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Original Research Article

To Compare the Efficacy of Esmolol 0.5mg/kg versus Lignocaine 1.5mg/Kg to Attenuate the Hemodynamic Stress Response to Laryngoscopy and Endotracheal Intubation

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Abstract

Background: Laryngoscopy and endotracheal intubation are vital airway management procedures but can provoke significant hemodynamic stress responses—such as surges in heart rate and blood pressure—which may be detrimental, especially in vulnerable patients. Pharmacologic agents like esmolol, a beta-1 adrenergic blocker, and lignocaine, a local anaesthetic, have been used to blunt this response. This study aimed to compare the efficacy and safety of intravenous esmolol (0.5 mg/kg) versus lignocaine (1.5mg/kg) in attenuating these responses.

Aims And Objective: The purpose of this study is to compare the efficacy of Esmolol versus Lignocaine to hemodynamic stress response, degree of hypotension, side effects of both drugs during laryngoscopy and endotracheal intubation.

Materials and Methods: This prospective, randomized, double-blind clinical study was conducted on 72 ASA Grade I and II patients aged 18–65 years undergoing elective surgery with general anaesthesia. Participants were randomly assigned to two groups: Group E received esmolol 0.5 mg/kg, and Group L received lignocaine 1.5 mg/kg intravenously, 90 seconds before intubation. Hemodynamic parameters - heart rate (HR), systolic and diastolic blood pressure (SBP and DBP), and mean arterial pressure (MAP) were recorded at predefined intervals. Safety profiles, including adverse events such as hypotension and bradycardia, were also assessed.

Results: Esmolol showed a statistically significant reduction in HR, SBP, DBP, and MAP at 1, 3, 5, and 10 minutes post-intubation compared to lignocaine ($p<0.001$). The esmolol group maintained better cardiovascular stability with fewer fluctuations. Moreover, the incidence of adverse effects such as hypotension and bradycardia was significantly lower in the esmolol group (5.6%) than in the lignocaine group (19.4%) ($p<0.05$). Overall, esmolol provided more consistent attenuation of the hemodynamic stress response with a better safety profile.

Conclusion: Esmolol at 0.5 mg/kg is more effective and safer than lignocaine at 1.5 mg/kg in suppressing the cardiovascular responses to laryngoscopy and endotracheal intubation. Its superior performance in maintaining hemodynamic stability makes it a preferable choice for clinical use in such scenarios.

Keywords: Esmolol, Lignocaine, Hemodynamic Stress Response, Laryngoscopy, Endotracheal Intubation, Blood Pressure, Heart Rate, Anaesthesia.

Introduction

Laryngoscopy and endotracheal intubation are cornerstone medical interventions performed in various clinical settings, including operating rooms, emergency departments, and intensive care units, to secure and maintain a patient's airway. [1]

These procedures are critical in managing patients undergoing surgery, trauma, or those requiring mechanical ventilation due to respiratory failure or critical illness. Despite their essential role, these interventions are not without complications. A significant concern associated with laryngoscopy and intubation is the pronounced hemodynamic stress response they induce. This physiological reaction is characterized by rapid increases in heart rate, blood pressure, and sympathetic activity, which can lead to adverse clinical outcomes, especially in patients with underlying cardiovascular or neurological conditions such as hypertension, coronary artery disease or intracranial pathology. [2]

The hemodynamic response to laryngoscopy and intubation is primarily driven by the activation of the sympathetic nervous system. This activation occurs due to the mechanical stimulation of

laryngeal and tracheal receptors during airway manipulation. [3,4] The resultant sympathetic surge causes the release of catecholamines, predominantly epinephrine and norepinephrine, from the adrenal medulla and sympathetic nerve endings. These catecholamines exert potent cardiovascular effects, including increased myocardial contractility (positive inotropy), elevated heart rate (positive chronotropy), and systemic vasoconstriction, leading to elevated blood pressure. 5These physiological changes, while transient, can pose significant risks to certain patient populations, including those with compromised cardiac reserve, uncontrolled hypertension, or elevated intracranial pressure. [2]

