

COMPARATIVE EFFICACY OF INTRAVENOUS ESMOLOL AND INTRAVENOUS LIGNOCAINE FOR ATTENUATION OF HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION DURING GENERAL ANESTHESIA

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation commonly produce a sympathetic haemodynamic response characterized by tachycardia, hypertension, and increased myocardial workload. Esmolol and lignocaine are frequently used to attenuate this response, but their comparative efficacy remains clinically relevant. **Aim:** To compare intravenous esmolol hydrochloride 1.5 mg/kg and intravenous lignocaine hydrochloride 1.5 mg/kg for attenuation of haemodynamic response to laryngoscopy and endotracheal intubation during general anaesthesia. **Materials and Methods:** This prospective randomized comparative study included 60 adult normotensive patients of either sex, aged 18–60 years, belonging to ASA physical status I or II and undergoing elective surgery under general anaesthesia with endotracheal intubation. Patients were randomly allocated into two groups of 30 each. Group E received intravenous esmolol 1.5 mg/kg and Group L received intravenous lignocaine 1.5 mg/kg, 3 minutes before intubation. Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, oxygen saturation, and rate pressure product were recorded at baseline, after premedication, before induction, during laryngoscopy, and at 1, 3, 5, 7, and 10 minutes after intubation. **Results:** Baseline demographic and haemodynamic parameters were comparable between groups. After intubation, heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and rate pressure product were significantly lower in the esmolol group at 1 and 3 minutes ($p < 0.001$), with significant attenuation persisting up to 5 minutes ($p < 0.05$). Values became comparable by 7 and 10 minutes. Oxygen saturation remained stable, and no significant adverse effects were observed. **Conclusion:** Intravenous esmolol 1.5 mg/kg was more effective than intravenous lignocaine 1.5 mg/kg in attenuating the early haemodynamic response to laryngoscopy and endotracheal intubation.

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of general anaesthesia, yet they represent one of the most stimulating moments during induction. Although the procedure is brief, mechanical stimulation of the airway can produce a sudden cardiovascular response, mainly in the form of tachycardia, hypertension, and increased myocardial workload. This response is usually transient in healthy patients, but it remains clinically

relevant because it occurs at a vulnerable phase when stable haemodynamics are desirable.^[1,2]

The haemodynamic response to intubation is primarily mediated through reflex sympathetic activation following stimulation of the upper airway, laryngeal structures, and trachea. This autonomic response is associated with increased heart rate, arterial pressure, and catecholamine release, with the maximum change commonly occurring soon after laryngoscopy and passage of the endotracheal tube. The magnitude of response depends on the intensity and duration of airway stimulation, depth of

anaesthesia, and individual cardiovascular reserve.^[3,4]

In patients with cardiovascular or cerebrovascular risk, this pressor response may have greater clinical consequences. Sudden increases in heart rate and blood pressure increase myocardial oxygen demand and may precipitate myocardial ischaemia, arrhythmia, left ventricular dysfunction, or cerebrovascular complications. Therefore, attenuation of the haemodynamic response to laryngoscopy and intubation is an important objective, particularly in patients with hypertension, coronary artery disease, valvular heart disease, raised intracranial pressure, or aneurysmal disease.^[5,6]

Several pharmacological approaches have been used to blunt this response, including opioids, beta-blockers, alpha-2 agonists, calcium-channel blockers, vasodilators, magnesium sulphate, and lignocaine. However, no single drug is ideal for all patients because each agent differs in onset, duration, haemodynamic selectivity, and adverse-effect profile. The preferred agent should provide rapid and predictable control during the short high-risk period of airway manipulation without causing prolonged hypotension, bradycardia, respiratory depression, or delayed recovery.^[7,8]

Intravenous lignocaine has been widely used for suppression of airway reflexes and attenuation of cardiovascular responses during intubation, whereas esmolol, an ultra-short-acting cardioselective beta-1 blocker, offers more direct control of tachycardia and myocardial workload. Since both agents are commonly used for this purpose, their comparative efficacy remains clinically relevant in routine anaesthesia practice.^[9,10] The present study was conducted to compare intravenous esmolol hydrochloride 1.5 mg/kg with intravenous lignocaine hydrochloride 1.5 mg/kg for attenuation of haemodynamic response to laryngoscopy and endotracheal intubation, using heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, and rate pressure product as outcome measures.

