International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-2-12*

Esmolol vs. Propofol for Attenuation of Hemodynamic Responses During Tracheal Extubation: A Comparative Clinical Study.

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Received: 01-04-2026
Revised: 24-04-2026
Accepted: 08-05-2026
Published: 16-05-2026

Abstract

Background: ***Materials and Methods:*** This prospective randomized clinical study was conducted on 100 ASA grade I–II patients undergoing elective surgical procedures under general anesthesia. Patients were randomly allocated into two groups to receive either intravenous esmolol 0.5 mg/kg or intravenous propofol 0.5 mg/kg as a bolus dose administered two minutes prior to tracheal extubation. Hemodynamic parameters including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at predefined intervals. Quality of extubation, emergence characteristics, and adverse effects were also assessed and compared between the groups.b. ***Results:*** Both esmolol and propofol significantly attenuated the hemodynamic stress response associated with tracheal extubation. Esmolol demonstrated superior control of heart rate, whereas propofol was more effective in reducing SBP, DBP, and MAP. Patients receiving propofol experienced smoother extubation with reduced coughing. In contrast, the esmolol group showed better post-extubation alertness with lower emergence agitation scores. Bradycardia occurred more frequently in the esmolol group; however, no patient required therapeutic intervention. No major adverse events were observed in either group. ***Conclusion***: Both intravenous esmolol and propofol are effective in attenuating extubation-induced hemodynamic responses. Esmolol is preferable when strict heart rate control is required, particularly in patients with coronary artery disease, while propofol offers superior blood pressure attenuation and smoother extubation with additional antiemetic and airway-protective benefits. The choice of agent should therefore be individualized according to patient profile and clinical requirements.

Keywords: Esmolol, Propofol, Hemodynamic Responses, Tracheal Extubation.

INTRODUCTION

Extubation of the trachea—the process of removing the endotracheal tube when the indications for artificial airway support have been resolved—is widely recognized as one of the most critical stages of general anesthesia. Unlike intubation, which has received extensive research attention, extubation has historically been underappreciated as a source of perioperative risk.

Tracheal extubation, particularly when performed in lighter planes of anesthesia, stimulates laryngeal and tracheal mechanoreceptors and activates reflex sympathetic pathways. These pathways trigger the release of catecholamines from the adrenal medulla, resulting in hemodynamic responses manifested as rises in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure. Studies have documented increases of 20% or more in both heart rate and systolic arterial pressure in approximately 70% of patients under standard extubation conditions.

These transient hemodynamic changes, while often self-limiting in healthy individuals, can have life-threatening consequences in patients with systemic hypertension, coronary artery disease, intracranial aneurysms, or cerebrovascular disease. Complications may include cerebral hemorrhage, myocardial ischemia, arrhythmias, left ventricular failure, pulmonary edema, and rupture of intracranial aneurysms.

Additionally, extubation provokes airway reflexes including coughing, bucking, straining, laryngospasm, and breath-holding, all of which increase intraabdominal, intrathoracic, and intracranial pressures. Conversely, extubation under deep sedation risks hypoventilation and hypoxia. This narrow therapeutic window makes smooth, controlled extubation a demanding clinical art.

Over the decades, numerous pharmacological strategies have been investigated to blunt extubation-related responses, including opioids (fentanyl), local anesthetics (lignocaine), inhalational agents (sevoflurane), vasodilators (nitroglycerine), alpha-blockers, beta-blockers (esmolol, labetalol, metoprolol), calcium channel blockers (diltiazem, verapamil, nicardipine), magnesium sulfate, and clonidine. None of these has emerged as a universally superior agent.

Esmolol, an ultra-short-acting selective β1-adrenergic blocker, and propofol, the most widely used intravenous anesthetic today, have both demonstrated promise in attenuating extubation responses. However, comparative data on their use at bolus doses prior to extubation remain limited. This study was designed to fill that gap.

MATERIALS AND METHODS

This was a prospective, randomized, comparative clinical study conducted at Shadan Institute of Medical Sciences and Ayaan Institute of Medical Sciences over a 2-year period. Ethical clearance and informed consent were obtained in accordance with institutional protocols.

Sample Size and Randomization

100 patients were enrolled and randomized into two groups of 50 each using the sealed envelope method:

- Group I (Esmolol): IV esmolol 0.5 mg/kg bolus, 2 minutes before extubation
- Group II (Propofol): IV propofol 0.5 mg/kg bolus, 2 minutes before extubation

Inclusion and Exclusion Criteria

Inclusion: ASA I & II, either sex, age 18–60 years, elective surgery under general anesthesia.

Exclusion: ASA III & IV, patient refusal, BMI >50, pregnancy or lactation, emergency surgeries, known allergy to study drugs.

