Original Article

DOI: 10.7860/JCDR/2026/89724.24181

Anaesthesia Section

Comparison of Dexmedetomidine as an Adjuvant to Propofol and Propofol as a Sole Agent in Induction of General Anaesthesia: A Randomised Clinical Trial

JAYAKRISHNA VIJAYAKUMAR[1], TANIA JOSE[2], PK RAJAN BABU[3], GAYATHRI G YEMUNA[4]

ABSTRACT

Introduction: Agents used for induction of anaesthesia often cause vasodilation and suppression of the sympathetic nervous system, leading to a drop in blood pressure. Additionally, laryngoscopy and endotracheal intubation can cause hypertension and tachycardia. So, maintaining haemodynamic stability during induction, intubation and maintenance of anaesthesia is important. Induction of anaesthesia needs sufficient depth of anaesthesia while preventing haemodynamic derangement.

Aim: To study the haemodynamic effects of dexmedetomidine when used as an adjuvant to propofol as compared to propofol as a sole agent in the induction of General Anaesthesia (GA) in patients undergoing elective surgeries under GA.

Materials and Methods: This double-blinded randomised clinical study was conducted at the Department of Anaesthesiology, SUT Academy of Medical Sciences, Trivandrum, Kerala, India, from October 2025 to April 2026. A total of 38 patients were enrolled and randomly assigned to two groups. Group A patients were induced with propofol and vecuronium as muscle relaxants, and in Group B patients were initially given dexmedetomidine before induction with propofol and vecuronium. Heart Rate (HR), Non Invasive Blood Pressure (NIBP) Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP) and Oxygen Saturation (SpO_2) were recorded in each group at pre op, before induction, one minute, five minutes, 10 minutes and 15 minutes post induction. An Independent t-test was used to compare quantitative parameters, Chi-square test for categorical variables and a Mann-Whitney U Test for comparing ordinal parameters.

Results: A total of 38 patients assessed showed no significant demographic differences. Post induction tachycardia (group A-91.11±5.78; group B-79.00±13.25) was significant in group A. There was significant hypotension (MAP, DBP) post induction in group A {MAP 5 min post induction (group A-84.1±11.0, group B-93.1±9.6), DBP 5 min post induction (group A-70.6±9.8; group B-77.8±10)}. Group A required a significantly higher mean dose of propofol (134.2±25.2 mg) compared to group B (101.1±18.8 mg)

Conclusion: Dexmedetomidine as an adjuvant to propofol provides better haemodynamic stability than propofol alone during induction of GA. The total propofol requirement is also significantly lower in the patients when induced along with Dexmedetomidine.

Keywords: α2-adrenergic receptor agonist, Endotracheal intubation, Hypotension, Tachycardia

INTRODUCTION

Intravenous anaesthetic agents used during induction can exert notable cardiovascular effects, including myocardial suppression, peripheral vasodilation, and attenuation of sympathetic tone. These physiological changes often manifest as hypotension, reduced cardiac output, and bradycardia [1]. Tailoring the induction agent dose to the individual patient's physiological condition has been shown to enhance haemodynamic control and minimise abrupt cardiovascular changes [2]. Additionally, combining two agents with complementary profiles can result in reduced dosing requirements for each drug while mitigating their individual haemodynamic drawbacks [3].

Airway instrumentation, particularly during laryngoscopy and endotracheal intubation, activates sensory receptors in the pharyngeal and laryngeal mucosa, triggering a sympathetic response. This response is influenced by the force and duration of airway manipulation and is characterised by increased HR and blood pressure [4]. Although generally tolerated in healthy individuals, this sympathetic surge can pose serious risks for patients with compromised cardiac function or pre-existing co-morbidities. Several pharmacological strategies have been employed to attenuate these haemodynamic perturbations. Technological advancements in airway management have also contributed to minimising stress responses. Techniques such as awake intubation, video laryngoscopy using the GlideScope, and fibreoptic bronchoscopy allow improved visualisation with reduced tissue stimulation, thereby limiting sympathetic activation and associated haemodynamic shifts [5].

Maintaining cardiovascular stability during the phases of induction, intubation, and anaesthetic maintenance is therefore critical. The challenge lies in achieving an adequate depth of anaesthesia while minimising haemodynamic fluctuations. The choice of induction agent plays a pivotal role in reducing complications such as organ hypoperfusion, ischaemia, and the risk of cardiac or respiratory arrest. Among the various induction agents, propofol remains a widely preferred choice in clinical anaesthesia practice [2]. Propofol (2,6-diisopropylphenol) is a non opioid, non barbiturate sedative-hypnotic that facilitates rapid onset of anaesthesia with quick recovery and notable antiemetic effects. Despite its benefits, propofol is known to reduce systemic vascular resistance and arterial pressure without triggering reflex tachycardia. It also diminishes tidal volume and respiratory response to elevated carbon dioxide. Furthermore,

propofol reduces intracranial pressure, cerebral perfusion, and oxygen consumption in the brain [6].

