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Original Article

Anaesthesia Section

Comparison of Intravenous Esmolol and Intravenous Lidocaine for Attenuation of Haemodynamic Responses to Laryngoscopy and Tracheal Intubation: A Double-blinded Randomised Controlled Study

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ABSTRACT

Introduction: Laryngoscopy and tracheal intubation are potent noxious stimuli that trigger significant haemodynamic surges through sympathetic activation and catecholamine release, leading to transient spikes in Heart Rate (HR) and Blood Pressure (BP). These changes may pose serious risks to patients with underlying cardiovascular conditions. Pharmacological attenuation of this response is a recognised goal of modern anaesthetic practice.

Aim: To compare the efficacy of intravenous esmolol (1.5 mg/kg) and intravenous lidocaine (1.5 mg/kg) in attenuating the haemodynamic response to laryngoscopy and tracheal intubation in American Society of Anaesthesiologists (ASA) I and II patients undergoing elective surgery under general anaesthesia.

Materials and Methods: This randomised, double-blind, controlled study included 60 ASA I and II patients aged 18-55 years, divided equally into two groups (n=30 each). Group E received i.v. esmolol 1.5 mg/kg, and Group L (control) received i.v. lidocaine 1.5 mg/kg, both administered three minutes before intubation. Haemodynamic parameters, HR, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Rate-Pressure Product (RPP), were recorded at nine time points from baseline through 10 minutes postintubation. Continuous variables were expressed as mean±Standard Deviation (SD) and compared using Student's unpaired t-test. A p-value <0.05 was considered statistically significant.

Results: There were no significant differences in demographic variables between the groups. The mean age was 30.20±9.16 years in Group E and 33.10±11.18 years in Group L (p-value=0.277). HR at one minute postintubation was significantly lower in the esmolol group (71.80±12.85 bpm) compared to the lidocaine group (95.60±15.08 bpm; p-value <0.0001), with this difference persisting up to 10 minutes. SBP at intubation was significantly reduced in the esmolol group (127.26±13.16 mmHg vs 138.26±8.38 mmHg; p-value <0.0001), with sustained lower values across subsequent time intervals. DBP and MAP were also significantly lower in the esmolol group following intubation. The RPP was markedly lower in the esmolol group at intubation (9294.27±1717.70 vs 13340.53±2365.12; p-value <0.0001), indicating superior attenuation of the sympathetic response and reduced myocardial oxygen demand.

Conclusion: While both agents help mitigate the stress response to intubation, intravenous esmolol provided superior control of HR, BP, and RPP, thereby ensuring better cardiovascular stability during the peri-intubation period. Hence, esmolol may be considered a more reliable agent for blunting intubation-induced sympathetic stress response.

Keywords: Adrenergic ß-1 receptor antagonists, Anaesthesia, Intubation, Pressor response, Tracheal

INTRODUCTION

Skilled airway management is a core competency for every anaesthesiologist, with laryngoscopy and Endotracheal Intubation (ETI) being crucial proficiencies acquired during training, yet it constitutes one of the most potent nociceptive stimuli encountered in the perioperative period. Reid LC and Brace DE first described the cardiovascular consequences of upper airway instrumentation as early as 1940, documenting a reflex rise in HR and arterial BP attributable to sympathetic nervous system activation [1]. Subsequent work confirmed that the underlying mechanism involves afferent stimulation of the glossopharyngeal and vagus nerves during laryngoscope blade insertion and tracheal tube passage, triggering a surge in circulating catecholamines, chiefly epinephrine and norepinephrine [2]. The resultant tachycardia and hypertension, exacerbate ephemeral but typically cresting within 30-45 seconds and lasting less than 10 minutes [2,3]. While transient, it is generally well tolerated and inconsequential in healthy individuals; these responses may be perilous in patients with cardiovascular co-morbidities. Therefore, mitigating these changes remains a desired target in anaesthetic practice [4].

