

Comparative Evaluation of Intravenous Lignocaine and Esmolol for Attenuating the Hemodynamic Response to Laryngoscopy and Endotracheal Intubation: A Prospective Randomized Comparative Study

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ABSTRACT

Background: Direct laryngoscopy and endotracheal intubation cause transient sympathetic stimulation, resulting in tachycardia, hypertension, and increased myocardial oxygen demand. Although generally tolerated by healthy individuals, these responses may cause serious cardiovascular complications in susceptible patients. Intravenous lignocaine and esmolol are commonly used to attenuate these hemodynamic changes. This study compared their effectiveness in controlling the hemodynamic response to laryngoscopy and intubation. **Objective:** To compare intravenous lignocaine and esmolol in attenuating hemodynamic responses to direct laryngoscopy and endotracheal intubation in adults undergoing elective surgery under general anaesthesia. **Methods:** This prospective randomized comparative study was conducted at the Department of Anaesthesiology, Chittagong Medical College Hospital, Chattogram, Bangladesh, from January to December 2025. One hundred ASA I–II patients aged 18–60 years were randomized equally into Group L (n=50), receiving intravenous lignocaine 1.5 mg/kg, and Group E (n=50), receiving intravenous esmolol 1.5 mg/kg, three minutes before laryngoscopy. Heart rate, systolic and diastolic blood pressure, mean arterial pressure, and rate pressure product were recorded at baseline, after induction, immediately after intubation and at 1, 3, 5, and 10 minutes. Adverse events were documented. Data were analyzed using SPSS version 26.0; $p < 0.05$ was considered significant. **Results:** Baseline characteristics were comparable between groups ($p > 0.05$). Immediately after intubation, esmolol produced significantly lower heart rate (88.4 ± 8.7 vs. 101.7 ± 10.5 beats/min) and

systolic blood pressure (132.8 ± 10.6 vs. 149.6 ± 12.7 mmHg) than lignocaine (both $p < 0.001$). Significant reductions in diastolic pressure, mean arterial pressure and rate pressure product were also observed with esmolol during the first five minutes ($p < 0.001$). Excellent hemodynamic control was achieved in 76.0% versus 34.0% ($p < 0.001$). **Conclusion:** Both agents attenuated intubation-related hemodynamic responses, but esmolol provided superior control with an acceptable safety profile.

Keywords: Laryngoscopy; Endotracheal Intubation; Esmolol; Intravenous Lignocaine; Hemodynamic Response; Heart Rate; Blood Pressure; General Anaesthesia; Randomized Comparative Study.

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INTRODUCTION

Laryngoscopy and endotracheal intubation are indispensable procedures for securing the airway during the administration of general anaesthesia. Despite their routine use, these procedures are associated with a marked sympathetic nervous system response caused by mechanical stimulation of the supraglottic and infraglottic structures. This reflex response results in the release of endogenous catecholamines leading to transient tachycardia, hypertension, increased myocardial contractility and elevated myocardial oxygen consumption. In healthy individuals, these hemodynamic changes are usually short-lived and clinically insignificant; however, in patients with cardiovascular, cerebrovascular or intracranial diseases, they may precipitate serious complications such as myocardial ischemia, cardiac arrhythmias, left ventricular failure, pulmonary edema or

intracranial hemorrhage. Consequently, attenuation of the cardiovascular response to laryngoscopy and endotracheal intubation remains an important objective in modern anaesthetic practice.^[1,2] The magnitude of the hemodynamic response depends on several factors, including the duration and difficulty of laryngoscopy, depth of anaesthesia, patient age, pre-existing cardiovascular status and the pharmacological agents used during induction. The pressor response generally begins within a few seconds after laryngoscopy reaches its maximum intensity within one minute after tracheal intubation and gradually subsides within five to ten minutes. Although this response is transient, it may substantially increase myocardial oxygen demand while reducing coronary perfusion, thereby increasing the risk of adverse perioperative cardiovascular events in susceptible patients.^[2,3] Various pharmacological strategies have been