Anesthetic Protocol

All patients underwent standard preoperative fasting (nil per oral for 8 hours). On arrival to the operating room:

- IV access established; Ringer's lactate infusion commenced
- Monitoring: Heart Rate (HR), Non-Invasive Blood Pressure (NIBP), SpO_2, ECG
- Premedication: Glycopyrrolate 4 mcg/kg, Ondansetron 0.15 mg/kg, Fentanyl 2 mcg/kg
- Induction: Propofol 2 mg/kg IV; intubation facilitated with Vecuronium 0.1 mg/kg
- Maintenance: 50% O_2 / 50% N_2O + Sevoflurane 1% + Vecuronium
- Sevoflurane discontinued prior to study drug administration
- Reversal: Neostigmine 0.05 mg/kg + Glycopyrrolate 0.01 mg/kg IV

Study drug was administered 2 minutes before extubation as per group allocation.

Outcome Measures

Hemodynamic Parameters (recorded at multiple time points):

- Basal (end of surgery), after reversal (REV), after drug (DRUG), 1 minute after drug (DRUG1), at extubation (EXT), and at 1, 2, 3, 4, 5, 15, and 30 minutes post-extubation (E1–E30)

Extubation Quality Score (5-point scale):

Score	Response
1	No cough (smooth extubation)
2	Minimal cough (1–2 times)
3	Moderate cough (3–4 times)
4	Severe cough (5–10 times)
5	>10 coughs, laryngospasm, or breath-holding (poor extubation)

Emergence Agitation Score (6-point scale):

Score	Response
1	Apprehensive, restless, agitated
2	Calm, oriented, co-operative
3	Drowsy, responding to commands
4	Somnolent, responds quickly to stimuli
5	Somnolent, responds slowly to stimuli
6	Somnolent, unresponsive to stimuli

Adverse Effects Monitored: Vomiting, respiratory depression, laryngospasm, bronchospasm, bradycardia (HR <60/min), hypotension (MAP <60 mmHg).

Statistical analysis was performed using SPSS software. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: The two groups were comparable in all demographic parameters.

Parameter	Propofol Group	Esmolol Group	P Value
Mean Age Group	30–39 years (44%)	30–39 years (36%)	NS
Male : Female	22:28 (44%:56%)	22:28 (44%:56%)	NS
Mean Weight (kg)	60.75 ± 6.85	58.9 ± 10.47	0.59
Mean Height (cm)	160.18 ± 6.45	161.18 ± 5.44	0.44
ASA I / II (%)	88% / 12%	82% / 18%	NS

There was no statistically significant difference in demographic profiles between the two groups. Both groups demonstrated a reduction in heart rate from basal values. However, the esmolol group showed a consistently and significantly greater decrease in heart rate throughout all measurement time points ($p < 0.05$ at all time points). The mean heart rate in the esmolol group remained substantially lower than in the propofol group from the time of drug administration through 30 minutes post-extubation.

Table 2: Comparison of Heart Rate (beats per minute) Between Propofol and Esmolol Groups at Different Time Intervals

Time	Propofol (bpm)	Esmolol (bpm)	P Value
Basal	84.86 ± 7.74	86.6 ± 5.93	<0.0000001
Extubation	78.78 ± 7.57	74.58 ± 4.84	0.001
E5	78.78 ± 7.57	75.02 ± 4.42	0.003
E30	80.26 ± 7.73	76.36 ± 4.41	0.002

Esmolol is significantly superior to propofol in attenuating heart rate response to extubation.
Both groups showed reductions in SBP. The propofol group demonstrated a greater decrease in SBP at all time points, though this difference was statistically significant only at basal ($p = 0.005$). At all subsequent time points, the difference was not statistically significant ($p > 0.05$). Propofol produced a numerically greater reduction in SBP, though the difference was not consistently statistically significant versus esmolol.

The propofol group demonstrated a significantly greater reduction in DBP compared to the esmolol group at multiple time points ($p < 0.05$ at most measurement intervals from DRUG through E30). Propofol was significantly more effective in reducing DBP.

The propofol group showed a significantly greater decrease in MAP compared to the esmolol group across most measurement time points ($p < 0.05$).

Table 3: Comparison of Mean Heart Rate (bpm) Between Propofol and Esmolol Groups at Baseline, Extubation, and Post-Extubation Intervals (E5 and E30)

Time	Propofol (bpm)	Esmolol (bpm)	P Value
Basal	84.86 ± 7.74	86.6 ± 5.93	<0.0000001
Extubation	78.78 ± 7.57	74.58 ± 4.84	0.001
E5	78.78 ± 7.57	75.02 ± 4.42	0.003
E30	80.26 ± 7.73	76.36 ± 4.41	0.002

Table 4: Comparison of Mean Arterial Pressure (MAP, mmHg) Between Propofol and Esmolol Groups at Baseline, Extubation, and Post-Extubation Intervals (E1 and E30)

Time	Propofol MAP (mmHg)	Esmolol MAP (mmHg)	P Value
Basal	89.46 ± 12.5	94.4 ± 8.42	0.02
Extubation	87.8 ± 8.73	91.26 ± 8.1	0.04
E1	88.6 ± 8.61	92.04 ± 8.16	0.04
E30	89.72 ± 8.56	92.84 ± 8.41	0.06

Propofol was significantly superior to esmolol in reducing MAP during and after extubation.