Dexmedetomidine, a highly selective alpha-2 adrenergic receptor agonist, is increasingly used as an adjunct during GA due to its sedative and analgesic effects with minimal impact on respiratory function. This agent is effective in attenuating the haemodynamic responses associated with intubation by lowering circulating catecholamine levels. It also reduces the requirement for anaesthetic drugs and opioids during surgery and blunts the physiological stress response, thereby enhancing the efficacy of intravenous induction agents [7].

Kamali A et al., studied the haemodynamic effects of dexmedetomidine and propofol during intubation which revealed that dexmedetomidine provided more consistent haemodynamic control compared to propofol, which was associated with greater variability in blood pressure and HR following intubation [8]. In a separate randomised clinical trial, Ghomeishi A et al., compared dexmedetomidine and propofol during laparoscopic cholecystectomy, which revealed that dexmedetomidine was associated with lower HR and MAP throughout the perioperative period, while propofol resulted in lower levels of stress hormones [9]. These studies compare the haemodynamic effects of dexmedetomidine and propofol. However, there is a dearth of literature at present that has explored the haemodynamic effects of the use of dexmedetomidine as an adjuvant to propofol [10,11]. The present study focuses on the same and aims to find out whether dexmedetomidine as an adjuvant to propofol provides better haemodynamic stability than propofol alone during induction of GA. The primary objective of the study was to compare the haemodynamic changes occurring as a result of induction with dexmedetomidine as an adjuvant to propofol and propofol as a sole agent in patients undergoing surgeries under GA, and the secondary objective was to compare the amount of propofol required in patients in each group.

MATERIALS AND METHODS

This was a double-blinded randomised clinical trial study conducted on patients who had consented to elective surgeries under GA at the Department of Anaesthesiology, SUT Academy of Medical Sciences, Trivandrum, Kerala, India, from October 2025 to April 2026. The study was approved by the Institutional Ethics Committee of the institute (No.16/IEC/SUTAMS/2023). Informed consent was obtained from the participants in the regional language. No cost was incurred from the participants. Confidentiality was ensured and maintained throughout the study. The study was registered with the Clinical Trial Registry of India (CTRI)-CTRI/2026/05/111472.

Sample size calculation: The sample size for the study was calculated as 38, with 19 patients each in the case and control groups, using the calculation below. Standard deviation of post induction MAP (15 min) in the study was 7.1 as per the study by Kamali A et al., [8]. To detect a difference of 6.5 in post induction MAP between the two groups with a power of 80% and alpha at 5%, the sample size required was 19 in each group.

$$N=\frac{2(Z\alpha+Z\beta)^2\times\sigma^2}{d2}$$

$$=\frac{2\times7.84\times(7.1)^2}{(6.5)2}$$

=19

=19 per group

Total sample size=38

Zα=95% confidence value for z

Zβ=80% power value

σ^2=pooled standard deviation

d^2=mean difference

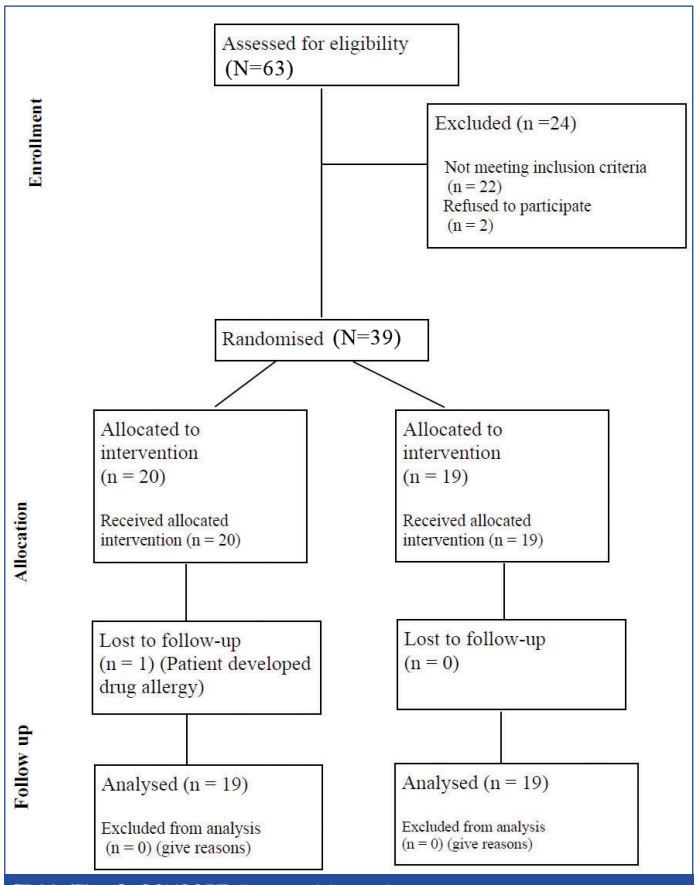
Inclusion criteria: Patients undergoing elective major surgeries posted in the Department of General Surgery, including Thyroid surgeries, Mastectomy under General Anaesthesia (GA), of American Society of Anaesthesiologists (ASA) physical status ASA grades I and II, and age group between 18 and 60, who provided informed consent were included. A total of 39 patients were selected as per the inclusion criteria.

