.073>.
- [12] Dhanapalan SS, Vyas BM. Efficacy of priming principle in propofol induction-a randomised controlled trial. *J Clin Diagn Res.* 2021;15(3):25-28. <http://doi.org/10.7860/jcdr/2021/47825.14715>.
- [13] Karlo R, Singh NR, Singh KM, Singh TH, Devi NA, Devi MB. Priming effects of propofol during induction of anaesthesia. *J Med Soc.* 2015;29(2):92-95. <http://doi.org/10.4103/0972-4958.163198>.
- [14] Hamid AH, Musad S, Waseem A, Bashir A, Samreen A, Ashraf S. Effect of priming on propofol dose required to induce general anaesthesia. *Journal of Bahria University Medical and Dental College.* 2020;10(1):40-43.
- [15] Gvalani SK, Bhodane SD. Effect of priming principle on the induction dose requirements of propofol in patients undergoing elective surgeries under general anaesthesia. *International Journal of Research in Medical Sciences.* 2016;4(9):3824-28. <https://doi.org/10.18203/2320-6012.ijrms20162787>.
- [16] Fairfield DA, Beale RJ. Haemodynamic effects of propofol induction with 2.5 mg/kg. *Br J Anaesth.* 1991;67:618-20. <http://doi.org/10.1093/bja/67.5.618>
- [17] Pensado A, Molin W, Alvarez J. Hemodynamic changes during anaesthesia induced and maintained with propofol. *Br J Anaesth.* 1993;71:586-88. <http://doi.org/10.1093/bja/60.1.3>.
- [18] Colson P, Saussine M, Séguin JR, Cuchet D, Chaptal P, Roquefeuil B. Hemodynamic effects of anaesthesia in patients chronically treated with angiotensin-converting enzyme inhibitors. *Anesth Analg.* 1992;74(6):805-08.
- [19] Malinowska-Zaprzałka M, Wojewódzka M, Dryl D, Grabowska SZ, Chabielska E. Hemodynamic effect of propofol in enalapril-treated hypertensive patients during induction of general anaesthesia. *Pharmacol Rep.* 2005;57(5):675-78.
- [20] Larsen JR, Torp P, Norrild K, Sloth E. Propofol reduces tissue-Doppler markers of left ventricle function: A transthoracic echocardiographic study. *Br J Anaesth.* 2007;98(2):183-88. <http://doi.org/10.1093/bja/ael345>.
- [21] Kataria R, Singhal A, Prakash S, Singh I. A comparative study of efficacy of propofol auto-co-induction versus midazolam propofol co-induction using the priming principle. *Indian J Anaesth.* 2010;54(6):558-61. <http://doi.org/10.4103/0019-5049.72647>.

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