Over the decades, a wide range of pharmacological interventions have been explored to mitigate this sympathetic surge. Opioids such as fentanyl and alfentanil, administered before induction, attenuate the response through central suppression of sympathetic outflow but carry the risk of respiratory depression and haemodynamic overshoot [5,6]. Topical and intravenous lidocaine have been in widespread use since the 1970s, with the weight of evidence supporting intravenous administration at 1.5 mg/kg given three minutes before laryngoscopy; its proposed mechanisms include suppression of laryngeal reflexes, membrane stabilisation in upper airway afferents,

and a modest central analgesic effect [7,8]. Beta-adrenergic blocking agents, calcium channel antagonists, vasodilators, and α-2 agonists such as clonidine and dexmedetomidine have each been evaluated with variable results [9,10].

Among beta-blockers, esmolol, a cardioselective, ultra-short-acting ß-1 receptor antagonist with a plasma half-life of approximately nine minutes, has attracted particular interest because its brevity of action and absence of significant interactions with common anaesthetic agents make it well suited to blunting the transient haemodynamic response to intubation without producing prolonged cardiovascular depression [11]. Lidocaine's suitability for attenuating this response arises from its analgesic, antiarrhythmic, and laryngeal reflex-suppressing properties, combined with its rapid onset and short duration of action [7].

Several investigators have compared esmolol and lidocaine head-to-head. Kindler CH et al., in a double-blind controlled trial, found that esmolol 1-2 mg/kg attenuated the HR response to intubation more effectively than lidocaine alone, which produced a statistically non significant change in HR [12]. Singh M et al., reported that esmolol achieved a significantly lower HR than lidocaine at one minute postintubation, with a difference of approximately 16 beats per minute, and also provided better control of MAP at three and five minutes [13]. Singh S et al., in a Ghanaian population, similarly found esmolol superior in controlling SBP and MAP [14]. Gupta A et al., found that esmolol (1.5 mg/kg) as a bolus attenuates the response more effectively, without any deleterious effects, when compared with Lidocaine (1.5mg/kg) [15]. Feng CK et al., and Gupta S and Tank P comparing esmolol against fentanyl and lidocaine, consistently demonstrated esmolol's superiority in rate control [3,5].

Despite this body of evidence, several important gaps remain. First, the majority of existing comparative trials were conducted in Western or East Asian populations; data from Indian tertiary care centres are limited and insufficiently powered [13-16]. Second, the RPP, a validated non invasive surrogate of myocardial oxygen consumption and a more clinically meaningful indicator of cardiac stress, has rarely been assessed as a primary safety outcome in comparative trials of esmolol versus lidocaine [16]. Third, prior studies have used heterogeneous anaesthetic induction regimens and varying doses, making firm conclusions difficult [3,12,13]. Fourth, the haemodynamic trajectory beyond the immediate postintubation period (at 3, 5, 7, and 10 minutes) has not been consistently reported. Fifth, published studies have rarely standardised laryngoscopy time or excluded cases with prolonged laryngoscopy as a confounding variable [13,14,16].

The present study was designed to address these gaps in a randomised, double-blind, parallel-group setting at a tertiary care teaching hospital in western India. The aim of study was to compare intravenous esmolol (1.5 mg/kg) and intravenous lidocaine (1.5 mg/kg) for attenuation of haemodynamic responses to laryngoscopy and ETI in a prospective randomised double-blind controlled study in a tertiary hospital setting in western India. The primary objective was to compare the incidence of tachycardia during laryngoscopy and tracheal intubation between intravenous esmolol and intravenous lidocaine groups. The secondary objectives were to compare changes in HR, SBP, DBP, MAP, RPP, and the incidence of hypertension, hypotension, bradycardia, and other adverse events between the two groups.

