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Original Research Article

Hemodynamic Comparison of Etomidate vs Propofol During Induction: A Prospective Randomized Controlled Trial

Bala MuraliKrishna Muppala[1], Hetal Modi[2], Ananya Rakshit[3]

[1]Assistant Professor, Department of Emergency Medicine, Great Eastern Medical School and Hospital, Srikakulam, Andhra Pradesh, India

[2]Anatomy Department, Assistant Professor, GMERS Medical College and Hospital, Himatnagar, Gujarat, India

[3]Assistant Professor, Department of Pharmacology, Rajiv Gandhi medical College and Chhatrapati Shivaji Maharaj Hospital, Thane, Maharashtra, India

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Corresponding Author: Dr. Ananya Rakshit

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Abstract:

Background: The selection of an appropriate induction agent is crucial in maintaining hemodynamic stability during general anesthesia. Propofol and etomidate are commonly used intravenous anesthetics with distinct cardiovascular profiles. Understanding their comparative hemodynamic effects is essential for optimizing patient outcomes, particularly in vulnerable populations.

Methods: This prospective, randomized, double-blind controlled trial enrolled 120 adult patients (ASA I-II) scheduled for elective surgery. Patients were randomly allocated to receive either etomidate (0.3 mg/kg) or propofol (2 mg/kg) for induction. Hemodynamic parameters including mean arterial pressure (MAP), heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded at baseline, immediately post-induction, and at 1, 3, 5, and 10 minutes post-induction.

Results: The propofol group demonstrated a significantly greater reduction in MAP compared to the etomidate group at 1 minute post-induction (68.4 ± 8.2 mmHg vs. 82.6 ± 7.8 mmHg, $p < 0.001$). The incidence of hypotension was significantly higher in the propofol group (31.7% vs. 8.3%, $p = 0.002$). Heart rate changes were comparable between groups. Etomidate maintained hemodynamic stability throughout the induction period with minimal fluctuations from baseline values.

Conclusion: Etomidate provides superior hemodynamic stability compared to propofol during anesthesia induction. These findings support the preferential use of etomidate in patients where cardiovascular stability is paramount.

Keywords: Etomidate; Propofol; Hemodynamic Stability; Anesthesia Induction; Mean Arterial Pressure; Hypotension.

Introduction

General anesthesia induction represents a critical phase during which patients are particularly vulnerable to hemodynamic instability [1]. The choice of induction agent significantly influences cardiovascular parameters, and selecting an appropriate agent is essential for maintaining optimal perfusion to vital organs [2]. Among the available intravenous anesthetics, propofol and etomidate remain the most frequently utilized agents for induction worldwide [3].

Propofol (2,6-diisopropylphenol) is characterized by its rapid onset, smooth induction profile, and favorable recovery characteristics [4]. However, its use is associated with significant cardiovascular depression, primarily through peripheral vasodilation and direct myocardial depression [5]. Studies have demonstrated that propofol can reduce systemic vascular resistance by 15-40%, leading to substantial decreases in arterial blood pressure [6].

Etomidate, an imidazole derivative, has gained recognition for its remarkable cardiovascular stability during induction [7]. Its minimal effects on myocardial contractility and systemic vascular resistance make it an attractive alternative for patients with compromised cardiovascular function [8]. Reich et al. demonstrated that etomidate maintains hemodynamic stability even in patients with significant cardiac disease [9].

Recent investigations have focused on comparing these agents in various clinical contexts. Pandey and colleagues reported significant differences in blood pressure responses between etomidate and propofol

in patients undergoing non-cardiac surgery [10]. Similarly, Aggarwal et al. observed that etomidate-induced hypotension occurred less frequently compared to propofol [11]. However, meta-analyses have yielded heterogeneous results, partly due to differences in study populations, dosing protocols, and outcome definitions [12].

Despite extensive research, several gaps remain in our understanding of the comparative hemodynamic profiles of these agents. Most existing studies have limited sample sizes or focus on specific patient populations such as those with cardiac disease or the elderly [13]. Furthermore, the temporal pattern of hemodynamic changes during the critical first ten minutes post-induction has not been adequately characterized in contemporary practice settings [14].

The aim of this study was to compare the hemodynamic effects of etomidate versus propofol during anesthesia induction in ASA I-II adult patients undergoing elective surgical procedures, with particular emphasis on the incidence of clinically significant hypotension and the temporal dynamics of cardiovascular parameters.

Materials and Methods

Study Design and Setting: This prospective, randomized, double-blind, parallel-group controlled trial was conducted at tertiary care hospital.

Sample Size Calculation: Based on previous literature reporting a mean difference of 12 mmHg in MAP between etomidate and propofol groups with a standard deviation of 15 mmHg, a sample size of 52 patients per group was required to achieve 80% power at a two-sided alpha level of 0.05. Accounting for a 15% dropout rate, we planned to enroll 60 patients per group, totaling 120 participants.

