ARC Journal of Anesthesiology
Volume 10, Issue 1, 2025, PP 1-6
ISSN 2455-9792
DOI: https://doi.org/10.20431/2455-9792.1001001
www.arcjournals.org

Assessment of Hemodynamic Changes during Propofol and Ketamine Induction in General Anesthesia

Dr. Md. Shahadat Hossain[1*], Dr. Shamim Ara Sultana[2], Dr. Md. Mahbubul Alam Sarker[3], Dr. Mohammed Badrul Alam[4], Dr. Mehedi Masud[5], Dr. Md. HassanulAlam[6], Prof. Dr. Md. Mustafa Kamal[7]

[1,2,3,5]*Assistant Professor, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.*

[4,6]*Assistant Professor, Department of Anesthesiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.*

[7]*Professor, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.*

Received: 30 December 2024 Accepted: 15 January 2025 Published: 20 January 2025

Corresponding Author:** ***Dr. Md. Shahadat Hossain, *Assistant Professor, Department of Anaesthesia, Analgesia and Intensive Care Medicine, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.*

Abstract

Background: *One of the primary objectives during general anesthesia is to maintain hemodynamic stability, as fluctuations in heart rate, blood pressure, and other vital signs can pose significant risks, particularly in patients with preexisting health conditions. This study aims to evaluate the hemodynamic changes induced by propofol and ketamine during anesthesia induction.*

Aim of the Study: *The aim of the study was to assess the hemodynamic changes induced by propofol and ketamine during the induction of general anesthesia.*

Methods: *This prospective, observational study, conducted from July 2022 to June 2023 in the Department of Anesthesiology at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, included 60 patients who were randomly assigned to receive either ketamine or propofol for anesthesia induction. Hemodynamic changes, including heart rate (HR) and mean arterial pressure (MAP), were monitored. Data on demographics, ASA classification, and adverse events were collected and analyzed using SPSS version 22.0, with significance set at $p < 0.05$.*

Results: *The study included 60 participants, equally divided between the Ketamine and Propofol groups. Heart rate decreases (>10 bpm) were more frequent in the Ketamine group (66.7% vs. 33.3%, $p = 0.01$), while MAP decreases (>15%) were also higher in the Ketamine group (41.7% vs. 16.7%, $p = 0.01$). Hypotension occurred more commonly in the Ketamine group (83.3% vs. 33.3%, $p = 0.001$). Nausea and vomiting were similar between groups ($p = 0.65$).*

Conclusion: *Propofol may offer a more stable hemodynamic profile than ketamine during the induction of general anesthesia.*

Keywords: *Hemodynamics, Propofol, Ketamine, Anesthesia induction, Cardiovascular stability.*

1. Introduction

Anesthesia is a medically induced, temporary loss of sensation or awareness, facilitating procedures that would otherwise cause significant pain or discomfort. It often includes elements such as amnesia, paralysis, and unconsciousness, ensuring patient comfort and safety during surgery.[1] Among commonly used induction agents, propofol stands out as a rapidly acting intravenous hypnotic that induces general anesthesia by enhancing inhibitory neurotransmission through GABA receptors.[2] Its rapid onset and short duration of action make it a preferred choice, though its use is tempered by dose-dependent hypotension and respiratory depression.[3,4] Propofol's cardiovascular effects, including reduced arterial blood pressure due to decreased preload, cardiac contractility, and

systemic vascular resistance, contrast sharply with the transient hypertension induced by laryngoscopy and tracheal intubation.[5,6,7,8] These hemodynamic fluctuations are of particular concern in high-risk patients, such as the elderly or those with comorbidities, as the hypotensive effects of propofol are more pronounced in these populations, necessitating careful dose management.[9]

Ketamine, another commonly used induction agent, complements propofol with its distinct pharmacological properties.[10] It induces "dissociative anesthesia," characterized by the preservation of respiratory drive and an increase in heart rate and blood pressure due to its sympathomimetic effects, making it an excellent option for patients at risk of hypotension, such as those with compromised cardiac function. However, ketamine's use as a standalone agent is often limited by its psychomimetic effects, including hallucinations and agitation.[11] The combination of ketamine and propofol, or "ketofol," has become a viable strategy to overcome these restrictions. Ketofol offers a synergistic balance by mitigating the dose-dependent hypotension caused by propofol while maintaining stable hemodynamics and providing adequate anesthesia. This combination is particularly advantageous in procedures like laparoscopic surgeries, where physiological changes due to pneumoperitoneum can challenge hemodynamic stability, highlighting ketofol's value in enhancing patient safety and surgical outcomes.[12,13]

