

ORIGINAL ARTICLE

Attenuation of Hemodynamic Responses to Laryngoscopy and Intubation- Lignocaine versus Esmolol in 100 Cases

DOI: 10.5281/zenodo.18116238


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Received: 18 Dec 2025

Accepted: 24 Dec 2025

Published: 01 Jan 2026

Published by:

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation cause sympathetic stimulation producing tachycardia and hypertension which may be harmful in patients with cardiovascular disease. This randomized study compared intravenous lignocaine and esmolol for attenuation of this pressor response. **Methods & Materials:** 100 adult patients (ASA I-II) undergoing elective surgery under general anesthesia were randomized into two groups of 50 each. Group L received lignocaine 1.5 mg·kg⁻¹ IV 90 seconds before laryngoscopy; Group E received esmolol 1.0 mg·kg⁻¹ IV 90 seconds before laryngoscopy. Hemodynamic variables (heart rate [HR], mean arterial pressure [MAP]) were recorded at baseline (T0), after induction but before study drug (T1), immediately after intubation (T2), and at 1 (T3), 3 (T4) and 5 (T5) minutes after intubation. Primary endpoint: peak change in HR and MAP from baseline. Secondary endpoints: incidence of significant tachycardia (>20% increase), hypotension (MAP <65 mmHg), bradycardia (HR <50 bpm), and adverse events. **Results:** Both drugs attenuated the pressor response compared with historical untreated responses, but esmolol produced significantly less increase in HR and MAP at T2–T4. Mean peak HR change at T2: Group L +18.6 ± 9.4 bpm vs Group E +4.2 ± 7.8 bpm (p < 0.001). Mean peak MAP change at T2: Group L +16.8 ± 10.2 mmHg vs Group E +6.9 ± 8.5 mmHg (p < 0.001). Clinically significant tachycardia occurred in 28/50 (56%) of Group L and 6/50 (12%) of Group E (p < 0.001). Two patients in Group E had transient bradycardia responsive to atropine. No episode of sustained hypotension requiring vasopressor occurred. **Conclusion:** A single IV bolus of esmolol 1.0 mg·kg⁻¹ given shortly before laryngoscopy attenuates the hemodynamic response to intubation significantly better than lignocaine 1.5 mg·kg⁻¹ in ASA I-II adults. Esmolol may be preferred when a rapid, short-acting sympatholytic effect is desired.

Keywords: Laryngoscopy, Endotracheal Intubation, Lignocaine, Esmolol, Hemodynamic Response, Tachycardia, Randomized Study

(The Insight 2025; 8(3): 652-655)

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INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of airway management but are also recognized as potent stimuli capable of provoking significant sympathetic activation. The resultant surge in catecholamine release often produces tachycardia, hypertension, and arrhythmias, particularly within the first few minutes after intubation. These responses, though transient in healthy individuals, may be detrimental in patients with cardiovascular disease, intracranial pathology, or reduced physiological reserves. The hemodynamic stress response to intubation was first described several decades ago and remains a critical concern in modern anesthetic practice due to its potential to precipitate myocardial ischemia, cerebrovascular events, or increased intracranial and intraocular pressures.^[1] Several pharmacological strategies have been evaluated to mitigate this sympathetic surge. Among the commonly used agents, lignocaine and esmolol have gained considerable attention. Lignocaine, a sodium channel blocker with both local anesthetic and antiarrhythmic properties, is believed to blunt

hemodynamic responses by depressing airway reflexes and attenuating catecholamine release.^[2] It has been administered intravenously in doses ranging from 1–2 mg/kg before laryngoscopy, with variable success reported across studies. Esmolol, an ultra-short-acting β_1 -selective adrenergic blocker, provides rapid onset and a predictable duration of action, making it particularly suitable for peri-intubation hemodynamic control. Its ability to reduce heart rate and myocardial oxygen consumption has been well documented, and multiple trials have shown promising results in attenuating tachycardia and hypertension associated with laryngoscopy.^[3] However, esmolol may induce transient bradycardia or hypotension in some individuals, requiring cautious dosing. Direct comparisons of lignocaine and esmolol have produced mixed results, with some studies reporting superior hemodynamic attenuation with esmolol, while others suggest comparable efficacy at higher doses of lignocaine.^[4,5] The optimal agent for routine clinical use remains a subject of debate, particularly in resource-limited settings where cost, availability, and safety profiles must be carefully weighed.