MATERIALS AND METHODS

This randomised, double-blind, controlled study was conducted in the Department of Anaesthesiology at Narendra Modi Medical College and LG Hospital, Ahmedabad, Gujarat, India, over a period of 25 months (December 2023 to December 2025). The study was approved by the Institutional Review Board (IEC Reference No.: NaMoMC/IRB/2023/Dissertation/74, and written informed consent was obtained from all participants prior to enrolment.

Sample size: The sample size of a study with reference to the study by Singh M et al., where the mean HR after one minute postintubation was 107±5.1 in the lidocaine group and 91.7±9.7 in the esmolol group, with a p-value of <0.0001 and sample size of 25 in each group, using the formula [13]:

Step 1: Mean HR 1 min postintubation

Group L: 107±5.1, **Group E:** 91.7±9.7

Mean difference (d) =107.7 - 91.7 = 16 bpm

Step 2: to calculate the pooled SD

$$SD = \sqrt{\frac{(5.1)^2 + (9.7)^2}{2}}$$

$$= \sqrt{\frac{26.01 + 94.09}{2}} = \sqrt{60.05} \approx 7.75$$

Pooled SD ≈ 7.75

Step 3: Sample size formula

$$n = \frac{2(Z_{\alpha/2} + Z_{\beta})^2 \sigma^2}{d^2}$$

- $\alpha = 0.05 \rightarrow 1.96$
- Power 80% $\rightarrow$ 0.84

$(1.96+0.84)^2 = 2.8^2 = 7.84$

Step 4:

$$n = \frac{2 \times 7.84 \times (7.75)^2}{(16)^2}$$

$$= \frac{2 \times 7.84 \times 60.06}{256}$$

$$= \frac{941.7}{256} \approx 3.67$$

≈ 4 patients per group

Since this result was very small, the difference was large (16 bpm), and the variability was low (SD ~7.7). So, statistically, very few patients were needed.

For any proper clinical study, the minimum calculated is four per group, which is too small and not acceptable. So, based on HR at one minute after intubation, with a mean difference of 16 beats per minute and a pooled SD of 7.75, the calculated sample size was approximately four patients per group at 95% confidence interval and 80% power. However, to improve the reliability of results, increase statistical power, and account for possible dropouts, a total of 30 patients were included in each group.

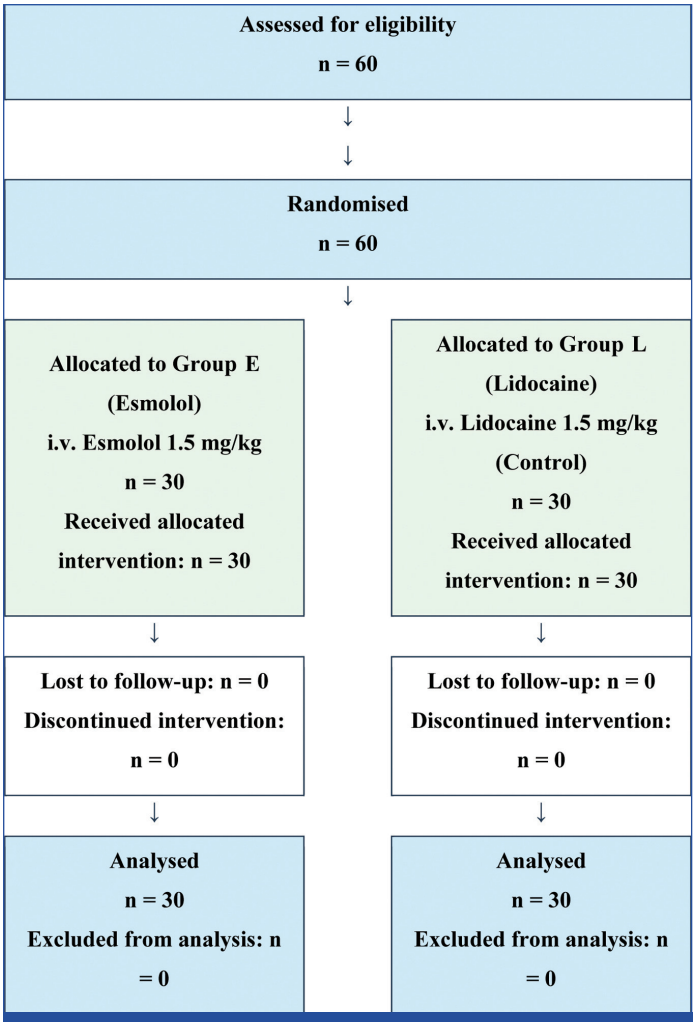
Inclusion criteria: Adult patients aged 18-55 years, of either sex, belonging to ASA physical status I or II, with Mallampati Grade I airway, who were normotensive and scheduled for elective surgery under general anaesthesia, were included in the study.