MATERIALS AND METHODS

Study design

This prospective randomized comparative study was conducted among adult patients undergoing elective surgical procedures under general anaesthesia with endotracheal intubation. Written informed consent was obtained from all participants before enrolment.

Study population

A total of 60 healthy normotensive patients of either sex were included. Patients aged 18–60 years, weighing 40–80 kg, belonging to American Society of Anesthesiologists physical status I or II, and scheduled for elective surgery requiring endotracheal intubation were selected. Patients with hypertension, diabetes mellitus, hypotension, baseline bradycardia, anticipated difficult intubation, cardiovascular

disease, renal dysfunction, hepatic dysfunction, respiratory disease, or allergy to the study drugs were excluded. Patients requiring more than one attempt at intubation, those with laryngoscopy lasting more than 15 seconds, and those who developed bucking or hiccoughing during the procedure were also excluded from analysis.

Randomization and study groups

Patients were randomly allocated into two equal groups of 30 patients each using computer-generated random numbers. Group E received intravenous esmolol hydrochloride 1.5 mg/kg diluted with 0.9% normal saline to 10 mL. Group L received intravenous lignocaine hydrochloride 1.5 mg/kg diluted with 0.9% normal saline to 10 mL. The study drug was administered 3 minutes before intubation.

Preoperative assessment

All patients underwent preoperative evaluation on the day before surgery. Detailed history, general examination, systemic examination, airway assessment, and review of routine investigations were performed. Airway assessment was done using Mallampati classification. All patients were kept nil per oral for at least 6 hours before surgery.

Anaesthesia protocol

On arrival in the operation theatre, an intravenous line was secured and Ringer lactate was started at 10–15 mL/kg/hour. Standard monitors were attached, including electrocardiography, non-invasive blood pressure, and pulse oximetry. Baseline haemodynamic parameters were recorded. All patients received glycopyrrolate 0.004 mg/kg, ondansetron 0.08 mg/kg, and midazolam 0.02 mg/kg intravenously 10 minutes before induction. Patients were preoxygenated with 100% oxygen for 3 minutes. Anaesthesia was induced with thiopentone sodium 3–5 mg/kg intravenously until loss of eyelash reflex, followed by succinylcholine 2 mg/kg intravenously to facilitate tracheal intubation. Laryngoscopy and intubation were performed with a standard Macintosh laryngoscope using an appropriate-sized cuffed oral endotracheal tube. Intubation was completed within 15 seconds. After intubation, vecuronium bromide 0.08 mg/kg intravenously was administered, and anaesthesia was maintained with oxygen, nitrous oxide, sevoflurane, and vecuronium as required.

Outcome assessment

Heart rate, systolic blood pressure, diastolic blood pressure, oxygen saturation, mean arterial pressure, and rate pressure product were recorded at baseline, after premedication, before induction, during laryngoscopy, and at 1, 3, 5, 7, and 10 minutes after laryngoscopy and intubation. Mean arterial pressure and rate pressure product were calculated from the recorded haemodynamic variables.

Safety monitoring

All patients were observed intraoperatively for adverse effects such as hypotension, bradycardia, bronchospasm, allergic reaction, pruritus, and electrocardiographic changes. Bradycardia was planned to be treated with intravenous atropine, and

hypotension was planned to be managed with intravenous fluids.

Statistical analysis

Data were entered in Microsoft Excel and analysed using appropriate statistical tests. Continuous variables were expressed as mean \pm standard deviation, and categorical variables were expressed as number and percentage. Intergroup comparison of continuous variables was performed using unpaired Student's t-test, while categorical variables were compared using chi-square test. A p-value <0.05 was considered statistically significant, and a p-value <0.001 was considered highly significant.

RESULTS

Table 1 shows that both groups were comparable at baseline. The mean age was 37.83 years in Group E and 35.40 years in Group L, while mean body weight was 64.40 kg and 62.87 kg, respectively. Sex distribution was almost similar, with 15 males and 15 females in Group E and 14 males and 16 females in Group L. General surgery was the commonest procedure in both groups (40.0% vs 43.3%), and no significant baseline difference was observed.