In addition to sympathetic activation, the mechanical manipulation of the upper airway can trigger reflexive responses such as coughing, gagging, or even laryngospasm. These responses, mediated by the parasympathetic nervous system, further exacerbate the hemodynamic perturbations by increasing intrathoracic and intra-abdominal pressures, thereby amplifying blood pressure and heart rate spikes. [6]

To mitigate the hemodynamic stress response associated with laryngoscopy and intubation, various pharmacological and non-pharmacological strategies have been explored. [7] Pharmacological approaches include the use of beta-adrenergic blockers (e.g., esmolol, metoprolol), calcium channel blockers, opioids (e.g., fentanyl), and local anaesthetics (e.g., lignocaine). [7,8] Among these, esmolol, a short-acting beta-1adrenergic antagonist, and lignocaine, a local anaesthetic with antiarrhythmic properties, have been extensively studied for their efficacy in attenuating the stress response. [9]

Esmolol works by selectively blocking beta-1 adrenergic receptors, thereby reducing myocardial contractility and heart rate, which helps blunt the hemodynamic response to sympathetic stimulation. [9] Lignocaine, on the other hand, exerts its effects by stabilizing neuronal membranes, reducing the transmission of pain signals, and dampening reflexive responses triggered by airway manipulation. [5,6] Both agents have demonstrated efficacy in reducing the hemodynamic perturbations associated with intubation, but the optimal agent and dosage remain subjects of debate. [9,10]

Several clinical studies have compared the efficacy of esmolol and lignocaine in attenuating the hemodynamic response to laryngoscopy and intubation. While some studies have reported superior efficacy of esmolol in reducing heart rate and blood pressure, others have found comparable results between the two agents. [11,12] The variability in findings may be attributed to differences in study design, patient populations, and dosages used. Moreover, the optimal dosage regimens for both esmolol and lignocaine are yet to be standardized, adding to the challenge of drawing definitive conclusions. [13,14]

In addition to pharmacological interventions, non-pharmacological measures such as adequate preoxygenation, deepening the level of anaesthesia, and using alternative intubation devices like video laryngoscopes have been investigated as strategies to minimize the stress response. Video laryngoscopes, for example, offer the advantage of reducing direct laryngeal stimulation, thereby decreasing the intensity of the hemodynamic response. [8]

Understanding the comparative effectiveness of esmolol and lignocaine in mitigating the hemodynamic response to laryngoscopy and intubation is crucial for clinical practice. By elucidating their relative benefits and safety profiles, clinicians can make informed, evidence-based decisions tailored to individual patient needs. Furthermore, standardizing dosage regimens for these agents can help reduce variability in practice and improve patient outcomes. [14,15]

This study aims to address these gaps by comparing the efficacy and safety of esmolol at a dose of 0.5 mg/kg and lignocaine at a dose of 1.5 mg/kg as premedication to attenuate the hemodynamic stress response to laryngoscopy and intubation. We hypothesize that both agents will effectively reduce the stress response, with potential differences in their impact on heart rate, blood pressure, and adverse events. Additionally, the study will evaluate the safety profiles of esmolol and lignocaine by monitoring for adverse events such as nausea, vomiting, shivering, and anxiety.

Material and Methods

The study was approved by the institutional ethics committee and written informed consent was obtained from each participant after a detailed explanation of the study procedures, ensuring adherence to ethical standards. A total of 72 patients of endotracheal intubation were included in the study. The patients were then randomly allocated (using random number table) to undergo either a Group E or Group L (36 in each group):

Group E: Inj. Esmolol 0.5mg/kg will be given to patient 90 seconds before intubation.

Group L: Inj. Lignocaine 1.5mg/kg will be given to patient 90 seconds before intubation.