Inclusion and Exclusion Criteria: Inclusion criteria comprised: age 18-65 years, ASA physical status I-II, scheduled for elective surgery under general anesthesia, and body mass index (BMI) between 18.5 and 30 kg/m². Exclusion criteria included: known hypersensitivity to study medications, cardiovascular disease (including uncontrolled hypertension, coronary artery disease, or heart failure), adrenal insufficiency or chronic corticosteroid use, pregnancy or lactation, chronic opioid use, and anticipated difficult airway.

Randomization and Blinding: Patients were randomly allocated to one of two groups using computer-generated random numbers in blocks of four. Group E (n=60) received etomidate, and Group P (n=60) received propofol. Randomization codes were sealed in opaque envelopes and opened immediately before induction. Study medications were prepared by an anesthesiologist not involved in patient care or data collection and administered in identical syringes to maintain blinding.

Anesthetic Protocol: All patients underwent standard preoperative fasting and received no premedication. Upon arrival in the operating room, standard monitoring was established including electrocardiography, non-invasive blood pressure measurement, pulse oximetry, and capnography. An 18-gauge intravenous cannula was secured, and Ringer's lactate solution was initiated at 2 mL/kg/hr.

After a 5-minute stabilization period, baseline hemodynamic parameters were recorded. Induction was performed using either etomidate 0.3 mg/kg or propofol 2 mg/kg administered intravenously over 30 seconds. Fentanyl 2 mcg/kg was administered 3 minutes prior to induction in both groups. Following loss of consciousness, rocuronium 0.6 mg/kg was administered to facilitate tracheal intubation, which was performed 90 seconds later.

Hemodynamic Measurements: Primary hemodynamic parameters including systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) were recorded at the following time points: baseline (T0), immediately after induction (T1), 1 minute (T2), 3 minutes (T3), 5 minutes (T4), and 10 minutes (T5) post-induction.

Outcome Measures: The primary outcome was the change in MAP from baseline at 1 minute post-induction. Secondary outcomes included incidence of hypotension (defined as MAP < 65 mmHg or > 20% decrease from baseline), incidence of hypertension (> 20% increase from baseline), bradycardia (HR < 50 beats/min), tachycardia (HR > 100 beats/min), and requirement for vasopressor support.

Statistical Analysis: Data analysis was performed using SPSS version 26.0 (IBM Corporation, Armonk, NY). Continuous variables were expressed as mean ± standard deviation (SD) and compared using independent samples t-test or Mann-Whitney U test as appropriate. Categorical variables were expressed as frequencies and percentages and analyzed using chi-square test or Fisher's exact test. Repeated measures ANOVA was used to compare hemodynamic parameters across time points between groups. A p-value < 0.05 was considered statistically significant.

Results

Patient Demographics and Baseline Characteristics: A total of 128 patients were assessed for eligibility, of which 120 were randomized and included in the final analysis (60 per group). There were no significant differences between groups regarding demographic

characteristics, ASA physical status, or baseline hemodynamic parameters (Table 1).

Table 1: Demographic and Baseline Characteristics

Parameter	Etomidate Group (n=60)	Propofol Group (n=60)	p-value
Age (years)	42.8 ± 12.4	44.2 ± 11.8	0.524
Sex (M/F)	32/28	29/31	0.582
Weight (kg)	68.4 ± 10.2	67.8 ± 9.8	0.734
BMI (kg/m²)	24.2 ± 3.1	23.9 ± 3.4	0.608
ASA I/II	38/22	35/25	0.582
Baseline SBP (mmHg)	128.6 ± 14.2	130.4 ± 13.8	0.478
Baseline DBP (mmHg)	78.4 ± 9.6	79.2 ± 10.2	0.661
Baseline MAP (mmHg)	95.1 ± 10.4	96.3 ± 10.8	0.538
Baseline HR (bpm)	76.8 ± 12.4	78.2 ± 11.6	0.524

Data expressed as mean ± SD or n; BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; MAP: Mean Arterial Pressure; HR: Heart Rate

Hemodynamic Changes During Induction: Significant differences in hemodynamic parameters were observed between groups throughout the study period (Table 2). The propofol group demonstrated significantly greater reductions in SBP, DBP, and MAP compared to the etomidate group at all post-induction time points.