Exclusion criteria: Patients with a known allergy to the study drugs, anticipated difficult intubation, cardiovascular or severe respiratory disease, those on beta-blockers, calcium channel blockers, or sympatholytic drugs, with a history of seizures, or in whom laryngoscopy/intubation was prolonged (>30 seconds) were excluded from the study.

Study Procedure

Both major and minor elective surgical procedures under general anaesthesia were included, spanning gynaecology, ENT, and general surgery. A total of 60 patients were enrolled, and all 60 completed the study (no dropouts). The CONSORT flow diagram is provided as [Table/Fig-1].

Randomisation was performed using a computer-generated random number table by a statistician not involved in the study. Sequentially numbered, sealed, opaque envelopes were prepared in advance. A dedicated research coordinator enrolled participants and assigned them to interventions by opening envelopes in sequence. Patients



[Table/Fig-1]: CONSORT flow diagram.

were randomly allocated into two groups of 30 each: Group E received intravenous esmolol 1.5 mg/kg [15], and Group L (control) received intravenous lidocaine 1.5 mg/kg, both prepared in identical 10 mL syringes and administered three minutes before intubation by Anaesthesiologist 1 (administering anaesthesiologist, aware of group allocation). All haemodynamic observations were recorded by Anaesthesiologist 2, who was blinded to group allocation. Patients were also blinded to their group assignment, constituting a double-blind design. To minimise bias, an identical syringe appearance was ensured, Mallampati grade was standardised to Grade I in all patients, laryngoscopy was restricted to ≤30 seconds, and all induction agents and premedication were standardised.

Group L (lidocaine, 1.5 mg/kg i.v.) was designated as the control group, reflecting its established use as a standard pharmacological intervention for blunting the pressor response to intubation. Group E (esmolol, 1.5 mg/kg i.v.) constituted the study/intervention group.

All patients underwent thorough preanaesthetic evaluation. They were kept nil per oral overnight and received tablet alprazolam 0.5 mg orally at bedtime. Injection glycopyrrolate 0.004 mg/kg [16] was administered intramuscularly 30 minutes before induction. On arrival in the operating theatre, intravenous access was secured with an 18-G cannula, and Ringer's lactate infusion commenced. Standard monitoring was instituted, and the following baseline parameters were recorded (BPM time point): HR (bpm), SBP (mmHg), DBP (mmHg), MAP (mmHg), SpO_2, and respiratory rate (breaths/min). Patients were preoxygenated with 100% oxygen for 3 minutes. Anaesthesia was induced with fentanyl 1 µg/kg and propofol 2 mg/kg. After loss of eyelash reflex, atracurium 0.5 mg/kg was administered to facilitate intubation. Laryngoscopy and intubation were performed three minutes after study drug administration, using an appropriately sized Macintosh blade and completed within ≤30 seconds. Anaesthesia was maintained with oxygen 50%, nitrous oxide 50%, and sevoflurane. Neuromuscular blockade was maintained with intermittent atracurium. Surgical stimulation was avoided for the first 10 minutes following intubation. At the end of surgery, neuromuscular blockade was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 8 µg/kg i.v.; tracheal extubation was performed after adequate neuromuscular recovery, and patients were transferred to PACU.