investigated to suppress these undesirable hemodynamic responses. These include opioids, vasodilators, calcium channel blockers, α_2 -adrenergic agonists, magnesium sulfate, local anaesthetic agents and β -adrenergic receptor antagonists. Among these agents, intravenous lignocaine and esmolol are widely used because of their rapid onset of action, ease of administration, favorable pharmacokinetic properties and relatively low incidence of adverse effects. Both drugs are inexpensive and readily available in routine anaesthetic practice, making them attractive options for controlling the sympathetic response associated with airway manipulation.^[3,4] Lignocaine is an amide-type local anaesthetic with membrane-stabilizing and class IB antiarrhythmic properties. When administered intravenously before laryngoscopy, it suppresses airway reflexes, reduces neuronal conduction and

decreases sympathetic stimulation arising from tracheal instrumentation. In addition to attenuating the pressor response, intravenous lignocaine has been shown to reduce coughing, bronchospasm and postoperative airway discomfort. However, several studies have reported inconsistent results regarding its ability to completely suppress increases in heart rate and arterial blood pressure during endotracheal intubation, particularly in patients with heightened sympathetic activity.^[4,5] Esmolol is an ultra-short-acting, cardioselective β 1-adrenergic receptor blocker characterized by rapid onset and a plasma elimination half-life of approximately nine minutes due to metabolism by red blood cell esterases. It effectively reduces heart rate, myocardial contractility, cardiac output and arterial blood pressure during periods of intense sympathetic stimulation without producing prolonged postoperative cardiovascular depression. Owing to these pharmacological characteristics, esmolol has become one of the most frequently studied β -blockers for attenuating the cardiovascular response to laryngoscopy and endotracheal intubation. Previous randomized controlled trials and systematic reviews have demonstrated that esmolol effectively limits tachycardia and hypertension while maintaining an acceptable safety profile in most patients.^[5,6] Several comparative studies have evaluated lignocaine and esmolol using different dosages, timing of administration and anaesthetic techniques, yielding variable results. Differences in study design, patient characteristics and outcome measures have contributed to inconsistencies in the available literature. Furthermore, limited evidence exists regarding the comparative efficacy of these agents in Bangladeshi patients undergoing elective surgery under standardized anaesthetic conditions. Therefore, additional prospective randomized studies are warranted to provide locally relevant evidence for optimizing perioperative cardiovascular management.^[6,7] The present prospective randomized comparative study was designed to evaluate and compare the effectiveness of intravenous lignocaine and intravenous esmolol in attenuating the hemodynamic responses to direct laryngoscopy and endotracheal intubation among adult patients undergoing elective surgery under general anaesthesia. The findings of this study are expected to assist anaesthesiologists in selecting the most appropriate pharmacological agent for improving perioperative cardiovascular stability and enhancing patient safety during airway management.^[7,8] To compare the efficacy of intravenous lignocaine and intravenous esmolol in

attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation.

METHODS & MATERIALS

Study Design and Setting

This prospective randomized comparative study was conducted in the Department of Anaesthesiology, Chittagong Medical College Hospital, Chattogram, Bangladesh, from January 2025 to December 2025. The study aimed to compare the efficacy of intravenous lignocaine and intravenous esmolol in attenuating the hemodynamic responses associated with direct laryngoscopy and endotracheal intubation during elective surgery under general anaesthesia. Written informed consent was obtained from all participants before enrolment.

Study Population

A total of 100 adult patients scheduled for elective surgical procedures requiring endotracheal intubation under general anaesthesia were enrolled. Patients aged 18–60 years of either sex with American Society of Anesthesiologists (ASA) physical status I or II were eligible for inclusion. Patients were excluded if they had an anticipated difficult airway, uncontrolled hypertension, ischemic heart disease, cardiac arrhythmia, congestive heart failure, bronchial asthma, chronic β -blocker therapy, hepatic or renal dysfunction, pregnancy, obesity (BMI >35 kg/m²), known hypersensitivity to lignocaine or esmolol or were undergoing emergency surgery.

Randomization and Study Groups

Participants were randomly allocated in a 1:1 ratio into two groups of 50 patients each using a computer-generated randomization sequence. Allocation was concealed using sequentially prepared, sealed, opaque envelopes.

Patients in Group L received intravenous lignocaine 1.5 mg/kg, whereas those in Group E received intravenous esmolol 1.5 mg/kg. The assigned study medication was diluted with normal saline to a total volume of 10 mL and administered intravenously over approximately 30 seconds, three minutes before laryngoscopy.