Table 2: Hemodynamic Parameters at Different Time Points

Parameter	Time Point	Etomidate (n=60)	Propofol (n=60)	p-value
MAP (mmHg)	T0 (Baseline)	95.1 ± 10.4	96.3 ± 10.8	0.538
	T1 (Immediate)	88.2 ± 9.6	74.8 ± 10.2	<0.001
	T2 (1 min)	82.6 ± 7.8	68.4 ± 8.2	<0.001
	T3 (3 min)	84.4 ± 8.2	72.6 ± 9.4	<0.001
	T4 (5 min)	86.8 ± 7.6	76.2 ± 8.8	<0.001
	T5 (10 min)	90.2 ± 8.4	82.4 ± 9.2	<0.001
HR (bpm)	T0 (Baseline)	76.8 ± 12.4	78.2 ± 11.6	0.524
	T1 (Immediate)	82.4 ± 14.2	84.6 ± 13.8	0.386
	T2 (1 min)	80.6 ± 12.8	86.2 ± 14.4	0.024
	T3 (3 min)	78.4 ± 11.6	82.8 ± 13.2	0.052
	T4 (5 min)	76.2 ± 10.8	80.4 ± 12.6	0.048
	T5 (10 min)	74.8 ± 10.2	78.2 ± 11.8	0.096
SBP (mmHg)	T0 (Baseline)	128.6 ± 14.2	130.4 ± 13.8	0.478
	T2 (1 min)	112.4 ± 12.6	94.8 ± 14.2	<0.001
	T5 (10 min)	122.6 ± 11.8	108.4 ± 13.4	<0.001

Data expressed as mean ± SD; MAP: Mean Arterial Pressure; HR: Heart Rate; SBP: Systolic Blood Pressure

The maximum decrease in MAP from baseline was 13.1% ± 6.8% in the etomidate group compared to 29.0% ± 8.4% in the propofol group ($p < 0.001$). Heart rate showed a compensatory increase in both groups, with slightly higher values observed in the propofol group at 1 minute post-induction.

Adverse Events and Interventions: The incidence of hypotension and need for vasopressor support were significantly higher in the propofol group (Table 3). No patients in either group experienced bradycardia requiring intervention.

Table 3: Incidence of Adverse Events and Interventions

Event	Etomidate (n=60)	Propofol (n=60)	p-value
Hypotension (MAP <65 mmHg)	5 (8.3%)	19 (31.7%)	0.002
Hypotension (>20% decrease)	8 (13.3%)	28 (46.7%)	<0.001
Hypertension (>20% increase)	2 (3.3%)	0 (0%)	0.496
Bradycardia (HR <50 bpm)	1 (1.7%)	2 (3.3%)	1.000
Tachycardia (HR >100 bpm)	4 (6.7%)	7 (11.7%)	0.343
Vasopressor requirement	3 (5.0%)	14 (23.3%)	0.004
Myoclonus	8 (13.3%)	1 (1.7%)	0.032
Pain on injection	2 (3.3%)	18 (30.0%)	<0.001

Data expressed as n (%)

Myoclonus was significantly more common in the etomidate group (13.3% vs. 1.7%, $p = 0.032$), while pain on injection was more frequent with propofol (30.0% vs. 3.3%, $p < 0.001$).

Discussion

The present study demonstrates that etomidate provides significantly superior hemodynamic stability compared to propofol during anesthesia induction in ASA I-II adult patients. The propofol group exhibited a 29% mean reduction in MAP from baseline, contrasting sharply with only 13% in the etomidate group. These findings align with the established pharmacological profiles of both agents and have important clinical implications.

The cardiovascular depression associated with propofol has been well-documented and is attributed to multiple mechanisms including direct myocardial depression, reduction in sympathetic nervous system activity, and peripheral vasodilation [15]. Larsen and colleagues demonstrated that propofol decreases systemic vascular resistance by approximately 30% while simultaneously reducing cardiac output by 15% [16]. Our observed reduction in MAP of nearly 30% is consistent with these mechanistic studies.

Etomidate's hemodynamic stability stems from its unique pharmacological profile. Unlike propofol, etomidate has minimal effects on sympathetic tone and does not significantly impair baroreflex function [17]. Brussel et al. reported that etomidate maintains cardiac output and systemic vascular resistance within 10% of baseline values, findings that are corroborated by our results [18].

The incidence of clinically significant hypotension (MAP < 65 mmHg) was nearly four-fold higher in the propofol group (31.7% vs. 8.3%). This difference is clinically meaningful, as intraoperative hypotension has been associated with increased morbidity and mortality.

The absence of significant bradycardia in either group suggests that the doses used were appropriate and did not cause excessive vagal stimulation. Pretreatment with benzodiazepines or small doses of opioids has been shown to reduce this phenomenon and should be considered in clinical practice.

Our study has several limitations. First, we excluded patients with significant cardiovascular disease, limiting generalizability to this vulnerable population where hemodynamic stability is most critical. Second, the follow-up period was limited to 10 minutes post-induction, and longer-term cardiovascular effects were not assessed. Third, we did not measure cardiac output or systemic vascular resistance directly, which would have provided additional mechanistic insights.

Conclusion

This prospective randomized controlled trial demonstrates that etomidate provides significantly superior hemodynamic stability compared to propofol during anesthesia induction in adult patients undergoing elective surgery. Etomidate was associated with smaller reductions in mean arterial pressure, a lower incidence of clinically significant hypotension, and reduced requirement for vasopressor support. While etomidate was associated with increased myoclonus, propofol caused significantly more injection pain. These findings support the consideration of etomidate as the preferred induction agent in clinical scenarios where maintaining cardiovascular stability is of paramount importance. Future studies should examine these findings in patients with cardiovascular comorbidities and evaluate longer-term outcomes.

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