Haemodynamic parameters (HR, SBP, DBP, MAP, and RPP) were recorded at nine time intervals: before premedication (BPM baseline), after premedication (APM), after Esmolol/Lidocaine (AE/L), after intubation (AI), and at 1 (ETI1), 3 (ETI3), 5 (ETI5), 7 (ETI7), and 10 (ETI10) minutes postintubation. Incidence of tachycardia (HR ≥20% above baseline) during and after laryngoscopy and tracheal intubation was taken as primary outcome and Changes in HR (bpm), SBP (mmHg), DBP (mmHg), MAP (mmHg), and RPP recorded at nine predefined time points; incidence of hypotension (SBP -20%of baseline in mmHg), hypertension (SBP ≥ 20% of baseline in mmHg), bradycardia (HR ≤20%of baseline in bpm), bronchospasm, and other adverse drug reactions.

STATISTICAL ANALYSIS

Data were expressed as mean±SD. Categorical variables were analysed using the Chi-square test. Continuous variables were compared between groups using the independent Student's unpaired t-test. Within-group comparisons with baseline were performed using the paired t-test. One-way Analysis of Variance (ANOVA) was applied for multi-time-point comparisons, with Bonferroni's post-hoc correction applied when a significant difference was found. Statistical significance was set at p-value ≤0.05 (95% confidence interval). All analyses were performed using Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Both groups were well-matched at baseline. There was no statistically significant difference in demographic variables between the two groups (p-value >0.05 for all parameters), confirming comparability [Table/Fig-2].

Variables	Group E (Esmolol) Mean±SD (n=30)	Group L (Lidocaine) Mean±SD (n=30)	p-value
Age (years)	30.20±9.16	33.10±11.18	0.277
Weight (kg)	56.50±8.99	61.53±11.45	0.063
Height (cm)	154.86±9.01	159.00±8.51	0.073
BMI (kg/m²)	23.79±4.65	24.50±5.14	0.572
Laryngoscopy time (sec)	8.83±2.47	8.63±1.75	0.719
Male : Female	6:24 (20:80%)	5:25 (16.67:83.33%)	0.732
ASA Grade I : II	28:2 (93.33:6.67%)	28:2 (93.33:6.67%)	1.000
SpO_2 (%)	99.23±0.57	99.10±0.61	0.388
Respiratory Rate (breaths/min)	16.40±1.35	16.73±1.41	0.344

[Table/Fig-2]: Demographic variables.
p-value <0.05 = Significant; p-value >0.05 = Not Significant

Surgery distribution between the two groups is depicted in [Table/Fig-3]. Gynaecological procedures were most common in both groups; the distribution was comparable between groups, minimising procedural confounding.

The HR variations are depicted in [Table/Fig-4]. Esmolol was highly effective in controlling tachycardia associated with intubation: a statistically significant and sustained reduction in HR was observed in Group E compared to Group L from the post-drug period onwards, persisting through all postintubation time points (p-value <0.0001). Lidocaine provided only partial and transient attenuation of tachycardia.

Procedure	Group E (n=30)	Group L (n=30)
Gynaecology (Lap TL/ MTP+ Lap TL/ D&E/ Diagnostic/Operative scopy/TLH)	16 (53.33%)	11 (36.67%)
ENT (FESS//DHL/Mastoid/Septoplasty/ Tympanoplasty)	7 (23.33%)	9 (30.00%)
General Surgery (Lap Appendicectomy/ Lap Cholecystectomy/TR/Neck I&D/Rectal Biopsy)	4 (13.33%)	4 (13.33%)
Thyroid/Zygomatic Plating/Other	3 (10.00%)	6 (20.00%)
Total	30 (100%)	30 (100%)