Exclusion criteria: Patients known to have an allergy to propofol or dexmedetomidine or both, those having cardiorespiratory disease, liver diseases and those with bradycardia and hypotension were excluded. A total of 24 patients were excluded as per the exclusion criteria.

Study Procedure

Preoperative vitals were recorded in the form of baseline pulse and BP during the pre-anaesthetic check-up. The patients were kept nil per oral for the last eight hours before surgery. On the day of surgery, informed written consent was obtained. Venous cannulation was done. All patients were given oral pantoprazole 40 mg, oral metoclopramide 10 mg. The i.v. fluids started from 6 am at 2 mL/kg/hr. All patients were premedicated with midazolam i.v. 0.02-0.03 mg/kg, glycopyrrolate i.v. 0.2 mg, and intravenous nalbuphine 0.1 mg/kg. After premedication and preoxygenation, patients were induced with propofol.

An anaesthesiologist in the premedication room created the random number allocation sequence using computer-generated random numbers. The principal investigator enrolled participants into two groups, group A and group B and assigned them to the intervention. The participants, the anaesthesiologist recording the outcome, and the statistician were blinded (double-blinded study). Randomisation and blinding were incorporated to reduce bias. Consolidated Standards of Reporting Trials (CONSORT) diagram of the study is shown in [Table/Fig-1].



[Table/Fig-1]: CONSORT diagram of the study.

In group A induction was done with propofol i.v. 1-2.5 mg/kg and vecuronium i.v. 0.1 mg/kg given as muscle relaxant and in group B patients were given dexmedetomidine i.v. 0.5 mcg/kg

in 100 mL normal saline over 10 minutes, and then induction was done 20 minutes later, with propofol i.v. 1-2.5 mg/kg and vecuronium i.v. 0.1 mg/kg given as muscle relaxant. Dosage was calculated as per standard references [12]. Patients were intubated with appropriate sized endotracheal tube. Electrocardiogram (ECG) was used for continuous cardiac monitoring, Non Invasive Blood Pressure (NIBP) monitor was used for measuring blood pressure at regular intervals, and a pulse oximeter was used for monitoring oxygen saturation levels. Heart Rate (HR), NIBP, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP) and SpO_2 were recorded in each group. The aforementioned parameters were recorded at the following stages: baseline, before induction, one minute, five minutes, 10 minutes and 15 minutes post induction. Anaesthesia was maintained by nitrous oxide in oxygen (50:50) with sevoflurane as the inhalational agent and vecuronium as the muscle relaxant. Patients were mechanically ventilated during the surgery. Intravenous paracetamol 15 mg/kg was used as an analgesic for the surgery intraoperatively in every patient. At the end of surgery, inhalation gases were withdrawn. All patients were given ondansetron 0.1 mg/kg towards the end of the surgery. Neostigmine 0.05 mg/kg with glycopyrrolate 0.008 mg/kg was given for recovery of muscle paralysis. Intravenous paracetamol 15 mg/kg eighth hourly and tramadol 1 mg/kg eighth hourly was given for postoperative analgesia.

The primary outcome measures of the study included HR, SBP, DBP, MAP, and oxygen saturation in the post induction period. The secondary outcome measure was the mean amount of Propofol required for induction of anaesthesia and drug side-effects.

STATISTICAL ANALYSIS

Statistical analyses were performed using a statistical software package, Statistical Package for Social Sciences (SPSS) version 20.0. The data entered in Microsoft Excel and analysed using appropriate software. Categorical and quantitative variables were expressed as frequency (percentage) and mean±SD, respectively. Independent t-test was used to compare quantitative parameters between categories. Chi-square test was used to find association between categorical variables. Mann-Whitney U Test was used to compare non normally distributed parameters between groups. For all statistical interpretations, $p<0.05$ was considered the threshold for statistical significance.

RESULTS

The comparison of demographic characteristics between the two groups is summarised in [Table/Fig-2]. It showed no statistically significant differences in terms of age, gender distribution, body weight, and ASA physical status classification, indicating that both groups were demographically and clinically comparable at baseline.