One of the primary objectives during general anesthesia is to maintain hemodynamic stability.[14] Propofol induction, while effective, often leads to hemodynamic changes that can cause transient cardiovascular instability.[15] These fluctuations, although short-lived, pose significant risks, especially in patients with preexisting cardiovascular or cerebrovascular conditions.[16] To minimize such risks, strategies to manage these hemodynamic changes are essential. One of the current approaches to enhance anesthesia safety is the use of balanced anesthesia, which involves the combination of two or more anesthetic agents. This technique helps to maximize the benefits of each agent while reducing their individual side effects. The ultimate goal is to ensure optimal cardiovascular homeostasis,[14] preventing dangerous fluctuations in heart rate, blood pressure, and other vital signs, particularly in critically ill patients or those with underlying health issues.[17] Achieving this balance is crucial to the safe management of anesthesia and the prevention of complications during surgery.

However, despite advancements in anesthetic devices and medications, reliable guidelines for determining the optimal propofol dose during anesthesia induction are still lacking, with the decision largely left to the anesthesiologist's clinical judgment.[18] While there have been improvements in anesthesia techniques, intraoperative hemodynamic fluctuations remain a common occurrence, signaling the need for better stabilization strategies. One such approach, co-induction, which involves the use of combined anesthesia agents, has been suggested for patients with poor hemodynamic stability. By using lower doses of intravenous anesthetics, this method may reduce the side effects typically associated with higher doses of individual agents. Nevertheless, there is still a significant gap in the available research, and further studies are needed to establish clear, evidence-based protocols and guidelines that can better address these challenges and optimize patient safety and outcomes during surgery. The purpose of the study was to evaluate the hemodynamic changes induced by propofol and ketamine during the induction of general anesthesia.

2. Objective

- The aim of the study was to assess the hemodynamic changes induced by propofol and ketamine during the induction of general anesthesia.

3. Methodology & Materials

This prospective, observational study was conducted in the Department of Anesthesiology at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, from July 2022 to June 2023. A total of 60 patients undergoing general anesthesia were included in the study, with 30 patients in the propofol group and 30 patients in the ketamine group, comparing the hemodynamic changes (heart rate and mean arterial pressure) during induction of anesthesia with both agents.

Inclusion Criteria

- Adults aged 18 years and older.
- Patients scheduled for elective surgery requiring general anesthesia.
- Individuals classified as ASA I or ASA II.
- Patients who provided written informed consent for participation in the study.

Exclusion Criteria

- Patients with contraindications to general anesthesia, propofol, or ketamine.
- Individuals with pre-existing cardiovascular, neurological, or other systemic disorders affecting hemodynamic stability.
- Patients who declined participation or had incomplete medical records.

Written informed consent was obtained from all participants to ensure confidentiality and ethical compliance. Preoperative assessments included medical history, physical examination, and baseline vital signs, with ASA classification recorded for stratification. Patients were randomly assigned to receive either 2–2.5 mg/kg intravenous ketamine (Ketamine group, n = 30) or propofol (Propofol group, n = 30) for anesthesia induction. Standard anesthesia protocols were followed, with continuous monitoring of heart rate (HR), mean arterial pressure (MAP), and oxygen saturation. HR and MAP changes were categorized into specific ranges, and adverse events, including hypotension, bradycardia, and nausea/vomiting, were documented. Data were analyzed using SPSS version 22.0, with descriptive statistics and appropriate tests. A p-value of <0.05 was considered statistically significant for all comparisons. Postoperative evaluations assessed adverse events, recovery, and clinical outcomes. The primary outcomes were hemodynamic changes (HR and MAP), with secondary outcomes focusing on adverse events.

4. Results

Table 1. *Demographic Characteristics of the Study Participants (n=60)*

Variable		Ketamine Group (n = 30)	Propofol Group (n =30)	Total (n = 60)
Age Groups	18–35 years	7 (23.3%)	7 (23.3%)	14 (23.3%)
	36–53 years	14 (46.7%)	14 (46.7%)	28 (46.7%)
	54–70 years	9 (30.0%)	9 (30.0%)	18 (30.0%)
	Mean Age (years)	45.3 ± 11.9	45.7 ± 12.3	45.5 ± 12.1
Gender	Male	14 (46.7%)	14 (46.7%)	28 (46.7%)
	Female	16 (53.3%)	16 (53.3%)	32 (53.3%)
ASA Classification	ASA I	7 (23.3%)	9 (30.0%)	16 (26.7%)
	ASA II	23 (76.7%)	21 (70.0%)	44 (73.3%)

Table 1 summarizes the demographic characteristics and ASA classification of the participants in the Ketamine and Propofol groups (n = 30 each). The mean age of the participants was 45.5 ± 12.1 years, with 23.3% aged 18–35 years, 46.7% aged 36–53 years, and 30.0% aged 54–70 years. Gender distribution was balanced across both groups, with 28 males (46.7%) and 32 females (53.3%). Regarding ASA classification, 16 participants (26.7%) were classified as ASA I, and 44 participants (73.3%) as ASA II.