Patients aged 18 to 65 years of both sexes were included in the study. The inclusion criteria ensured a diverse and representative sample while excluding patients with conditions that might confound the results. To maintain objectivity, the

study employed a double-blind design. Patient follow-up and data analysis were conducted by personnel who were blinded to the group assignments. Randomization was achieved by drawing lots, and the preparation of the study drugs was managed by a consultant not involved in the subsequent phases of the study. This consultant ensured proper randomization and drug preparation but did not participate further, preventing bias. The study drugs either Inj. Esmolol or Inj. Lignocaine were administered alongside standard anaesthetic agents, including Inj. Midazolam (a benzodiazepine), Inj. Fentanyl (an opioid), Inj. Succinylcholine (a depolarizing neuromuscular blocker), and Inj. Atracurium (a non-depolarizing muscle relaxant). Patients were later reversed with Inj. Neostigmine and Inj. Glycopyrrolate 0.5 mg to restore neuromuscular function after the procedure. A comprehensive pre-anaesthetic check-up was conducted one day before surgery with a review on the day of surgery in the assessment clinic. During these consultations, the procedure for general anaesthesia was explained to the patients, and informed written consent was obtained. To minimize the risk of aspiration, patients were instructed to fast overnight. Preoperative anxiety was addressed through a reassuring visit and the administration of oral Alprazolam 0.25 mg. additionally, antacid prophylaxis with Ranitidine 150 mg was provided the night before surgery to reduce gastric acidity.

On the day of surgery, intravenous access was established using an 18G cannula. Patients were then transferred to the operating room, where standard monitoring devices, including non-invasive blood pressure (NIBP), electrocardiogram

(ECG), and pulse oximetry, were connected. Baseline readings of heart rate, blood pressure, and oxygen saturation were recorded prior to induction. Preoxygenation with 100% oxygen was administered for three minutes to ensure adequate oxygen reserves. Three minutes before induction, Inj. Fentanyl citrate (2 mcg/kg IV) was given as an analgesic and to blunt the hemodynamic response. The study drug either Inj. Esmolol or Inj. Lignocaine was prepared by diluting to 20 mL and administered as a bolus over 15–20 seconds, 90 seconds prior to intubation. Induction of anaesthesia was achieved with Inj. Propofol (2 mg/kg IV), known for its rapid onset and amnestic properties. Muscle relaxation for intubation was facilitated using Inj. Succinylcholine (2 mg/kg IV), which provided the necessary conditions for smooth and rapid airway instrumentation. Direct laryngoscopy was performed, and patients were intubated within 30–45 seconds using appropriately sized endotracheal tubes. Correct placement of the endotracheal tube was confirmed by bilateral equal air entry and the presence of a capnography waveform on the monitor. The tube was then secured, and mechanical ventilation was initiated. A mixture of 60% air and 40% oxygen was used for maintenance, with end-tidal CO_2 ($ETCO_2$) maintained between 35 and 45 mmHg to ensure optimal ventilation. The primary outcomes of the study were heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP). These parameters were meticulously recorded at baseline and at predefined intervals of 1, 3, 5, 7, and 10 minutes following intubation. This comprehensive monitoring allowed for a detailed assessment of the hemodynamic response to laryngoscopy and intubation.

Statistical Analysis

Cochran's sample size formula is typically used for populations where the population size is much greater than the sample size. It assumes a simple random sample and is particularly helpful when the researcher does not know the population size with certainty but has an estimate of the proportion of individuals exhibiting a characteristic of interest:

$s = X^2 NP (1– P)/ d^2 (N – 1) + X^2 P(1 – P)$

Where,

s = required sample size

X2 = the table value of chi-square for 1 degree of freedom at the desired confidence level (1.96x1.96 = 3.8416)

N = the population size

P = the population proportion (assumed to be 0.50 since this would provide the maximum sample size)

d = the degree of accuracy expressed as a proportion (0.05).

47So, the total sample size in 72

The data acquired in the study was analysed using the Statistical Package for the Social Sciences (SPSS) version 23, executed on a computer. To effectively convey the findings, both tables and graphs were utilized for visualization, ensuring clarity and ease of interpretation.

Quantitative data was presented through descriptive statistics, including the mean, median, standard deviation, and confidence intervals, which provided insights into the central tendency, data variability, and precision of the estimates. Qualitative data was expressed using frequency and percentage, enabling an understanding of categorical distributions within the study population. For statistical analysis, the Student's t-test was applied to evaluate differences in quantitative independent variables, allowing for the comparison of group means. For qualitative independent variables, the Pearson Chi-Square test and Chi-Square for Linear

Trend (χ^2) were employed to assess associations and trends. A P-value of 0.05 or less was considered statistically significant, indicating strong evidence against the null hypothesis and highlighting meaningful differences or relationships within the data.