Baseline haemodynamic parameters such as HR, SBP, DBP, MAP, and SpO_2 showed mild variations in both groups, but with no statistical significance, confirming uniformity prior to induction. However, significant differences emerged following the administration of anaesthetic agents. Group A exhibited a more pronounced decrease in HR, DBP, and MAP at various time intervals post induction when compared to group B, with these differences reaching statistical significance [Table/Fig-3-6]. Though SBP was also reduced in group A, it showed a significant difference only at the 5-minute post induction mark. Throughout the monitoring period, oxygen saturation remained stable and comparable in both groups, suggesting adequate oxygenation despite haemodynamic changes [Table/Fig-7]. No patients included in the study developed any drug side effects.

Factors	Group A n (%)	Group B n (%)	p-value
1. Age group (in years)			
<30	4 (21.1 %)	3 (15.7%)	0.549
30-39	5 (26.3%)	2 (10.5%)	
40-49	6 (31.5%)	8 (42.1%)	
≥50	4 (21.1%)	6 (31.5%)	
2. Gender			
Male	3 (15.8%)	8 (42.1%)	0.074
Female	16 (84.2%)	11 (57.9%)	
3. ASA Grade			
ASA Grade I	14 (73.6%)	9 (47.4%)	0.097
ASA Grade II	5 (26.4%)	10 (52.6%)	
4. Weight	Mean±SD	Mean±SD	
	61.9±11.7	63.2±10.9	0.732
5. Propofol requirement (in mg)	Mean±SD	Mean±SD	
	134.2±25.2	101.1±18.8	0.007

[Table/Fig-2]: Comparison of background characteristics and propofol requirement in Group A and Group B.
ASA: American society of anaesthesiologists

Heart Rate (HR) (beats/minute)	Group A (Mean±SD)	Group B (Mean±SD)	p-value
PAC HR	81.05±10.28	79.74±12.96	0.731
Pre op HR	81.32±12.31	78.74±11.54	0.510
Pre induction HR	81.63±11.45	78.05±10.71	0.326
Post induction 1 min	91.11±5.78	79.00±13.25	0.001*
Post induction 5 min	94.42±6.34	80.47±12.17	< 0.001*
Post induction 10 min	95.42±8.36	81.68±12.03	0.001*
Post induction 15 min	80.79±9.32	80.32±11.48	0.890

[Table/Fig-3]: Heart Rate (HR) variation at different time intervals in Group A and Group B before and after induction of anaesthesia.
p<0.05 is significant; PAC: Pre-Anaesthetic Check-up

Systolic Blood Pressure (SBP) (mmHg)	Group A (Mean±SD)	Group B (Mean±SD)	p-value
PAC	124.3±17.2	124.3±13.5	1.000
Pre-op	126.3±16.8	131.4±11.4	0.280
Pre induction	124.1±15.4	129.2±13	0.273
Post induction 1 minute	112.3±13.1	117.5±15.4	0.264
Post induction 5 minute	110.1±10.8	123.1±11.1	0.0013
Post induction 10 minute	109.9±18.8	115.9±12.7	0.393
Post induction 15 minute	111.8 ±16.3	112.5±24.2	0.919

[Table/Fig-4]: Systolic Blood Pressure (SBP) variation at different time intervals in Group A and Group B before and after induction of anaesthesia.
p < 0.05 is significant

Diastolic Blood Pressure (DBP) (mmHg)	Group A (Mean±SD)	Group B (Mean±SD)	p-value
PAC	75.4±10.9	75.4±7.6	0.986
Pre op	77.5±8.2	80.3±5.9	0.237
Pre induction	74.5±10.8	75.4±7.7	0.966
Post induction 1 minute	68.8±9.9	75.9±11.0	0.044
Post induction 5 minute	70.6±9.8	77.8±10.0	0.031
Post induction 10 minute	68.5±9.2	75.9±11.4	0.035
Post induction 15 minute	68.8±8.1	77.2±10.1	0.008

[Table/Fig-5]: Diastolic Blood Pressure (DBP) variation at different time intervals in Group A and Group B before and after induction of anaesthesia.
*p-value <0.05 is significant

Mean Arterial Pressure (MAP) (mmHg)	Group A (Mean±SD)	Group B (Mean±SD)	p-value
Pre op	93.6±9.6	98.6±7.0	0.073
Pre induction	91.2±10.8	97.9±12.1	0.079

Post induction 1 minute	83.4±11.1	89.4±12.5	0.127
Post induction 5 minute	84.1±11.0	93.1±9.6	0.011
Post induction 10 minute	82.6±10.1	90.1±10.9	0.037
Post induction 15 minute	83.3±10.3	91.0±11.2	0.034