Anaesthetic Technique and Monitoring

All patients underwent standard pre-anaesthetic evaluation and fasting according to institutional guidelines. On arrival in the operating room, standard monitoring was established, including electrocardiography (ECG), non-invasive blood pressure (NIBP), pulse oximetry (SpO₂) and continuous heart rate monitoring. Baseline hemodynamic measurements were recorded after five minutes of resting in a standardized position. Premedication consisted of intravenous ondansetron 4 mg and fentanyl 2 μ g/kg. General anaesthesia was induced

with propofol 2 mg/kg and neuromuscular blockade was achieved using atracurium 0.5 mg/kg. Direct laryngoscopy was performed with a Macintosh laryngoscope by an experienced anaesthesiologist and tracheal intubation was completed within 20 seconds whenever possible. Anaesthesia was maintained with oxygen, nitrous oxide, and isoflurane with intermittent doses of atracurium administered as required.

Outcome Measures

The primary outcome measure was the change in heart rate following laryngoscopy and endotracheal intubation. Secondary outcome measures included systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), rate pressure product (RPP), oxygen saturation and perioperative adverse events. Hemodynamic variables were recorded at baseline, after induction of anaesthesia, immediately after endotracheal intubation and at 1, 3, 5 and 10 minutes following intubation. Adverse events, including bradycardia, hypotension, arrhythmia, bronchospasm and oxygen desaturation, were documented throughout the perioperative period.

Statistical Analysis

Data were entered into Microsoft Excel and subsequently analyzed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean $\pm$ standard deviation (SD), while categorical variables were presented as frequencies and percentages. Between-group comparisons of continuous variables were performed using the independent Student's *t*-test. Changes in hemodynamic parameters over time within each group were assessed using repeated-measures analysis of variance (ANOVA). Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. All statistical tests were two-tailed and a *p*-value <0.05 was considered statistically significant.

RESULTS

A total of 100 patients were enrolled and completed the prospective randomized comparative study. Participants were equally allocated to Group L (intravenous lignocaine, *n*=50) and Group E (intravenous esmolol, *n*=50) with no exclusions or protocol violations. Baseline demographic characteristics are presented in Table 1. Mean age was 39.4 $\pm$ 10.6 years in Group L and 40.2 $\pm$ 9.8 years in Group E (*p*=0.692). Male participants comprised 56.0% and 60.0%, respectively (*p*=0.684). Mean body weight, height, and BMI were also comparable between groups (all *p*>0.05). Thus, both groups were well matched at baseline, minimizing potential demographic confounding.

Table I
Demographic Characteristics of the Study Population (n=100).

Variables	Group L (n=50)	Group E (n=50)	t/ χ^2	p-value
Age (years), Mean $\pm$ SD	39.4 $\pm$ 10.6	40.2 $\pm$ 9.8	0.40	0.692
Male, n (%)	28 (56.0)	30 (60.0)	0.17	0.684
Female, n (%)	22 (44.0)	20 (40.0)		
Weight (kg), Mean $\pm$ SD	63.8 $\pm$ 8.5	64.7 $\pm$ 8.1	0.54	0.594
Height (cm), Mean $\pm$ SD	162.8 $\pm$ 7.2	163.5 $\pm$ 6.9	0.50	0.618
BMI (kg/m ²), Mean $\pm$ SD	24.5 $\pm$ 2.8	24.8 $\pm$ 2.9	0.50	0.618

As shown in *Table II*, most participants belonged to ASA Physical Status I, comprising 62.0% of patients in the lignocaine group and 58.0% in the esmolol

group. The remaining patients were classified as ASA II, representing 38.0% and 42.0%, respectively. The difference in ASA distribution between the two groups

was not statistically significant (p=0.682), confirming comparable preoperative physical status.

Table II
Distribution of ASA Physical Status.

ASA Physical Status	Group L (n=50)	Group E (n=50)	χ^2	p-value
ASA I	31 (62.0%)	29 (58.0%)		
ASA II	19 (38.0%)	21 (42.0%)	0.17	0.682

Baseline cardiovascular parameters were similar between the two groups. The mean heart rate was 80.4 $\pm$ 8.3 beats/min in Group L and 79.8 $\pm$ 7.9 beats/min in Group E (p=0.711) (*Table III*). Mean systolic blood pressure was 124.6 $\pm$ 9.8 mmHg and

123.7 $\pm$ 10.2 mmHg, respectively (p=0.653), while mean diastolic blood pressure was 78.5 $\pm$ 7.1 mmHg versus 77.9 $\pm$ 6.9 mmHg (p=0.664). Mean arterial pressure and oxygen saturation were likewise comparable. These findings

demonstrate that the two groups had similar baseline hemodynamic status prior to administration of the study drugs.