Table 2. *Heart Rate Changes in the Study Participants (n=60)*

HR Change	Ketamine Group (n = 30)	Propofol Group (n = 30)	p-value
Decrease (>10 bpm)	20 (66.7%)	10 (33.3%)	0.01
Increase (>10 bpm)	8 (26.7%)	2 (6.7%)	0.03
No Significant Change	2 (6.7%)	18 (60.0%)	0.20

Table 2 summarizes the heart rate (HR) changes observed in the Ketamine and Propofol groups (n = 30 each). A decrease in HR (>10 bpm) was more common in the Ketamine group, with 20 participants (66.7%) compared to 10 participants (33.3%) in the Propofol group (p = 0.01). Conversely, an increase in HR (>10 bpm) was observed in 8 participants (26.7%) in the Ketamine group and 2 participants (6.7%) in the Propofol group (p = 0.03). No significant HR change was noted in 2 participants (6.7%) from the Ketamine group, whereas 18 participants (60.0%) in the Propofol group showed no significant change (p = 0.2).

Table 3. *Mean Arterial Pressure (MAP) Changes in the Study Participants (n=60)*

Timepoint	Ketamine Group (n = 30)	Propofol Group (n = 30)	p-value
MAP Decrease (>15%)	25 (41.7%)	10 (16.7%)	0.01
MAP Increase (>5%)	15 (25.0%)	3 (5.0%)	0.01
No Change (<5%)	20 (33.3%)	40 (66.7%)	0.20

Table 3 summarizes the changes in Mean Arterial Pressure (MAP) observed in the Ketamine and Propofol groups (n = 30 each). A decrease in MAP of more than 15% was more common in the Ketamine group, with 25 participants (41.7%) compared to 10 participants (16.7%) in the Propofol group (p = 0.01). Similarly, an increase in MAP of more than 5% was observed in 15 participants (25.0%) in the Ketamine group and 3 participants (5.0%) in the Propofol group (p = 0.01). No significant MAP change (<5%) was noted in 20 participants (33.3%) from the Ketamine group, while 40 participants (66.7%) in the Propofol group showed no significant change (p = 0.2).

Table 4. *Adverse Events Observed in the Study Participants (n=60)*

Adverse Event	Ketamine Group (n = 30)	Propofol Group (n = 30)	p-value
Hypotension	25 (83.3%)	10 (33.3%)	0.001
Bradycardia	16 (53.3%)	8 (26.7%)	0.03
Nausea/Vomiting	2 (6.7%)	3 (10.0%)	0.65

This table summarizes the adverse events observed in the Ketamine and Propofol groups (n = 30 each). Hypotension was significantly more common in the Ketamine group, occurring in 25 participants (83.3%) compared to 10 participants (33.3%) in the Propofol group (p = 0.001). Bradycardia was also more frequent in the Ketamine group, affecting 16 participants (53.3%) versus 8 participants (26.7%) in the Propofol group (p = 0.03). Conversely, nausea and vomiting were comparable between the groups, observed in 2 participants (6.7%) in the Ketamine group and 3 participants (10.0%) in the Propofol group, with no statistically significant difference (p = 0.65).

5. Discussion

This study, conducted at the Department of Anesthesiology at Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh, aimed to assess the hemodynamic changes induced by propofol and ketamine during the induction of general anesthesia. The results highlight the distinct cardiovascular responses associated with each agent, focusing on heart rate (HR) and mean arterial pressure (MAP) changes, as well as adverse events such as hypotension, bradycardia, and nausea/ vomiting. These findings provide valuable insights into the hemodynamic profile of propofol and ketamine, emphasizing the clinical significance of choosing the appropriate anesthetic agent to ensure patient safety and stability during anesthesia induction.