Furthermore, there is limited local data from South Asian populations, where variations in demographics, comorbidities, and anesthetic techniques may influence pharmacodynamic responses. The present study was designed to compare the efficacy of intravenous lignocaine and esmolol in attenuating cardiovascular responses during laryngoscopy and endotracheal intubation in a tertiary hospital setting. By assessing changes in heart rate (HR), mean arterial pressure (MAP), and the incidence of adverse hemodynamic events, this study aims to provide evidence that may guide practical decision-making in airway management. The findings are expected to contribute to ongoing efforts to optimize peri-intubation safety through targeted pharmacological interventions.

METHODS & MATERIALS

Study design and setting

Prospective, randomized, single-blind (observer-blind) clinical trial conducted at Department of Anaesthesiology, Dinajpur Medical College Hospital, Dinajpur, Bangladesh between February 2024 to July 2024. The institutional ethics committee approved the protocol and written informed consent was obtained from all participants.

Participants

Inclusion criteria: adult patients aged 18–65 years, ASA physical status I–II, scheduled for elective surgery under general anesthesia requiring orotracheal intubation. Exclusion criteria: known cardiac conduction abnormalities, baseline bradycardia (HR <50 bpm), uncontrolled hypertension, treatment with β -blockers or anti-arrhythmic drugs, allergy to study drugs, difficult airway anticipated (Mallampati 3–4), pregnancy, or significant hepatic/renal impairment.

Randomization and blinding

100 patients were randomized (computer-generated random numbers) into two equal groups: Group L (lignocaine, $n = 50$) and Group E (esmolol, $n = 50$). The anesthesia provider administering the drug was aware of allocation, but the observer recording hemodynamics was blinded to group assignment.

Anesthetic technique

Standard monitors were applied (ECG, noninvasive blood pressure, pulse oximetry). Baseline hemodynamics recorded (T0). All patients received midazolam $0.03 \text{ mg}\cdot\text{kg}^{-1}$ IV as premedication. Induction was standardized: fentanyl $2 \text{ }\mu\text{g}\cdot\text{kg}^{-1}$ IV (given 3 minutes before induction), propofol $2 \text{ mg}\cdot\text{kg}^{-1}$ IV for induction, and succinylcholine $1.5 \text{ mg}\cdot\text{kg}^{-1}$ IV to facilitate tracheal intubation. After loss of consciousness and confirmation of mask ventilation, the allocated study drug was given IV over 10–15 seconds at 90 seconds before laryngoscopy. Laryngoscopy and intubation were performed

by an experienced anesthetist using Macintosh blade size appropriate to the patient, keeping laryngoscopy time under 15 seconds when possible. Additional anesthetic management after T5 was at discretion of the attending anesthetist.

Study drugs

- Group L (Lignocaine): lignocaine $1.5 \text{ mg}\cdot\text{kg}^{-1}$ IV bolus (preservative-free).
- Group E (Esmolol): esmolol $1.0 \text{ mg}\cdot\text{kg}^{-1}$ IV bolus.

Measurements

HR and systolic/diastolic BP were recorded at:

- T0 — baseline (before premedication)
- T1 — after induction but before study drug (to capture induction effects)
- T2 — immediately after tracheal intubation (within 1 min)
- T3 — 1 minute after intubation
- T4 — 3 minutes after intubation
- T5 — 5 minutes after intubation

MAP was calculated as $(\text{SBP} + 2 \times \text{DBP}) / 3$. Adverse events (hypotension MAP <65 mmHg, bradycardia HR <50 bpm, arrhythmias, bronchospasm) were recorded and treated per standard practice.