[Table/Fig-3]: Surgery distribution.
Lap TL: Laparoscopic Tubal Ligation; MTP: Medical termination of pregnancy; D&E : Dilatation and evacuation; DHL: Diagnostic hystero-laparoscopy; TLH: Total laparoscopic hysterectomy; TR: Tonsillar resection

Time point	Group E Mean±SD	Group L Mean±SD	p-value
BPM (Baseline)	90.33±15.60	94.00±17.24	0.391
APM	84.33±11.70	93.07±17.00	0.024
AE/L	75.60±11.95	94.67±17.02	<0.0001
AI	74.13±12.48	96.60±16.14	<0.0001
ETI 1 min	71.80±12.85	95.60±15.08	<0.0001
ETI 3 mins	71.00±12.44	95.80±15.32	<0.0001
ETI 5 mins	69.40±12.21	96.60±14.89	<0.0001
ETI 7 mins	68.67±11.48	98.40±16.06	<0.0001
ETI 10 mins	67.67±11.28	100.53±15.29	<0.0001

[Table/Fig-4]: Heart Rate (HR) Variations (bpm).
p-value <0.05 = Significant; p-value >0.05 = Not Significant

The SBP variations are shown in [Table/Fig-5]. Esmolol provided superior attenuation of the SBP surge at intubation and all postintubation intervals compared to lidocaine (p-value <0.0001 at most time points).

Time point	Group E Mean±SD	Group L Mean±SD	p-value
BPM (Baseline)	124.40±11.73	125.86±7.75	0.570
APM	119.53±11.33	122.00±7.68	0.238
AE/L	120.80±12.18	123.13±8.29	0.390
AI	127.26±13.16	138.26±8.38	<0.0001
ETI 1 min	127.26±14.29	134.46±7.49	0.018
ETI 3 mins	123.46±13.28	131.46±9.30	0.009
ETI 5 mins	117.33±12.51	128.80±10.70	<0.0001
ETI 7 mins	112.06±11.54	127.06±8.73	<0.0001
ETI 10 mins	111.26±11.87	126.73±8.99	<0.0001

[Table/Fig-5]: Systolic Blood Pressure (SBP) variations (mmHg).
p-value <0.05 = Significant; p-value >0.05 = Not Significant

The DBP was better controlled in the esmolol group, with significant differences emerging from ETI3 onwards and persisting through ETI10 (p-value ≤0.007), confirming more comprehensive cardiovascular protection with esmolol [Table/Fig-6].

Time point	Group E Mean±SD	Group L Mean±SD	p-value
BPM (Baseline)	80.66±8.10	84.00±5.35	0.065
APM	78.13±7.44	80.46±4.47	0.147
AE/L	79.00±8.69	81.90±4.67	0.128
AI	85.13±11.98	96.13±6.06	<0.0001
ETI 1 min	87.66±12.12	92.00±6.24	0.074
ETI 3 mins	85.06±10.64	91.33±6.22	0.007
ETI 5 mins	82.46±9.13	85.53±6.92	<0.001
ETI 7 mins	79.06±8.21	86.60±5.82	<0.0001
ETI 10 mins	77.53±9.19	85.80±5.95	<0.0001

[Table/Fig-6]: Diastolic Blood Pressure (DBP) Variations (mmHg).
p-value <0.05 = Significant; p-value >0.05 = Not Significant

MAP was significantly lower in the esmolol group compared to the lidocaine group at intubation and at all subsequent postintubation time points, indicating superior haemodynamic control with esmolol [Table/Fig 7].