Table 1: Baseline demographic and operative profile of study participants

Variable	Group E: Esmolol (n=30)	Group L: Lignocaine (n=30)	p-value
Age group, years			
• ≤ 30	12 (40.0%)	14 (46.7%)	
• 31-40	4 (13.3%)	5 (16.7%)	
• >40	14 (46.7%)	11 (36.7%)	
Mean age, years	37.83 \pm 14.03	35.40 \pm 14.34	0.51
Sex			
• Male	15 (50.0%)	14 (46.7%)	0.79
• Female	15 (50.0%)	16 (53.3%)	
Weight, kg	64.40 \pm 11.20	62.87 \pm 10.29	0.583
Type of surgery			
• General surgery	12 (40.0%)	13 (43.3%)	
• ENT surgery	8 (26.7%)	7 (23.3%)	
• Gynaecological surgery	7 (23.3%)	7 (23.3%)	
• Orthopaedic surgery	3 (10.0%)	3 (10.0%)	



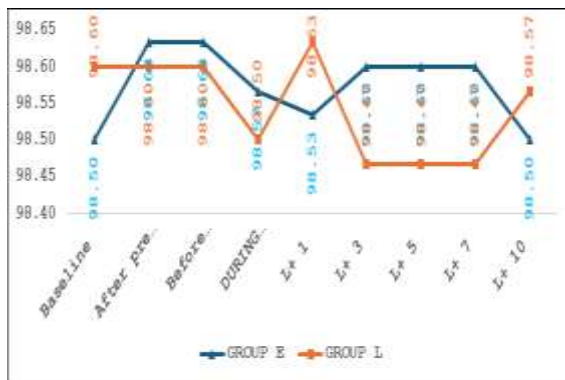
Graph 1: Comparison of Heart rate between both groups

Table 2 shows that systolic and diastolic blood pressures were comparable between the two groups at baseline, after premedication, before induction, and during laryngoscopy. After intubation, both SBP and DBP were significantly lower in Group E than Group L at L+1 minute and L+3 minutes ($p < 0.001$), with a significant difference persisting at L+5 minutes ($p < 0.05$). By L+7 and L+10 minutes, both groups again became comparable. Similarly, Graph 1 shows that heart rate followed the same pattern, with Group E showing better attenuation of tachycardia during the early post-intubation period, especially at L+1 and L+3 minutes.

Table 2: Comparison of systolic and diastolic blood pressure between both groups at different time intervals

Time interval	SBP Group E	SBP Group L:	p-value	DBP Group E:	DBP Group L:	p-value
Baseline	118.27 \pm 8.13	118.93 \pm 7.55	>0.05	76.13 \pm 5.41	76.07 \pm 5.21	>0.05
After premedication	118.40 \pm 8.76	119.13 \pm 8.25	>0.05	75.60 \pm 5.24	75.80 \pm 5.42	>0.05
Before induction	114.20 \pm 8.21	115.00 \pm 7.53	>0.05	74.00 \pm 5.09	74.07 \pm 5.19	>0.05
During laryngoscopy	128.67 \pm 6.83	129.13 \pm 5.62	>0.05	82.87 \pm 5.98	85.53 \pm 4.86	>0.05
L+1 min	131.00 \pm 7.00	137.13 \pm 6.60	<0.001	85.67 \pm 6.60	91.27 \pm 5.45	<0.001
L+3 min	125.13 \pm 6.62	131.07 \pm 5.98	<0.001	80.07 \pm 5.50	85.47 \pm 5.20	<0.001
L+5 min	121.33 \pm 6.24	124.60 \pm 6.11	<0.05	77.60 \pm 5.13	80.40 \pm 4.83	<0.05
L+7 min	120.67 \pm 6.31	122.60 \pm 6.26	>0.05	76.53 \pm 5.28	78.33 \pm 4.73	>0.05
L+10 min	118.40 \pm 7.78	121.07 \pm 6.74	>0.05	76.27 \pm 4.42	77.40 \pm 4.37	>0.05

Values are expressed as mean \pm SD. SBP: systolic blood pressure; DBP: diastolic blood pressure; L+1, L+3, L+5, L+7 and L+10 indicate minutes after laryngoscopy and endotracheal intubation.