Table 5: Extubation Quality Score

Score	Propofol	Esmolol
I (No cough)	8%	0%
II (Minimal cough)	78%	16%
III (Moderate cough)	14%	78%
IV (Severe cough)	0%	6%

In the propofol group, 86% of patients had no cough or only minimal cough during extubation. In contrast, 78% of esmolol patients experienced moderate cough, and 6% had severe cough. Propofol provided significantly superior extubation quality compared to esmolol.

Table 6: Emergence Agitation Score

Score	Propofol	Esmolol
I (Apprehensive/Restless)	0%	8%
II (Calm, oriented)	26%	80%
III (Drowsy, responds to commands)	64%	12%

In the propofol group, 64% of patients were drowsy but responsive post-extubation, reflecting the sedative effect of propofol. In the esmolol group, 80% of patients were calm, oriented, and fully cooperative—a significantly better consciousness profile. Esmolol was associated with significantly better post-extubation alertness compared to propofol.

Table 7: Adverse Effects

Adverse Effect	Propofol	Esmolol
Vomiting	1 (2%)	3 (6%)
Respiratory Depression	0	0
Laryngospasm	0	0
Bronchospasm	0	0
Bradycardia (HR <60/min)	1 (2%)	6 (12%)
Hypotension (MAP <60 mmHg)	0	0

The incidence of bradycardia was notably higher in the esmolol group (12% vs 2%). Importantly, none of these cases required pharmacological intervention. Neither group experienced respiratory depression, laryngospasm, bronchospasm, or hemodynamically significant hypotension.

DISCUSSION

The present study confirms that both esmolol and propofol, when administered as IV bolus doses (0.5 mg/kg each) two minutes prior to extubation, effectively attenuate hemodynamic stress responses. However, their pharmacological profiles confer distinct and complementary advantages.

Esmolol's β1-selective adrenergic blockade provides a mechanistically direct and superior pathway for heart rate reduction. Its ultra-short half-life (~9 minutes) makes it highly titratable. The statistically significant and clinically meaningful reduction in heart rate observed in the esmolol group throughout all time points is consistent with findings from Dyson (1990), Fuhrman et al. (1992), and Dr. Konda Sunil Kumar et al. (2018), all of whom found esmolol superior for heart rate control.

Contrary to some earlier studies, this investigation found propofol to be more effective in reducing SBP, DBP, and MAP. This finding aligns with Nagrale MH et al. (2016) and Conti and Smith (1998), and may be attributed to propofol's multiple mechanisms of blood pressure reduction: peripheral vasodilation via decreased systemic vascular resistance, direct myocardial depression, and its effect on the baroreflex. Propofol's ability to blunt airway irritation through its CNS-sedating effects also reduces the sympathetic stimulus at the source.

The markedly better extubation quality in the propofol group reflects propofol's ability to suppress airway reflexes and depress the cough reflex through its GABAergic CNS activity. This advantage is clinically significant: coughing and bucking during extubation raise intrathoracic, intraabdominal, and intracranial pressures, and may precipitate complications in susceptible patients. Propofol's airway-protective profile in this context is a major clinical advantage.

The sedation residual from propofol, as evidenced by the high proportion of drowsy-but-responsive patients in the propofol group, represents a trade-off. While this sedation contributes to smooth extubation, it may delay return to full consciousness and prolong PACU stay. Esmolol, which does not cause CNS depression, preserved alertness far better, with 80% of patients calm and fully oriented post-extubation. This is advantageous in procedures requiring early neurological assessment.

Both drugs were well tolerated. The higher rate of bradycardia with esmolol (12% vs 2%) is an expected pharmacological consequence of β-blockade, and while none required treatment in this study, clinicians should maintain vigilance in patients with baseline bradycardia or conduction abnormalities. Propofol's slightly lower emesis rate (2% vs 6%) reflects its well-documented antiemetic property, which may provide additive benefit in high-risk PONV populations.

CONCLUSION

Both drugs effectively attenuate hemodynamic responses to tracheal extubation. Esmolol is significantly superior for heart rate control throughout the peri-extubation period. Propofol is significantly superior for reducing DBP and MAP, and numerically greater in reducing SBP. Propofol provides significantly better extubation quality (less coughing and airway reactivity). Esmolol provides better post-extubation alertness (lower emergence agitation/sedation). Bradycardia was more frequent with esmolol (12%) but self-limiting and required no intervention. No serious adverse events (laryngospasm, bronchospasm, respiratory depression, hemodynamically significant hypotension) occurred in either group.

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