Time point	Group E Mean±SD	Group L Mean±SD	Independent t-test p-value
BPM	95.66±7.16	98.46±5.37	0.092
APM	91.60±6.65	94.86±4.97	0.035
AE/L	93.93±7.13	95.86±4.95	0.228
AI	100.60±10.14	110.53±6	<0.0001
ETI 1 min	101.13±10.79	106.46±6.07	0.022
ETI 3 mins	97.93±9.51	105.73±5.81	<0.0001
ETI 5 mins	94.40±8.26	102.60±7.02	<0.0001
ETI 7 mins	90±6.78	100.2±5.56	<0.0001
ETI 10 mins	88.46±6.31	99.66±6.03	<0.0001

[Table/Fig-7]: Mean Arterial Pressure (MAP) variations (mmHg).
p-value <0.05 = Significant; p-value >0.05 = Not Significant

The RPP (RPP=HR×SBP), a surrogate marker of myocardial oxygen demand, was significantly lower in the esmolol group at all postintubation intervals (p-value <0.0001), indicating superior reduction in myocardial oxygen demand and cardioprotective benefit [Table/Fig-8]. In both groups, RPP at one minute postintubation remained below 20,000, the threshold associated with myocardial ischaemia risk.

Time point	Group E Mean±SD	Group L Mean±SD	p-value
BPM (Baseline)	11046.60±2072.04	11828.13±2084.23	0.150
APM	9970.60±1563.75	11337.33±1994.36	0.005
AE/L	8897.60±1629.54	11611.07±2080.13	<0.0001
AI	9294.27±1717.70	13340.53±2365.12	<0.0001
ETI 1 min	8993.33±1873.57	12916.80±2181.51	<0.0001
ETI 3 mins	8647.33±1769.60	12632.80±2126.33	<0.0001
ETI 5 mins	8023.73±1661.75	12500.40±2159.89	<0.0001
ETI 7 mins	7648.40±1526.51	12546.67±2206.80	<0.0001
ETI 10 mins	7454.53±1496.65	12805.20±2220.41	<0.0001

[Table/Fig-8]: Rate-Pressure Product (RPP) variations.
p-value <0.05 = Significant; p-value >0.05 = Not Significant

No clinically significant adverse events were recorded in either group during the study period. No patient developed hypotension (SBP <90 mmHg), severe bradycardia requiring intervention, bronchospasm, or any other serious drug-related adverse event.

DISCUSSION

In the present study, esmolol produced a statistically significant and sustained reduction in HR at all postintubation time points compared to lidocaine (p-value <0.0001 at all intervals from AE/L to ETI10). This is attributable to its selective ß-1 adrenergic blockade, which directly antagonises the catecholamine-mediated tachycardia triggered by laryngoscopy and intubation. Studies of Reid LC and Brace DE and Shribman AJ et al., established that HR and BP can rise by 20-40% during intubation, peaking within the first minute [1,2]. The present study findings align with those of Kindler CH et al., who demonstrated that esmolol 1-2 mg/kg effectively attenuates HR responses, while lidocaine alone showed a statistically non significant increase in HR [12]. Similarly, Singh M et al., reported a difference of approximately 16 beats/min in favour of esmolol at one minutes postintubation, with baseline-comparable HR levels in the esmolol group through all intervals, and these findings were closely mirrored in the present study [13].

SBP, DBP, and MAP were all significantly lower in Group E compared to Group L at intubation and across most postintubation time points (p-value <0.0001 to p-value=0.009). Lidocaine

provided only partial and transient BP attenuation, particularly in the early postintubation period, consistent with its predominantly peripheral mechanism of reducing airway reflexes rather than direct cardiovascular blockade. Singh S et al., corroborated these findings, noting better control of SBP, DBP, and MAP with esmolol [14]. Similarly, Rao DS and Osman AOA reported superior haemodynamic control with esmolol across all BP parameters [16]. The haemodynamic response is known to be directly proportional to the force exerted to expose the glottis, with direct laryngoscopy increasing HR and BP by 20-27% and 30-50%, respectively [17,18]. By standardising laryngoscopy time (Group E: 8.83±2.47 sec; Group L: 8.63±1.75 sec; p-value=0.719), the present study observed that differences were attributable to the study drugs rather than procedural variation.