Table III
Baseline Hemodynamic Parameters.

Parameter	Group L	Group E	t-value	p-value
Heart Rate (beats/min)	80.4 $\pm$ 8.3	79.8 $\pm$ 7.9	0.37	0.711
Systolic BP (mmHg)	124.6 $\pm$ 9.8	123.7 $\pm$ 10.2	0.45	0.653
Diastolic BP (mmHg)	78.5 $\pm$ 7.1	77.9 $\pm$ 6.9	0.44	0.664
Mean Arterial Pressure (mmHg)	93.9 $\pm$ 6.8	93.2 $\pm$ 6.4	0.52	0.602
Oxygen Saturation (%)	99.2 $\pm$ 0.8	99.3 $\pm$ 0.7	0.66	0.511

Table IV demonstrates heart rate changes following induction and intubation. Both groups showed a slight reduction after induction. Heart rate increased

significantly after laryngoscopy and intubation, with a greater rise in the lignocaine group than esmolol group (101.7 $\pm$ 10.5 vs. 88.4 $\pm$ 8.7 beats/min;

p<0.001). Significant differences persisted at 1, 3 and 5 minutes confirming superior tachycardia control with esmolol.

Table IV
Comparison of Heart Rate at Different Time Intervals (beats/min).

Time	Group L	Group E	p-value
Baseline	80.4 $\pm$ 8.3	79.8 $\pm$ 7.9	0.711
After induction	75.8 $\pm$ 7.4	72.6 $\pm$ 6.8	0.026*
Immediately after intubation	101.7 $\pm$ 10.5	88.4 $\pm$ 8.7	<0.001*
1 minute	99.3 $\pm$ 9.8	86.6 $\pm$ 8.1	<0.001*
3 minutes	92.5 $\pm$ 8.7	81.8 $\pm$ 7.6	<0.001*
5 minutes	85.9 $\pm$ 7.9	78.4 $\pm$ 6.8	<0.001*
10 minutes	80.7 $\pm$ 7.2	78.9 $\pm$ 6.7	0.198

*Significant at p<0.05.

Table V presents the comparison of systolic blood pressure between the two study groups. Baseline systolic blood pressure was similar in both groups. Following induction, systolic blood pressure decreased modestly without significant intergroup difference. Immediately after

laryngoscopy and intubation, systolic blood pressure increased markedly in both groups but was significantly lower in patients receiving esmolol (132.8 $\pm$ 10.6 mmHg) than in those receiving lignocaine (149.6 $\pm$ 12.7 mmHg) (p<0.001). Significant differences persisted at 1, 3 and 5 minutes

after intubation. At 10 minutes, systolic blood pressure returned close to baseline values in both groups with no statistically significant difference. These findings indicate that esmolol was more effective in attenuating the systolic pressor response.

Table V
Comparison of Systolic Blood Pressure (mmHg).

Time	Group L	Group E	p-value
Baseline	124.6 ± 9.8	123.7 ± 10.2	0.653
After induction	114.8 ± 8.6	111.5 ± 8.2	0.051
Immediately after intubation	149.6 ± 12.7	132.8 ± 10.6	<0.001*
1 minute	146.2 ± 11.9	129.5 ± 10.2	<0.001*
3 minutes	137.8 ± 10.8	124.8 ± 9.5	<0.001*
5 minutes	129.6 ± 9.4	121.7 ± 8.8	<0.001*
10 minutes	124.8 ± 8.6	122.8 ± 8.2	0.233

*Significant at $p < 0.05$.

Table VI compares diastolic blood pressure changes following intubation. Baseline values were comparable. Both groups showed a reduction after induction

followed by a significant rise after intubation. The increase was greater with lignocaine than esmolol (93.8 ± 8.6 vs. 84.4 ± 7.5 mmHg; $p < 0.001$). Significant

differences persisted at 1, 3 and 5 minutes confirming superior attenuation with esmolol.

Table VI
Comparison of Diastolic Blood Pressure (mmHg).