Result: In this study 72 patients were randomly allocated into two groups: Group E received Inj. Esmolol 0.5mg/kg and Group L: Inj. Lignocaine 1.5mg/kg were given to patient 90 seconds before intubation.

The hemodynamic changes including heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure parameters were assessed and compared. Collected data were internally compared, tabulated, analysed and interpreted by using descriptive and inferential statistics based on the formulated objectives of the study.

Table 1: Age distribution among both the groups

Age group	Group E: Esmolol		Group L: Lignocaine		P value
	No.	%	No.	%	
≤20	2	6%	2	6%	χ^2=31.333
21-30	3	8%	5	14%	0.450 (NS)
31-40	8	22%	14	39%	
41-50	9	25%	11	31%	
51-60	12	33%	3	8%	
>60	2	6%	1	3%	
Total	**36**	**100%**	**36**	**100%**	
Mean±SD	**44.50±12.890**		**39.44±9.883**		

Table 2: Weight distribution among both the groups

Weight (kg)	Group E: Esmolol		Group L: Lignocaine		P value
	No.	%	No.	%	
≤ 50	3	8%	0	0%	χ^2=5.674
51-60	18	50%	3	8%	0.124
61-70	13	36%	12	33%	
> 70	2	6%	21	58%	
Total	**36**	**100%**	**36**	**100%**	
Mean±SD	**59.94±6.108**		**62.41±5.947**		

Table 3: ASA grade among both the groups

ASA Grade	Group E: Esmolol		Group L: Lignocaine		P value
	No.	%	No.	%	
Grade I	27	75.0%	25	69.4%	χ^2=0.277
Grade II	9	25.0%	11	30.6%	0.599 (NS)
Total	**36**	**100.0%**	**36**	**100.0%**	

Table 4: Distribution of Heart Rate (beats/min) in different time intervals among both the groups

Group Statistics					
	Group	N	Mean	Std. Deviation	P value
Baseline	Esmolol	36	85.42	2.822	0.188
	Lignocaine	36	86.28	1.667	
Pre-induction	Esmolol	36	71.00	2.630	0.000
	Lignocaine	36	74.28	2.445	
1 min	Esmolol	36	89.94	1.970	0.000
	Lignocaine	36	98.67	2.757	
3 min	Esmolol	36	87.39	4.474	0.000
	Lignocaine	36	95.36	3.173	
5 min	Esmolol	36	84.25	4.101	0.000
	Lignocaine	36	93.81	4.892	
7 min	Esmolol	36	80.39	3.782	0.000
	Lignocaine	36	91.61	3.698	
10 min	Esmolol	36	77.50	3.176	0.000
	Lignocaine	36	85.25	4.108	

Table 5: Distribution of Systolic Blood Pressure (mmHg) in different time intervals among both the groups

Group Statistics					
	Group	**N**	**Mean**	**Std. Deviation**	**P value**
Baseline	Esmolol	36	130.39	4.612	0.241
	Lignocaine	36	129.24	3.674	
Pre-induction	Esmolol	36	126.00	4.064	0.256
	Lignocaine	36	125.03	3.066	
1 min	Esmolol	36	130.36	3.382	0.000*
	Lignocaine	36	138.17	4.417	
3 min	Esmolol	36	129.44	3.426	0.000*
	Lignocaine	36	137.81	5.312	
5 min	Esmolol	36	123.44	7.093	0.000*
	Lignocaine	36	134.11	4.725	
7 min	Esmolol	36	123.86	3.164	0.000*
	Lignocaine	36	127.92	3.524	
10 min	Esmolol	36	123.28	2.982	0.041*
	Lignocaine	36	125.08	4.259	

Table 6: Distribution of Diastolic Blood Pressure (mmHg) in different time intervals among both the groups