[Table/Fig-6]: Mean Arterial Pressure (MAP) variation at different time intervals in Group A and Group B before and after induction of Anaesthesia.
*p-value < 0.05 is significant

SpO_2 (Percentage)	Group A (Mean±SD)	Group B (Mean±SD)	p-value
Pre op	99.1±1.0	98.6±1.3	0.275
Pre induction	99.6±0.7	99.4±0.8	0.402
Post induction 1 minute	99.6±0.8	99.7±0.6	0.631
Post induction 5 minute	99.8±0.5	99.8±0.4	0.728
Post induction 10 minute	99.7±0.7	99.6±0.8	0.822
Post induction 15 minute	99.8±0.5	99.6±0.8	0.464

[Table/Fig-7]: Oxygen saturation at different time intervals in Group A and Group B before and after induction of anaesthesia; SpO_2: Saturation of peripheral oxygen.
*p-value < 0.05 is significant

DISCUSSION

Intravenous anaesthetic agents used during induction can exert notable cardiovascular effects, including myocardial suppression, peripheral vasodilation, and attenuation of sympathetic tone, which often manifest as hypotension, reduced cardiac output, and bradycardia [1]. Additionally, airway instrumentation, particularly during laryngoscopy and endotracheal intubation, activates sensory receptors in the pharyngeal and laryngeal mucosa, triggering a sympathetic response, characterised by increased HR and blood pressure. Several pharmacological strategies have been employed to attenuate these haemodynamic perturbations. Furthermore, combining two agents with complementary profiles can result in reduced dosing requirements for each drug while mitigating their individual haemodynamic drawbacks [3]. The current study explored the haemodynamic efficacy of the use of dexmedetomidine as an adjuvant to propofol as compared to propofol alone.

The findings in the current study suggest a transient tachycardic response in group A following induction of anaesthesia, which is absent in group B. This could be related to either insufficient attenuation of the sympathetic response or a higher dosage of induction agents. Similar transient elevations in HR post induction have been documented in previous studies evaluating induction with propofol, especially in the absence of adjuncts like opioids or alpha-2 agonists. Dewhirst E et al., studied the haemodynamic effects of propofol following endotracheal intubation and found there was a dose-dependent increase in HR in approximately half of the patients [13]. The current study concurs with the above findings. It was found that the use of dexmedetomidine as an adjuvant necessitated a lower dose of propofol in group B, which might have resulted in the significant increase in HR in group A.

The current study also found a significant decrease in blood pressure in the post induction phase in group A (SBP at 5 min post induction, DBP and MAP at all post induction time points, namely 5, 10 and 15 minutes). These observations suggest that group A patients were more prone to vasodilation and reduced peripheral resistance, which is consistent with known pharmacodynamic properties of agents like propofol, which can cause a dose-dependent drop in vascular resistance and cardiac output. Nega MH et al., conducted a prospective study and studied factors causing post induction hypotension and found that propofol is an independent risk factor for the same [14]. Li B et al., studied the haemodynamic effects of propofol, which reiterates the risk of hypotension [15].

Group A required a significantly higher mean dose of propofol (134.2±25.2 mg) compared to Group B (101.1±18.8 mg), with the difference being highly significant (p<0.01). This has profound implications on both haemodynamic stability and anaesthetic depth. Higher propofol dosages have been linked with greater cardiovascular depression, explaining the more pronounced hypotension and bradycardia observed in group A [14]. Group B's lower requirement suggests either a synergistic effect from premedication or adjuncts or inherent pharmacodynamic modulation that warrants further investigation.