Time	Group L	Group E	p-value
Baseline	78.5 ± 7.1	77.9 ± 6.9	0.664
After induction	71.6 ± 6.8	69.8 ± 6.2	0.176
Immediately after intubation	93.8 ± 8.6	84.4 ± 7.5	<0.001*
1 minute	91.2 ± 8.2	82.8 ± 7.2	<0.001*
3 minutes	85.4 ± 7.8	79.4 ± 6.8	<0.001*
5 minutes	81.2 ± 7.3	77.2 ± 6.4	0.004*
10 minutes	78.6 ± 6.7	77.4 ± 6.5	0.364

*Significant at $p < 0.05$.

Table VII demonstrates MAP changes following induction and intubation. Baseline MAP was comparable between groups (93.9 ± 6.8 vs. 93.2 ± 6.4 mmHg;

$p = 0.602$). Following intubation, MAP increased significantly in both groups, with a greater rise in the lignocaine group (112.4 ± 8.8 vs. 100.5 ± 7.9 mmHg; $p < 0.001$).

Significant differences persisted at 1, 3 and 5 minutes, confirming superior attenuation with esmolol.

Table VII
Comparison of Mean Arterial Pressure (MAP) at Different Time Intervals (mmHg).

Time Interval	Group L (n=50) Mean ± SD	Group E (n=50) Mean ± SD	p-value
Baseline	93.9 ± 6.8	93.2 ± 6.4	0.602
After induction	86.0 ± 6.1	83.7 ± 5.9	0.060
Immediately after intubation	112.4 ± 8.8	100.5 ± 7.9	<0.001*
1 minute	109.5 ± 8.3	98.4 ± 7.6	<0.001*
3 minutes	103.2 ± 7.5	94.5 ± 6.9	<0.001*
5 minutes	97.3 ± 6.8	92.1 ± 6.2	<0.001*
10 minutes	93.7 ± 6.3	92.5 ± 5.8	0.329

*Significant at $p < 0.05$.

Table VIII compares RPP, an indirect indicator of myocardial oxygen consumption and cardiac workload. Baseline values were comparable.

Following intubation, RPP increased markedly in both groups but remained significantly lower with esmolol than lignocaine ($11,738 \pm 1,508$ vs. $15,198 \pm$

$1,785$; $p < 0.001$). Differences persisted at 1, 3 and 5 minutes, indicating superior attenuation of myocardial workload with esmolol.

Table VIII
Comparison of Rate Pressure Product (RPP).

Time Interval	Group L Mean ± SD	Group E Mean ± SD	p-value
Baseline	10017 ± 1398	9873 ± 1342	0.596
Immediately after intubation	15198 ± 1785	11738 ± 1508	<0.001*
1 minute	14562 ± 1714	11214 ± 1452	<0.001*
3 minutes	12753 ± 1512	10186 ± 1326	<0.001*
5 minutes	11142 ± 1325	9538 ± 1187	<0.001*
10 minutes	10074 ± 1262	9698 ± 1208	0.129

*Significant at $p < 0.05$.

The percentage increase in heart rate immediately following endotracheal intubation was significantly higher in the lignocaine group than in the esmolol group. Patients receiving lignocaine experienced a

mean increase of 26.5%, whereas those receiving esmolol showed only a 10.8% increase ($p<0.001$). This demonstrates that intravenous esmolol provided substantially better suppression of reflex tachycardia

associated with laryngoscopy and tracheal intubation (*Table IX*).

Table IX
Percentage Increase in Heart Rate Immediately After Intubation.

Variable	Group L (n=50)	Group E (n=50)	p-value
Percentage increase in Heart Rate (%)	26.5 ± 7.4	10.8 ± 5.9	<0.001*

*Significant at $p<0.05$.

Table X the percentage increase in systolic blood pressure following airway instrumentation. Patients treated with intravenous lignocaine demonstrated a

mean increase of 20.1% from baseline, whereas the increase in the esmolol group was only 7.4%. The difference between the groups was highly significant ($p<0.001$).

These findings further support the superior efficacy of esmolol in minimizing the pressor response during laryngoscopy and endotracheal intubation.

Table X
Percentage Increase in Systolic Blood Pressure Immediately After Intubation.

Variable	Group L (n=50)	Group E (n=50)	p-value
Percentage increase in SBP (%)	20.1 ± 6.2	7.4 ± 5.1	<0.001*

*Significant at $p<0.05$.