Graph 2: Comparison of Oxygen saturation between both the groups

Table 3 shows that MAP and rate pressure product were comparable between both groups at baseline, after premedication, before induction, and during laryngoscopy. After intubation, both parameters were significantly lower in Group E compared with Group L at L+1 and L+3 minutes ($p < 0.001$), with the difference remaining significant at L+5 minutes ($p < 0.05$). By L+7 and L+10 minutes, the values became comparable again. Graph 2 shows that SpO₂ remained stable throughout the observation period in both groups, with no clinically significant desaturation.

Table 3: Comparison of mean arterial pressure and rate pressure product between both groups at different time intervals

Time interval	MAP Group E:	MAP Group L:	p-value	RPP Group E:	RPP Group L:	p-value
Baseline	90.18 ± 3.54	90.36 ± 3.81	>0.05	9066.67 ± 810.07	9128.53 ± 867.64	>0.05
After premedication	89.87 ± 3.65	90.24 ± 4.04	>0.05	9767.33 ± 923.52	9859.20 ± 935.23	>0.05
Before induction	87.40 ± 3.60	87.71 ± 3.98	>0.05	9100.00 ± 830.09	9240.53 ± 846.58	>0.05
During laryngoscopy	98.13 ± 4.32	100.07 ± 3.55	>0.05	11022.40 ± 807.77	11289.20 ± 670.57	>0.05
L+1 min	100.97 ± 4.70	106.56 ± 4.26	<0.001	11614.80 ± 866.50	12750.80 ± 663.33	<0.001
L+3 min	95.09 ± 4.12	100.67 ± 3.94	<0.001	10402.67 ± 726.89	11403.33 ± 601.23	<0.001
L+5 min	92.18 ± 3.77	95.13 ± 3.42	<0.05	9798.13 ± 754.56	10404.53 ± 692.27	<0.05
L+7 min	91.24 ± 4.18	93.09 ± 3.39	>0.05	9498.67 ± 795.14	9856.80 ± 770.03	>0.05
L+10 min	90.31 ± 3.57	91.96 ± 3.27	>0.05	9110.53 ± 922.59	9506.40 ± 801.58	>0.05

Values are expressed as mean ± SD. MAP: mean arterial pressure; RPP: rate pressure product; L+1, L+3, L+5, L+7 and L+10 indicate minutes after laryngoscopy and endotracheal intubation.

DISCUSSION

The present study compared intravenous esmolol 1.5 mg/kg with intravenous lignocaine 1.5 mg/kg for attenuation of haemodynamic response to laryngoscopy and endotracheal intubation. Both groups were similar at baseline in terms of mean age, body weight, sex distribution and type of surgery, which allowed a valid comparison of post-intubation haemodynamic changes. The main finding of the study was that esmolol produced better attenuation of heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and rate pressure product than lignocaine, particularly at 1 and 3 minutes after laryngoscopy and intubation, with the effect persisting up to 5 minutes.

Heart rate response was better controlled in the esmolol group. In the present study, heart rate was comparable between both groups before intubation. After intubation, the maximum rise occurred at L+1 minute, where mean heart rate was 88.67 beats/min in Group E and 93.07 beats/min in Group L. At L+3 minutes, heart rate remained lower in Group E than Group L, and the difference was highly significant at both L+1 and L+3 minutes. The difference persisted at L+5 minutes, after which the values became comparable. This pattern is close to the findings of Rama Rao et al,^[11] who observed that heart rate remained better controlled in the esmolol group,

while the lignocaine group showed a rise of more than 20% at 1 and 3 minutes after intubation. Singh et al,^[12] also reported a much smaller rise in heart rate with esmolol, with only 1.50% rise at 1 minute compared with 26.00% in the lidocaine group. Goyal et al,^[13] similarly found significantly better heart rate control with esmolol at 1, 3, 5 and 10 minutes after intubation. These findings support the present observation that esmolol is more effective than lignocaine in controlling the tachycardic component of intubation response.

Systolic and diastolic blood pressure also showed better attenuation with esmolol. In the present study, SBP increased from 118.27 mmHg at baseline to 131.00 mmHg at L+1 minute in Group E, whereas it increased from 118.93 mmHg to 137.13 mmHg in Group L. Similarly, DBP increased from 76.13 mmHg to 85.67 mmHg in Group E and from 76.07 mmHg to 91.27 mmHg in Group L. The difference between groups was highly significant at L+1 and L+3 minutes and remained significant at L+5 minutes. Rama Rao et al,^[11] also found that systolic and diastolic pressures increased more in the lignocaine group, while esmolol attenuated these changes more effectively. Goyal et al,^[13] reported significantly lower SBP and DBP with esmolol compared with lignocaine at 1, 3, 5 and 10 minutes after intubation. Sharma et al,^[14] also observed that lignocaine had the least effect on haemodynamic

stability compared with esmolol and diltiazem. Thus, the present blood pressure findings are consistent with published data showing stronger pressor-response control with esmolol than lignocaine.