In our study, participants had a mean age of 45.5 ± 12.1 years, consistent with findings by Ali et al. regarding anesthetic induction. The majority (46.7%) were aged 36–53 years, representing a typical adult surgical population. Gender distribution was nearly balanced, with 46.7% males and 53.3% females, similar to Ali et al.'s[19] observations. Regarding ASA classification, 26.7% were ASA I and 73.3% ASA II, aligning with Sanuki et al.'s[20] findings. This predominance of ASA II patients, indicating mild systemic conditions, emphasizes the importance of selecting anesthetic agents that maintain hemodynamic stability.

These demographic characteristics support the generalizability of our findings in clinical practice. In our study, heart rate (HR) changes differed between the Ketamine and Propofol groups, similar to findings by Iuliana et al.[21] A decrease in HR (>10 bpm) was more common in the Ketamine group (66.7%) compared to the Propofol group (33.3%) (p = 0.01), reflecting Ketamine's sympathomimetic effects. On the other hand, an increase in HR (>10 bpm) was more frequently observed in the Ketamine group (26.7%) than the Propofol group (6.7%) (p = 0.03). Notably, 60.0% of participants in the Propofol group showed no significant HR change, compared to just 6.7% in the Ketamine group (p = 0.2). These findings underline the contrasting cardiovascular effects of Propofol and Ketamine, with Propofol causing more pronounced HR changes and Ketamine helping to maintain HR stability.

In our study, we observed notable differences in mean arterial pressure (MAP) changes between the Ketamine and Propofol groups. A decrease in MAP of more than 15% was more common in the Propofol group (41.7%) compared to the Ketamine group (16.7%) (p = 0.01), highlighting Propofol's depressant effects on the cardiovascular system. Conversely, the Ketamine group showed a higher incidence of MAP increase (>5%) in 25.0% of participants compared to 5.0% in the Propofol group (p = 0.01). Additionally, a higher percentage of participants in the Ketamine group (66.7%)

showed no significant MAP change, whereas 33.3% of participants in the Propofol group experienced no significant MAP change (p = 0.2). These findings are consistent with the results of Abbasivash et al.[22], who highlighted the differing hemodynamic effects of Propofol and Ketamine. Propofol induced more significant decreases in MAP, while Ketamine maintained more stable MAP levels, reflecting its sympathomimetic properties.

In our study, significant differences in adverse events were observed between the Ketamine and Propofol groups, aligning with findings by Iuliana et al.[21] Hypotension was notably more frequent in the Ketamine group (83.3%) compared to the Propofol group (33.3%) (p = 0.001), reflecting the more pronounced vasodilatory effects of Ketamine. Bradycardia was also more common in the Ketamine group (53.3%) than the Propofol group (26.7%) (p = 0.03), consistent with Ketamine's sympathomimetic properties. Nausea and vomiting were similar across both groups, with no statistically significant difference (p = 0.65). These findings underscore the contrasting hemodynamic effects of the two anesthetic agents, with Ketamine showing more frequent hypotension and bradycardia, while both drugs exhibited comparable tolerability in terms of gastrointestinal side effects.

6. Limitations of the Study

This study had some limitations:

- The study was conducted in a selected tertiary-level hospital.
- The sample was not randomly selected.
- The study's limited geographic scope may introduce sample bias, potentially affecting the broader applicability of the findings.

7. Conclusion

The study evaluated hemodynamic changes during the induction of general anesthesia with propofol and ketamine in participants with a mean age of 45 years and a balanced gender distribution. Ketamine was associated with a higher incidence of heart rate and MAP decreases, as well as hypotension, compared to propofol. Both drugs had comparable rates of nausea and vomiting. These findings indicate that propofol may provide greater hemodynamic stability than ketamine during anesthesia induction.

References

[1] Bonhomme V, Jeanne M, Boselli E, Gruenewald M, Logier R, Richebé P. Physiological signal processing for individualized anti-nociception management during general anesthesia: a review. Yearbook of medical informatics. 2015;24(01):95-101.review. Yearbook of medical informatics. 2015;24(01):95-101.

[2] Sahinovic MM, Struys MM, Absalom AR. Clinical pharmacokinetics and pharmacodynamics of propofol. Clinical pharmacokinetics. 2018 Dec; 57(12):1539-58.

[3] Sahinovic MM, Struys MM, Absalom AR. Clinical pharmacokinetics and pharmacodynamics of propofol. Clinical pharmacokinetics. 2018 Dec; 57(12):1539-58.

[4] Bassett KE, Anderson JL, Pribble CG, Guenther E. Propofol for procedural sedation in children in the emergency department. Annals of emergency medicine. 2003 Dec 1;42(6):773-82.