Outcomes

Primary outcome: difference in peak change in HR and MAP from baseline between groups. Secondary outcomes: incidence of tachycardia (>20% increase from baseline), bradycardia, hypotension, and other adverse events.

Sample size and statistics

Sample size calculation (pilot data and literature) determined 50 patients per group would provide >80% power to detect a 10-bpm difference in peak HR with $\alpha = 0.05$. Data were analyzed using [statistical package SPSS version 25]. Continuous variables presented as mean $\pm$ SD and compared with Student's t-test (independent) or repeated measures ANOVA where appropriate. Categorical variables compared with Chi-square or Fisher's exact test. A p-value < 0.05 was considered statistically significant.

RESULTS

Participant Flow and Baseline Characteristics

All 100 enrolled patients completed the study and were included in the final analysis. Fifty patients received lignocaine (Group L) and fifty received esmolol (Group E). Both groups were comparable in terms of age, sex distribution, BMI, and baseline hemodynamic variables. No statistically significant differences were observed at baseline, ensuring comparability before drug administration.

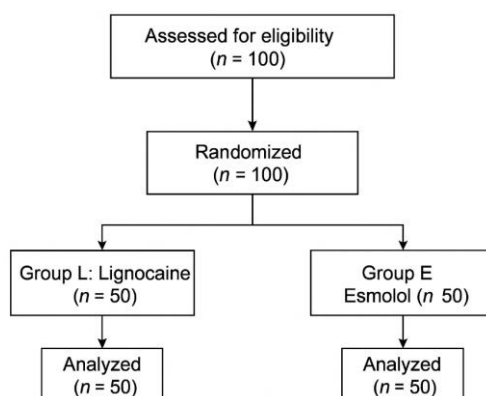


Figure – 1: Study flow diagram (CONSORT style)-pic show

Table – I: Baseline Characteristics of Participants (n = 100)

Variable	Group L (n=50)	Group E (n=50)	p-value
Age (years), mean ± SD	38.6 ± 11.2	39.4 ± 10.6	0.72
Sex (Male/Female)	28/22	26/24	0.68
BMI (kg/m ²), mean ± SD	24.1 ± 3.2	24.5 ± 3.6	0.56
ASA class (I/II)	32/18	30/20	0.66
Baseline HR (bpm), mean ± SD	78.4 ± 9.1	79.0 ± 8.6	0.74
Baseline MAP (mmHg), mean ± SD	91.2 ± 9.4	90.7 ± 8.8	0.78

Both groups demonstrated similar demographic and clinical profiles. Baseline HR and MAP were nearly identical between groups ($p > 0.70$). No significant differences were found in ASA physical status, sex distribution, or BMI. This confirmed successful randomization and allowed valid comparison of hemodynamic responses.

Hemodynamic Response to Laryngoscopy and Intubation

Significant increases in HR and MAP occurred after tracheal intubation in both groups, but the magnitude of increase was consistently lower in Group E (esmolol). The highest surge was observed at T2 (immediately after intubation). Esmolol significantly blunted both HR and MAP rises at all post-intubation time points compared with lignocaine.

Table – II: Hemodynamic Parameters at Each Time Point (mean ± SD)

Time Point	HR Group L	HR Group E	p-value	MAP Group L (mmHg)	MAP Group E (mmHg)	p-value
T0 – Baseline	78.4 ± 9.1	79.0 ± 8.6	0.74	91.2 ± 9.4	90.7 ± 8.8	0.78
T1 – Post-induction	72.1 ± 7.8	71.8 ± 8.0	0.85	83.5 ± 8.7	82.9 ± 8.2	0.70
T2 – Immediately after intubation	97.0 ± 12.1	83.2 ± 10.3	<0.001	108.0 ± 12.6	97.6 ± 10.8	<0.001
T3 – 1 min after intubation	92.8 ± 11.5	81.9 ± 9.6	<0.001	103.5 ± 11.9	94.2 ± 10.0	<0.001
T4 – 3 min after intubation	87.6 ± 10.2	79.8 ± 8.9	0.001	99.1 ± 10.8	91.4 ± 9.5	0.002
T5 – 5 min after intubation	82.9 ± 9.8	78.6 ± 8.4	0.03	95.6 ± 9.9	89.8 ± 8.9	0.01