Table XI summarizes perioperative adverse events. Bradycardia occurred in 8.0% of esmolol and 2.0% of lignocaine patients, while hypotension occurred in 10.0% and

4.0%, respectively; neither difference was significant. One lignocaine patient developed transient arrhythmia. No bronchospasm or desaturation occurred. All

events were mild and resolved without significant intervention.

Table XI
Comparison of Perioperative Adverse Events.

Adverse Events	Group L (n=50)	Group E (n=50)	p-value
Bradycardia	1 (2.0%)	4 (8.0%)	0.169
Hypotension	2 (4.0%)	5 (10.0%)	0.238
Arrhythmia	1 (2.0%)	0 (0.0%)	0.315
Bronchospasm	0	0	—
Oxygen desaturation	0	0	—
Nausea/Vomiting	2 (4.0%)	1 (2.0%)	0.558

Table XII presents the overall assessment of hemodynamic control achieved with the two study drugs. Excellent hemodynamic stability was observed in 76.0% of patients receiving intravenous esmolol compared with only 34.0% of patients receiving

intravenous lignocaine. Conversely, poor hemodynamic control was noted in 20.0% of the lignocaine group but only 4.0% of the esmolol group. The difference between the two treatment groups was highly statistically significant ($p<0.001$),

indicating that intravenous esmolol provided markedly superior cardiovascular stability during laryngoscopy and endotracheal intubation.

Table XII
Overall Hemodynamic Control Following Laryngoscopy and Intubation.

Hemodynamic Response	Group L (n=50)	Group E (n=50)	p-value
Excellent	17 (34.0%)	38 (76.0%)	<0.001*
Good	23 (46.0%)	10 (20.0%)	
Poor	10 (20.0%)	2 (4.0%)	

*Significant at $p<0.05$.

DISCUSSION

The present prospective randomized comparative study evaluated the effectiveness of intravenous lignocaine and intravenous esmolol in attenuating the hemodynamic responses associated with direct laryngoscopy and endotracheal intubation among adult patients undergoing elective surgery under general anaesthesia. Airway manipulation produces a transient

but marked sympathetic response characterized by tachycardia, hypertension and increased myocardial oxygen demand. Although generally well tolerated in healthy individuals, these changes may have important consequences in patients with hypertension, ischemic heart disease, cerebrovascular disease and other cardiovascular risk factors.^[3] Therefore, attenuation of the pressor response remains

an important objective of anaesthetic practice.

In the present study, both groups were comparable with respect to age, sex, body mass index, ASA physical status and baseline hemodynamic parameters. The absence of statistically significant baseline differences indicates successful randomization and suggests that subsequent differences were primarily

related to the administered study drugs rather than demographic or clinical characteristics. Similar baseline comparability has been reported in previous randomized studies evaluating pharmacological attenuation of the cardiovascular response to laryngoscopy.^[4,5]

Following induction of anaesthesia, both groups demonstrated modest reductions in heart rate and arterial blood pressure, which may be attributed to the cardiovascular depressant effects of propofol and opioid administration. In contrast, direct laryngoscopy and endotracheal intubation produced significant increases in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure in both groups. These findings are consistent with the established physiological response to airway instrumentation, which results from stimulation of sympathetic nerve endings and increased catecholamine release.^[2,6]

The principal finding was that intravenous esmolol provided significantly greater attenuation of the heart-rate response than intravenous lignocaine. Patients receiving esmolol had significantly lower heart rates immediately after intubation and at 1, 3 and 5 minutes. This effect is pharmacologically plausible because esmolol is a cardioselective β_1 -adrenergic receptor antagonist that suppresses sympathetic cardiac stimulation, reduces sinoatrial node activity and decreases myocardial contractility.^[7] Previous studies have similarly demonstrated that esmolol effectively limits reflex tachycardia associated with laryngoscopy and intubation while maintaining acceptable cardiovascular stability.^[8-10]

A comparable pattern was observed for systolic, diastolic and mean arterial pressures. Although lignocaine reduced the magnitude of the pressor response, blood pressure remained significantly higher than in the esmolol group during the early post-intubation period. The superior blood-pressure control achieved with esmolol is consistent with previous randomized trials and systematic reviews suggesting that β -blockers are among the more effective pharmacological agents for controlling both tachycardia and hypertension during airway manipulation.^[9-12] Esmolol reduces heart rate, myocardial contractility and cardiac output, whereas lignocaine primarily suppresses airway reflexes and does not directly antagonize adrenergic stimulation.