Multiple studies have studied the efficacy of Dexmedetomidine in the induction of anaesthesia and found the stable haemodynamic profile of the drug. The current study concurs with the current literature on that front. Kamali A et al., conducted a clinical trial comparing the efficacy of dexmedetomidine and propofol in attenuating haemodynamic fluctuations following endotracheal intubation and found that dexmedetomidine resulted in greater haemodynamic stability post intubation compared to propofol [8]. Similarly, Sheikh TA et al., studied 60 cardiac surgery patients and evaluated the effects of dexmedetomidine and propofol on intraoperative haemodynamics and postoperative outcomes which demonstrated that patients in the dexmedetomidine group had significantly lower HR and MAP, shorter ICU stays, and reduced postoperative delirium [16]. In a separate study, Ghomeishi A et al., compared dexmedetomidine and propofol during laparoscopic cholecystectomy and found out that dexmedetomidine offers superior haemodynamic control, whereas propofol might better attenuate stress responses [9]. Keshri RK et al., evaluated different doses of dexmedetomidine in geriatric patients undergoing spinal surgery and concluded that dexmedetomidine groups exhibited significantly lower HR and MAP post intubation compared to the control group, and the 0.5 µg/kg dose provided adequate haemodynamic control with fewer adverse effects [12]. Xiong J et al., assessed the effectiveness of oral dexmedetomidine as a premedication in patients undergoing elective neurosurgery and reported significant reductions in HR and MAP at various time intervals following tracheal intubation in the dexmedetomidine group. Moreover, cortisol and ACTH levels were significantly lower post intubation in dexmedetomidine group [17]. Chaitanya G et al., conducted a study to evaluate the haemodynamic responses associated with dexmedetomidine infusion among the 40 patients who underwent surgery for drug-resistant epilepsy, and it was found that 87.5% experienced a transient decline in HR and 62.5% showed an increase in MAP, with both trends gradually returning to baseline within 10 minutes post infusion [18]. De Cassai A et al., performed a meta-analysis to evaluate the effectiveness of intravenous dexmedetomidine in reducing sympathetic responses during tracheal intubation and found that the dexmedetomidine group showed significantly lower SBP, MAP, and HR during laryngoscopy [19]. Zhai W et al., conducted a study to evaluate the impact of dexmedetomidine on haemodynamic stability and inflammatory response in 300 patients undergoing off-pump coronary artery bypass grafting. Results showed that patients who received dexmedetomidine had significantly lower HRs and MAPs than those in the control group [20]. Tanskanen PE et al., conducted a study on 54 patients undergoing brain tumor surgery to assess the haemodynamic effects of dexmedetomidine infusion. Results showed that dexmedetomidine infusion shortened extubation time, with the group demonstrating the greatest blood pressure stability, maintaining systolic values within ±20% of the intraoperative mean for 85% of the time. It also reduced tachycardic response to intubation and hypertensive response to extubation (p<0.01), and improved HR variability [21].

Multiple studies have explored the utility of combination of drugs in induction of anaesthesia. Hosseinzadeh H et al., conducted a study to compare the haemodynamic responses of two anaesthetic combinations- ketamine with propofol (ketofol) and etomidate with propofol (etofol)- during the induction of anaesthesia, which suggested that both ketamine with propofol and etomidate with propofol regimens are effective in preserving haemodynamic stability during anaesthesia induction and mitigate the cardiovascular

depressant effects typically associated with propofol [22]. Jakob SM et al., performed two large, multicentre, double-blind, randomised phase 3 trials- MIDEX and PRODEX- to compare dexmedetomidine with midazolam and propofol, respectively, in ICU patients requiring long-term sedation. In both studies, dexmedetomidine maintained target sedation levels more effectively and reduced the duration of mechanical ventilation. It also allowed better communication regarding pain. However, it was associated with more side effects compared to propofol or midazolam [23]. Patel CR et al., conducted a randomised trial to investigate the impact of continuous dexmedetomidine infusion, without the use of opioids, on sevoflurane requirements during GA, with anaesthesia depth monitored through entropy analysis and concluded that dexmedetomidine, when used as an adjunct, lowers the sevoflurane requirement needed to maintain adequate anaesthetic depth [24]. These studies however, do not explore the haemodynamic profile of Dexmedetomidine on its use as a combination drug for induction of anaesthesia.

Multiple studies demonstrate that dexmedetomidine not only contributes to stable intraoperative haemodynamics but also provides benefits in terms of sedation quality, reduced stress response, and improved recovery outcomes, particularly in high-risk and elderly populations [8,16-19]. Propofol has its advantages; however, combining propofol with an adjuvant mitigates the adverse effects of the drug. There are multiple studies which have explored the use of multiple drugs like ketamine or etomidate with propofol. However, there is a dearth of literature at present which explore haemodynamic effects of the use of dexmedetomidine as an adjuvant to propofol [10,11]. The current study explores this area and concludes that this combination offers the best of two worlds.

Limitation(s)

While the present study shows evidence that dexmedetomidine as premedication prior to induction with propofol provides better haemodynamic stability than propofol alone, certain limitations ought to be acknowledged. The current study does not shed light on the utility of dexmedetomidine in physiological stress response, depth of anaesthesia and pain relief. Comparative studies in paediatric and high-risk populations were also not performed in the current study. Addressing these limitations and building on the current study can contribute to a more comprehensive understanding of the clinical utility and safety of dexmedetomidine in anaesthesia.

CONCLUSION(S)

Dexmedetomidine as an adjuvant to propofol provides better haemodynamic stability than propofol alone during induction of GA. The combination causes no significant tachycardia or hypotension post induction unlike the use of Propofol as a sole agent. Use of dexmedetomidine as an adjuvant helps in reducing haemodynamic variations caused by propofol. The total propofol requirement is also significantly lower in the patients when induced along with dexmedetomidine.