HR and MAP decreased at T1 following induction in both groups, reflecting expected pharmacologic effects. However, a pronounced rise occurred at T2 in Group L, with HR increasing by nearly 19 bpm from baseline and MAP rising by 16.8 mmHg. In contrast, Group E demonstrated significantly smaller increases, with HR rising by only 4.2 bpm and MAP by

6.9 mmHg ($p < 0.001$ for both). Esmolol maintained better control at T3 and T4 as well, while values began normalizing toward baseline in both groups by T5. The hemodynamic curves demonstrated a consistently lower sympathetic surge in the esmolol group.

Peak Changes and Comparative Outcomes

Peak changes from baseline confirm the superior attenuation of esmolol. Lignocaine allowed larger HR and MAP spikes following intubation.

Table – III: Peak Change in HR and MAP from Baseline (T2)

Parameter	Group L	Group E	p-value
Peak HR increase (bpm)	+18.6 ± 9.4	+4.2 ± 7.8	<0.001
Peak MAP increase (mmHg)	+16.8 ± 10.2	+6.9 ± 8.5	<0.001

At the moment of greatest physiological stress (T2), esmolol suppressed the increase in HR by approximately 77% compared with lignocaine. MAP elevation was reduced by nearly 60%. These findings emphasize that esmolol provides a much more effective blunting of sympathetic response compared with lignocaine, with large and statistically robust differences.

Categorical Hemodynamic Events

The incidence of clinically significant tachycardia was markedly lower in patients receiving esmolol, while bradycardia events remained minimal and manageable.

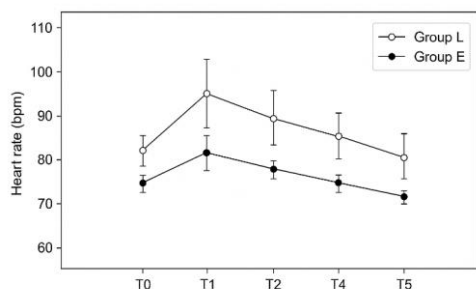


Figure 1. Heart rate trend (T0-T5)

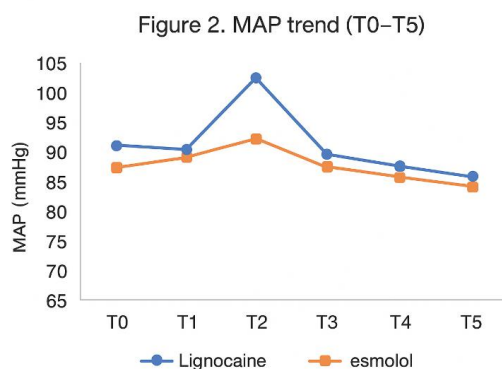
Figure – 2: Heart rate trend (T0-T5)

Figure – 3: MAP trend (T0-T5)

Table – IV: Categorical Adverse Hemodynamic Events

Event	Group L (n=50)	Group E (n=50)	p-value
Tachycardia (>20% HR rise)	28 (56%)	6 (12%)	<0.001
Bradycardia (HR <50 bpm)	0 (0%)	2 (4%)	0.15
Hypotension (MAP <65 mmHg)	1 (2%)	2 (4%)	0.56
Arrhythmias	0	0	–
Bronchospasm	0	0	–

Tachycardia occurred in more than half of the lignocaine group but in only 12% of the esmolol group, a highly significant difference ($p < 0.001$). Two cases of transient bradycardia were noted in the esmolol group; both resolved promptly with atropine. No clinically significant arrhythmias or bronchospasm occurred in either group. Hypotension was uncommon and mild in both groups, requiring no vasopressor therapy.