The RPP, a validated non invasive surrogate of myocardial oxygen demand, was significantly lower in the esmolol group at all postintubation intervals (p-value <0.0001). In both groups, RPP at one minute postintubation remained below 20,000, the threshold above which the risk of angina and myocardial ischaemia increases considerably [19]. This finding confirms the cardioprotective effect of both drugs, with esmolol offering a more robust degree of protection. Lower RPP values correlate with reduced risk of perioperative myocardial ischaemia, a significant concern in cardiovascularly vulnerable patients. Our failure to detect a significant lidocaine effect on RPP was consistent with Stoelting RK whose studies, conducted in patients with heart disease, reported a modest favourable response with lidocaine but noted it to be less reliable than direct sympatholytic agents [7].

The present study results are further supported by Singh S et al., who found esmolol superior in controlling MAP compared to lidocaine in a Ghanaian population [14]. Sintetos AL et al., reported that haemodynamic variables in the esmolol group returned to near-baseline by three minutes and fell below baseline by five minutes, reflecting esmolol's ultra-short half-life (~9 minutes) and rapid redistribution [11]. This pattern was reproduced in the present study. In contrast, the current study did not corroborate the findings of Rao DS and Osman AOA, where minimal HR variability was noted between groups, a discrepancy possibly attributable to patient population differences, variation in concurrent anaesthetic regimens, or dosing protocols [16]. Esmolol's cardioselectivity, short duration of action, and absence of major interactions with standard anaesthetic agents [11] make it particularly suitable for attenuating the transient haemodynamic response to intubation.

Clinically, maintaining haemodynamic stability during laryngoscopy and intubation is critical, particularly in patients with cardiovascular co-morbidities, hypertension, or cerebrovascular disease. This study reinforces the clinical utility of esmolol as a preferred agent for attenuating pressor and tachycardic responses associated with airway manipulation. Pre-emptive administration of esmolol prior to intubation should be considered, especially in high cardiovascular risk patients, to reduce the risk of perioperative myocardial ischaemia, arrhythmias, and hypertensive crises and to enhance patient safety.

Future studies should include higher-risk patient populations such as ASA III-IV patients and the elderly, measure plasma catecholamine levels as a direct biochemical surrogate of sympathetic attenuation, employ larger multicentre designs, assess long-term perioperative outcomes, including postoperative myocardial injury, and explore combination regimens (e.g., esmolol plus an opioid or esmolol plus lidocaine) that may offer synergistic haemodynamic benefits with patient-centred outcome measures.

Limitation(s)

The present study had several limitations. First, the population was restricted to ASA I and II patients aged 18-55 years, limiting generalisability to higher-risk patients with overt cardiovascular disease, the elderly, or those with autonomic dysfunction. Second, plasma catecholamine levels were not measured, which would have provided direct biochemical corroboration of the degree of sympathetic attenuation. Third, the sample size may have been underpowered to detect modest differences in some secondary endpoints at early time intervals. Fourth, long-term perioperative outcomes such as postoperative myocardial injury or arrhythmia incidence were not assessed. Fifth, this was a single-centre study, which may introduce centre-specific bias.

CONCLUSION(S)

Both intravenous esmolol and lidocaine are effective to varying degrees in attenuating the haemodynamic response to laryngoscopy and ETI in ASA I and II patients undergoing elective surgery under general anaesthesia. However, esmolol demonstrated statistically superior and more sustained control of HR, SBP and DBP, MAP, and RPP at all postintubation time intervals compared to lidocaine. The significantly lower RPP in the esmolol group across all time points indicates superior reduction in myocardial oxygen demand and a clinically meaningful cardioprotective advantage. Lidocaine, while providing partial and transient haemodynamic attenuation, was less reliable in controlling tachycardia and did not match the degree of BP control achieved by esmolol. Based on these findings, intravenous esmolol at 1.5 mg/kg administered three minutes before intubation may be considered the preferred pharmacological agent for blunting sympatho-adrenal responses during airway management.

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