REFERENCES

[1] Butterworth JF, Mackey DC, Wasnick JD. (Eds.). (2022). Morgan & Mikhail's clinical anesthesiology (7th ed.). McGraw Hill.

[2] Miller RD, Cohen NH, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL. (2020). Miller's Anesthesia (9th ed.). Elsevier.

[3] Raman V, Segaran S, Ramyavel T, George SK, Zachariah M. Comparison of haemodynamic changes between propofol and ketofol as induction agents in patients undergoing laparoscopic surgeries under general anaesthesia. J Anaesthesiol Clin Pharmacol. 2022;38(2):215-20. Doi: 10.4103/joacp.JOACP_251_20. Epub 2022 Jan 13. PMID: 36171936; PMCID: PMC9511839.

[4] Kanumetta R, Vishal PA, Sri Soumya R, Vangapandu TD, Kotturu DV, Sayana SB. Evaluation of cardiovascular responses to endotracheal intubation with alkalinized lignocaine and air cuff inflation techniques. Cureus. 2025;17(8):e89734. Doi: 10.7759/cureus.89734. PMID: 40932979; PMCID: PMC12419491.

[5] Avva U, Lata JM, Hendrix JM, Kiel J. Airway Management. [Updated 2025 Jan 19]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470403/.

[6] Vuyk J, Sitsen E, Reekers M. Intravenous anesthetics. In: Miller RD, editor. Miller's Anesthesia. 9th ed. Elsevier; 2020. p. 821-63.

[7] Tufanogullari B, White PF, Peixoto MP, Kianpour D, Lacour T, Griffin J, et al. Dexmedetomidine infusion during laparoscopic bariatric surgery: The effect on recovery outcome variables. Anesth Analg. 2008;106(6):1741-48. Doi: 10.1213/ane.0b013e318172c47c.

[8] Kamali A, Taghizadeh M, Esfandiar M, Akhtari AS. A Comparison of the effects of dexmedetomidine and propofol in controlling the hemodynamic responses after intubation: A double-blind, randomized, clinical trial study. Open Access Maced J Med Sci. 2018;6(11):2045-50. [cited 2025 Apr. 10]. Available from: https://oamjms.eu/index.php/mjms/article/view/oamjms.2018.385.

[9] Ghomeishi A, Mohtadi AR, Behaeen K, Nesioonpour S, Bakhtiari N, Khalvati Fahlyani F. Comparison of the effect of propofol and dexmedetomidine on hemodynamic parameters and stress response hormones during laparoscopic cholecystectomy surgery. Anesth Pain Med. 2021;11(5):e119446. Doi: 10.5812/aapm.119446. PMID: 35075417; PMCID: PMC8782195.

[10] Khare A, Sharma SP, Deganwa ML, Sharma M, Gill N. Effects of dexmedetomidine on intraoperative hemodynamics and propofol requirement in patients undergoing laparoscopic cholecystectomy. Anesth Essays Res. 2017;11(4):1040-45. Doi: 10.4103/aer.AER_107_17. PMID: 29284872; PMCID: PMC5735447.

[11] Gavali AS, Ashok MA. Comparison of hemodynamic effects of dexmedetomidine versus propofol for sedation. Journal of Contemporary Clinical Practice. 2025;11(6):137-43.

[12] Keshri RK, Prasad MK, Choudhary AK, Jheetay GS, Singh Y, Kapoor K. Comparative evaluation of different doses of intravenous dexmedetomidine on hemodynamic response during laryngoscopy and endotracheal intubation in geriatric patients undergoing spine surgeries: A prospective, double-blind study. Anesth Essays Res. 2018;12(4):897-902. Doi: 10.4103/aer.AER_156_18. PMID: 30662127; PMCID: PMC6319078.

[13] Dewhirst E, Lancaster C, Tobias JD. Hemodynamic changes following the administration of propofol to facilitate endotracheal intubation during sevoflurane anesthesia. Int J Clin Exp Med. 2013;6(1):26-29. Epub 2012 Nov 18. PMID: 23236555; PMCID: PMC3515968.

[14] Nega MH, Ahmed SA, Tawuye HY, Mustofa SY. Incidence and factors associated with post-induction hypotension among adult surgical patients: Prospective follow-up study, International Journal of Surgery Open. 2022;49:100565. ISSN 2405-8572. https://doi.org/10.1016/j.ijso.2022.100565. (https://www.sciencedirect.com/science/article/pii/S2405857222001280).

[15] Li B, Pop C, Johnson M, Sklar MC, Lawler PR. Hemodynamic effects of propofol. JACC Adv. 2025;4(8):101769. Available from: https://doi.org/10.1016/j.jacadv.2025.101769.