Mean arterial pressure followed the same trend. In the present study, baseline MAP was almost identical in both groups. At L+1 minute, MAP was 100.97 mmHg in Group E and 106.56 mmHg in Group L, while at L+3 minutes it was 95.09 mmHg and 100.67 mmHg, respectively. The difference was highly significant at L+1 and L+3 minutes and significant at L+5 minutes, after which both groups became comparable. Singh et al,^[12] reported that MAP increased by 10.20% at 1 minute in the esmolol group compared with 15.89% in the lidocaine group. Goyal et al. also found significantly better MAP control with esmolol than lignocaine across the early post-intubation period. Shah et al,^[13] reported that mean arterial pressure increased significantly in the lignocaine group after intubation, while values in the esmolol group remained closer to baseline. These findings are in agreement with the present study and show that esmolol provides more consistent control of overall arterial pressure response.

Rate pressure product was lower in Group E than Group L during the early post-intubation period. In the present study, RPP increased from 9066.67 at baseline to 11614.80 at L+1 minute in Group E, while it increased from 9128.53 to 12750.80 in Group L. At L+3 minutes, RPP was 10402.67 in Group E and 11403.33 in Group L. The difference was highly significant at L+1 and L+3 minutes and remained significant at L+5 minutes. Singh et al,^[12] reported that RPP increased by only 11.68% at 1 minute in the esmolol group compared with 40.86% in the lidocaine group. In their study, RPP at 1 minute was 12681 in the esmolol group and 15459 in the lidocaine group. The present study showed the same direction of effect, although the absolute values were lower. This is clinically meaningful because RPP reflects myocardial oxygen demand; therefore, the lower RPP in Group E indicates better control of cardiac workload during the most stimulating phase of intubation.

Oxygen saturation remained stable in both groups throughout the observation period, and no clinically significant desaturation was observed. No abnormal ECG changes, hypotension, bradycardia, bronchospasm, allergic reaction or pruritus were recorded in either group. This finding indicates that both esmolol 1.5 mg/kg and lignocaine 1.5 mg/kg were safe in the studied ASA I and II patients. In comparison, Goyal et al,^[13] reported fewer adverse effects in the esmolol group than in the lignocaine group, with hypotension and bradycardia being less frequent with esmolol. Sharma et al,^[14] reported mild bradycardia in 10% of patients receiving esmolol, while Singh et al,^[12] reported that both esmolol and lidocaine were effective without deleterious effects. The absence of adverse events in the present study may be related to careful patient selection, exclusion

of high-risk patients and use of a controlled anaesthetic protocol.

Overall, the present findings show that both drugs attenuated the haemodynamic response to laryngoscopy and intubation, but esmolol was superior to lignocaine in controlling the early rise in heart rate, blood pressure, MAP and RPP. The effect was most evident at L+1 and L+3 minutes and remained significant up to L+5 minutes. By L+7 and L+10 minutes, the haemodynamic parameters became comparable between both groups. The results are consistent with previous studies, who also reported better attenuation of intubation-related haemodynamic response with esmolol compared with lignocaine.^[11-14] Therefore, intravenous esmolol 1.5 mg/kg appears to be a more effective option than intravenous lignocaine 1.5 mg/kg for short-duration control of haemodynamic stress response during laryngoscopy and endotracheal intubation in ASA I and II adult patients.

CONCLUSION

Intravenous esmolol 1.5 mg/kg was more effective than intravenous lignocaine 1.5 mg/kg in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation in adult ASA I and II patients undergoing general anaesthesia. Esmolol provided better control of heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and rate pressure product, particularly during the first 5 minutes after intubation, with the most marked difference observed at 1 and 3 minutes. Oxygen saturation remained stable in both groups, and no significant adverse effects were observed. Thus, esmolol may be considered a useful and safe option for short-duration haemodynamic control during laryngoscopy and endotracheal intubation.

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