[5] Russell WJ, Morris RG, Frewin DB, Drew SE. Changes in Plasma Catecholamine Concentrations during Endotracheal Intubation. Survey of Anesthesiology. 1982 Oct 1;26(5): 276-7.

[6] Rajbhandari PK. Lignocaine and Esmolol on Stress Response to Laryngoscopy and Intubation. Journal of the Nepal Medical Association. 2014 Apr 1;52(194).

[7] Forbes AM, Dally FG. Acute hypertension during induction of anaesthesia and endotracheal intubation in normotensive man. BJA: British Journal of Anaesthesia. 1970 Jul 1; 42(7): 618-24.

[8] Thomson IR. The haemodynamic response to intubation: a perspective. Canadian journal of anaesthesia= Journal canadien d'anesthesie. 1989 Jul;36(4):367-9.

[9] Dundee JW, Robinson FP, McCollum JS, Patterson CC. Sensitivity to propofol in the elderly. Anaesthesia. 1986 May;41(5):482-5.

[10] Yan JW, McLeod SL, Iansavitchene A. Ketamine-propofol versus propofol alone for procedural sedation in the emergency department: a systematic review and meta-analysis. Academic Emergency Medicine. 2015 Sep;22(9):1003-13.

[11] White PF. Clinical pharmacology of intravenous induction drugs. International Anesthesiology Clinics. 1988 Jul 1;26(2):98-104.

[12] Joris JL, Chiche JD, Canivet JL, Jacquet NJ, Legros JJ, Lamy ML. Hemodynamic changes induced by laparoscopy and their endocrine correlates: effects of clonidine. Journal of the American College of Cardiology. 1998 Nov 1;32(5):1389-96.

[13] McLaughlin JG, Scheeres DE, Dean RJ, Bonnell BW. The adverse hemodynamic effects of

laparoscopic cholecystectomy. Surgical endoscopy. 1995 Feb;9:121-4.

[14] JG R. Intravenous anesthetics. Miller's anesthesia. 2010:719-28.

[15] Baradari AG, Firouzian A, Kiasari AZ, Aarabi M, Emadi SA, Davanlou A, Motamed N, Abdolmaleki EY. Effect of etomidate versus combination of propofol-ketamine and thiopental-ketamine on hemodynamic response to laryngoscopy and intubation: A randomized double blind clinical trial. Anesthesiology and pain medicine. 2016 Feb;6(1).

[16] King BD, Harris LC, Greifenstein FE, Elder JD, Dripps RD. Reflex circulatory responses to direct laryngoscopy and tracheal intubation performed during general anesthesia. The Journal of the American Society of Anesthesiologists. 1951 Sep 1;12(5):556-66.

[17] Abbasivash R, Aghdashi MM, Sinaei B, Kheradmand F. The effects of propofol-midazolam-ketamine co-induction on hemodynamic changes and catecholamine response. Journal of clinical anesthesia. 2014 Dec 1;26(8):628-33.

[18] Bijker JB, Van Klei WA, Kappen TH, Van Wolfswinkel L, Moons KG, Kalkman CJ. Incidence of intraoperative hypotension as a function of the chosen definition. Anesthesiology. 2007 Aug 1;107(2):213-20.

[19] Ali ZH, Hussein Z, Radeef AM. The Effect of Ketamine-Propofol Administration versus Propofol on Hemodynamic Stability during Induction and Intubation and Maintenance of Anesthesia. System. 2022;13:14.

[20] Sanuki T, Mishima G, Kurata S, Watanabe T, Kiriishi K, Tachi M, Ozaki Y, Okayasu I, Kawai M, Matsushita Y, Miura K. Comparison of the hemodynamic effects of propofol and ketamine as anesthetic induction agents during high-dose remifentanil administration: a single-center retrospective comparative study. Journal of dental anesthesia and pain medicine. 2015 Sep 1;15(3):129-34.

[21] Iuliana F. Effect of induction of general anesthesia with propofol and fentanyl on hemodynamic response. The Moldovan Medical Journal. 2019;62(3):7-12.

[22] Abbasivash R, Aghdashi MM, Sinaei B, Kheradmand F. The effects of propofol-midazolam-ketamine co-induction on hemodynamic changes and catecholamine response. Journal of clinical anesthesia. 2014 Dec 1;26(8):628-33.

Citation: *Dr. Md. Shahadat Hossain et al. Assessment of Hemodynamic Changes during Propofol and Ketamine Induction in General Anesthesia. ARC Journal of Anesthesiology. 2025; 10(1):1-6. DOI: https://doi.org/10.20431/2455-9792.1001001.*