Adverse Events and Safety

No major complications occurred. Esmolol was associated with mild, short-lived bradycardia in two cases, while lignocaine caused no bradycardia. Overall adverse effect profiles were minimal and did not differ significantly between groups. Both drugs were well tolerated. The safety profile remained acceptable, with no patient requiring ICU transfer, re-intubation, or prolonged monitoring. The esmolol-related bradycardia events were minor and expected based on drug pharmacology.

DISCUSSION

In this study involving 100 patients undergoing elective surgical procedures, esmolol demonstrated significantly superior attenuation of hemodynamic responses to laryngoscopy and intubation compared with lignocaine. The findings build upon a growing body of evidence suggesting that β_1 -selective blockade provides more effective modulation of sympathetic activation than sodium channel inhibition alone. The marked reductions in HR and MAP observed in the esmolol group at all post-intubation time points highlight its strong capacity to blunt catecholamine-mediated cardiovascular surges. The rise in HR and MAP immediately after intubation (T2) is considered the critical moment of hemodynamic stress. In our study, patients receiving lignocaine exhibited a substantial increase in HR (mean +18.6 bpm) and MAP (mean +16.8 mmHg), consistent with earlier reports that lignocaine, at conventional doses, offers only partial suppression of the reflex response.^[2,4] In contrast, esmolol markedly reduced HR and MAP elevation, aligning with previous findings demonstrating its rapid and predictable β -blocking action.^[6] Studies by Kovac et al. and Singh et al. similarly reported that esmolol doses of 0.5–1 mg/kg effectively suppressed tachycardia and hypertension during laryngoscopy.^[7,8] The physiological rationale for these differences is well established. Lignocaine suppresses airway reflexes and provides mild sympathetic dampening, but it does not directly counteract the β -adrenergic surge triggered by noxious airway stimulation. Esmolol's competitive antagonism of β_1 -receptors provides more direct modulation of cardiovascular responses, resulting in lower myocardial oxygen demand and improved hemodynamic stability.^[3] This mechanistic advantage likely explains the superior performance of esmolol observed in this study. An important clinical consideration is safety. Although esmolol was associated with two cases of transient bradycardia, both were mild and easily treated, consistent with previous literature indicating that the drug's ultra-short half-life minimizes risk

when appropriately titrated.^[9] Lignocaine did not cause bradycardia in our study, but its overall ability to prevent tachycardia was limited. Hypotension was infrequent in both groups, suggesting that both drugs are safe when administered at standard doses. The incidence of tachycardia was significantly higher in the lignocaine group (56%) than in the esmolol group (12%), further strengthening the argument for esmolol as the preferred agent. Tachycardia during induction is particularly concerning in patients with coronary artery disease, as even brief episodes may reduce diastolic coronary perfusion and precipitate ischemia.^[10] Esmolol's favorable profile in reducing HR makes it especially suitable for such high-risk populations. The findings from this study are consistent with global data, although variations exist in reported effect sizes. Some studies have shown modest efficacy of lignocaine when administered both intravenously and via laryngotracheal sprays.^[11–13] However, most modern trials favor esmolol due to its dose-dependent and reliable effects. Our study contributes valuable evidence from a South Asian setting, reinforcing that esmolol remains effective across diverse patient populations. A limitation of this study is the focus on ASA I and II patients, which may limit generalizability to higher-risk patients. Additionally, only single-dose regimens were evaluated; continuous or titrated infusions may yield different outcomes. Nonetheless, the findings offer practical guidance for routine anesthesia practice. In conclusion, esmolol provides significantly better attenuation of cardiovascular responses to laryngoscopy and intubation compared with lignocaine, with minimal adverse effects. Its use should be strongly considered, particularly in patients where hemodynamic stability is crucial.

CONCLUSION

In this randomized study of 100 adults undergoing elective surgery, esmolol 1.0 mg·kg⁻¹ IV given before laryngoscopy provided superior attenuation of increases in HR and MAP associated with intubation compared with lignocaine 1.5 mg·kg⁻¹ IV. Esmolol was well tolerated, with only transient bradycardia in a minority of patients. Esmolol may be a preferred agent when brief, effective control of the intubation response is required.

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