[16] Sheikh TA, Dar BA, Akhter N, Ahmad N. A comparative study evaluating effects of intravenous sedation by dexmedetomidine and propofol on patient hemodynamics and postoperative outcomes in cardiac surgery. Anesth Essays Res. 2018;12(2):555-60. Doi: 10.4103/aer.AER_46_18. PMID: 29962634; PMCID: PMC6020583.

[17] Xiong J, Gao J, Pang Y, Zhou Y, Sun Y, Sun Y. Dexmedetomidine premedication increases preoperative sedation and inhibits stress induced by tracheal intubation in adult: A prospective randomized double-blind clinical study. BMC Anesthesiol. 2022;22(1):398. Doi: 10.1186/s12871-022-01930-z. PMID: 36544098; PMCID: PMC9768986.

[18] Chaitanya G, Arivazhagan A, Sinha S, Madhusudan Reddy KR, Thennarasu K, Bharath RD, et al. Hemodynamic effects of dexmedetomidine during intra-operative electrocorticography for epilepsy surgery. J Neurosci Rural Pract. 2014;5(Suppl 1):S17-S21. Doi: 10.4103/0976-3147.145195. PMID: 25540532; PMCID: PMC4271375.

[19] De Cassai A, Boscolo A, Geraldini F, Zarantonello F, Pettenuzzo T, Pasin L, et al. Effect of dexmedetomidine on hemodynamic responses to tracheal intubation: A meta-analysis with meta-regression and trial sequential analysis. J Clin Anesth. 2021;72:110287. Doi: 10.1016/j.jclinane.2021.110287. Epub 2021 Apr 16. PMID: 33873003.

[20] Zhai W, Yang L, Sun P, Li Y, Han J, Wang G. Effect of dexmedetomidine on hemodynamic changes and inflammatory responses in patients undergoing off-pump coronary-artery bypass grafting. Exp Ther Med. 2020;20(6):250. Doi: 10.3892/etm.2020.9380. Epub 2020 Oct 23. PMID: 33178348; PMCID: PMC7651881.

[21] Tanskanen PE, Kyttä JV, Randell TT, Aantaa RE. Dexmedetomidine as an anaesthetic adjuvant in patients undergoing intracranial tumour surgery: A double-blind, randomized and placebo-controlled study. Br J Anaesth. 2006;97(5):658-65. Doi: 10.1093/bja/ael220. Epub 2006 Aug 16. Erratum in: Br J Anaesth. 2006;97(6):908. PMID: 16914460.

[22] Hosseinzadeh H, Eidy M, Golzari SE, Vasebi M. Hemodynamic stability during induction of anesthesia in elderly patients: Propofol + Ketamine versus Propofol + Etomidate. J Cardiovasc Thorac Res. 2013;5(2):51-54. Doi: 10.5681/jcvtr.2013.011. Epub 2013 Jun 27. PMID: 24251011; PMCID: PMC3825382.

[23] Jakob SM, Ruokonen E, Grounds RM, Sarapohja T, Garratt C, Pocock SJ, et al; Dexmedetomidine for long-term sedation investigators. Dexmedetomidine vs midazolam or propofol for sedation during prolonged mechanical ventilation: Two randomized controlled trials. JAMA. 2012;307(11):1151-60. Doi: 10.1001/jama.2012.304. PMID: 22436955.

[24] Patel CR, Engineer SR, Shah BJ, Madhu S. The effect of dexmedetomidine continuous infusion as an adjuvant to general anesthesia on sevoflurane requirements: A study based on entropy analysis. J Anaesthesiol Clin Pharmacol. 2013;29(3):318-22. Doi: 10.4103/0970-9185.117066. PMID: 24106354; PMCID: PMC3788228.

PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Anaesthesiology, SUT Academy of Medical Sciences, Trivandrum, Kerala, India.
2. Assistant Professor, Department of Anaesthesiology, SUT Academy of Medical Sciences, Trivandrum, Kerala, India.
3. Professor and Head, Department of Anaesthesiology, SUT Academy of Medical Sciences, Trivandrum, Kerala, India.
4. Assistant Professor, Department of Anaesthesiology, SUT Academy of Medical Sciences, Trivandrum, Kerala, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Tania Jose,
Assistant Professor, Department of Anaesthesiology, SUT Academy of Medical Sciences, Vencode, Vattapara, Trivandrum-695028, Kerala, India.
E-mail: anandbinumangal@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Apr 30, 2026
- Manual Googling: Jul 06, 2026
- iThenticate Software: Jul 08, 2026 (7%)

ETYMOLOGY: Author Origin

EMENDATIONS: 8

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Apr 25, 2026**
Date of Peer Review: **May 29, 2026**
Date of Acceptance: **Jul 10, 2026**
Date of Publishing: **Aug 01, 2026**