Rate-pressure product, an indirect marker of myocardial oxygen demand and cardiac workload, increased significantly after intubation in both groups but remained substantially lower with esmolol. Because an increased rate-pressure product reflects greater myocardial oxygen requirement and may be associated with myocardial

ischemia in susceptible patients, the lower values observed with esmolol suggest a potential advantage in reducing peri-intubation myocardial stress.^[13,14] Previous investigations have likewise reported greater reduction in myocardial oxygen demand with β -blockade compared with intravenous local anaesthetic therapy.^[10,13]

The safety profile of both interventions was satisfactory. Mild transient bradycardia and hypotension occurred somewhat more frequently in the esmolol group; however, these events were self-limiting and did not require pharmacological treatment. No clinically significant arrhythmia, bronchospasm, prolonged hypotension or oxygen desaturation was observed. These findings are consistent with previous evidence describing esmolol as a short-acting β -blocker with rapid metabolism by red blood cell esterases and a short elimination half-life, permitting relatively rapid recovery after discontinuation.^[7,15] Nevertheless, appropriate patient selection remains important, particularly in patients with severe bradycardia, conduction abnormalities, uncompensated heart failure or significant bronchospastic disease.

The clinical implications of these findings are relevant to routine anaesthetic practice. Although lignocaine is inexpensive, widely available and capable of partially reducing airway reflexes and cardiovascular stimulation, esmolol provided more effective overall hemodynamic control in this study. Improved control of heart rate and blood pressure may reduce perioperative cardiovascular stress, particularly during the vulnerable period immediately following tracheal intubation.

STRENGTHS OF THE STUDY

The major strengths of this study include its prospective randomized design, equal group allocation, standardized anaesthetic protocol, identical drug administration approach, predefined assessment intervals and evaluation of multiple clinically relevant hemodynamic parameters. These features minimized selection bias, reduced potential confounding and strengthened the internal validity of the findings.

LIMITATIONS

Several limitations should be acknowledged. This was a single-centre study with a relatively modest sample size of 100 patients, which may limit the generalizability of the findings. Only ASA I–II patients undergoing elective surgery were included; therefore, the results cannot be directly extrapolated to emergency surgery or patients with significant cardiovascular disease. Plasma catecholamine and other stress-hormone concentrations were not measured, preventing direct biochemical assessment of sympathetic activation. In addition,

long-term postoperative cardiovascular outcomes were not evaluated.

CONCLUSION

The present prospective randomized comparative study demonstrated that both intravenous lignocaine and intravenous esmolol attenuated the hemodynamic responses associated with direct laryngoscopy and endotracheal intubation. However, esmolol provided significantly superior control of heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and rate-pressure product during the critical early post-intubation period. Although mild transient bradycardia and hypotension occurred somewhat more frequently with esmolol, these events were clinically insignificant and required no therapeutic intervention. Overall, esmolol provided greater cardiovascular stability with an acceptable safety profile and may therefore be considered a preferable pharmacological option when optimal attenuation of the intubation response is desired.

RECOMMENDATIONS

Intravenous esmolol may be considered in appropriately selected adult patients when effective hemodynamic control during induction of general anaesthesia is required. Careful assessment should precede β -blocker administration, particularly in patients with bradycardia, conduction abnormalities, heart failure or bronchospastic disease. Larger multicentre randomized controlled trials are recommended to confirm these findings and determine the optimal dosing regimen and administration technique. Future studies should also include elderly and high-risk cardiac patients, emergency surgical populations, biochemical markers of sympathetic activation and longer-term perioperative cardiovascular outcomes.

ETHICAL APPROVAL

The study was approved by the Institutional Review Board/Ethical Review Committee of Chittagong Medical College Hospital, Chattogram, Bangladesh and conducted according to the Declaration of Helsinki. Written informed consent was obtained from all participants. Participation was voluntary, with the right to withdraw at any time. Participant confidentiality and anonymity were maintained throughout the study.

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CONFLICT OF INTEREST

The authors declare no conflict of interest regarding the publication of this study.

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