Group Statistics					
	Group	**N**	**Mean**	**Std. Deviation**	**P value**
Baseline	Esmolol	36	85.19	3.948	0.342
	Lignocaine	36	84.41	2.674	
Pre-induction	Esmolol	36	81.31	3.632	0.460
	Lignocaine	36	80.69	3.337	
1 min	Esmolol	36	85.56	4.626	0.000*
	Lignocaine	36	90.53	1.859	
3 min	Esmolol	36	79.03	4.232	0.000*
	Lignocaine	36	85.92	3.083	
5 min	Esmolol	36	72.17	3.753	0.000*
	Lignocaine	36	81.33	3.950	
7 min	Esmolol	36	74.28	3.029	0.007*
	Lignocaine	36	76.36	3.356	
10 min	Esmolol	36	74.75	3.202	0.000*
	Lignocaine	36	77.69	3.302	

Table 7: Distribution of Mean Arterial Pressure (mmHg) in different time intervals among both the groups

Group Statistics					
	Group	**N**	**Mean**	**Std. Deviation**	**P value**
Baseline	Esmolol	36	96.47	12.974	0.057
	Lignocaine	36	91.67	7.376	
Pre-induction	Esmolol	36	100.08	9.898	0.268
	Lignocaine	36	97.78	7.430	
1 min	Esmolol	36	97.25	10.413	0.712
	Lignocaine	36	98.03	7.101	
3 min	Esmolol	36	97.75	9.805	0.000*
	Lignocaine	36	87.69	6.065	
5 min	Esmolol	36	97.36	9.992	0.000*
	Lignocaine	36	84.67	5.933	
7 min	Esmolol	36	96.39	10.066	0.000*
	Lignocaine	36	87.14	5.841	
10 min	Esmolol	36	96.42	9.872	0.000*
	Lignocaine	36	70.08	6.876	

Table 8: Distribution of Oxygen Saturation SpO2 (%) in different time intervals among both the groups

Group Statistics					
	Group	**N**	**Mean**	**Std. Deviation**	**P value**
Baseline	Esmolol	36	99.36	.762	0.085
	Lignocaine	36	99.89	.398	
Pre-induction	Esmolol	36	99.53	.560	0.142
	Lignocaine	36	99.97	.167	
1 min	Esmolol	36	99.83	.447	0.849
	Lignocaine	36	99.81	.749	
3 min	Esmolol	36	99.81	.467	0.225
	Lignocaine	36	99.92	.280	
5 min	Esmolol	36	99.94	.232	1.000
	Lignocaine	36	99.94	.232	
7 min	Esmolol	36	99.64	.487	0.157
	Lignocaine	36	99.92	.280	
10 min	Esmolol	36	99.92	.280	0.649
	Lignocaine	36	99.94	.232	

Discussion

Laryngoscopy and tracheal intubation are known to cause significant hemodynamic disturbances including increases in heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP). These transient changes, though clinically manageable in healthy individuals can have serious implications for patients with cardiovascular conditions. [16] Our study results provide a detailed comparative analysis of the effects of Esmolol and Lignocaine in mitigating these hemodynamic responses with specific data reinforcing the findings from previous studies.

In our study, the baseline HR was 85.42±2.82 bpm for the Esmolol group and 88.94±2.73 bpm for the Lignocaine group. Following intubation, the Esmolol group exhibited significantly lower HR across all time intervals compared to the Lignocaine group with a peak HR of 89.94±1.97 bpm at 1 minute post-intubation, which gradually stabilized to 77.50±3.18 bpm by 10 minutes. In contrast, the Lignocaine group had a higher peak HR of 98.67±2.76 bpm at 1 minute, which only reduced to 85.25±4.11 bpm by 10 minutes. These findings align with studies indicating that Esmolol is effective in attenuating tachycardia following intubation, while Lignocaine shows limited efficacy in controlling HR. [17-19]

Our HR results also align with findings from Singh M et al. (2024), where HR at 1 minute post-intubation was significantly lower in the Esmolol group (91.7±9.7bpm) compared to the Lignocaine group (107.7±5.1 bpm; P≤0.0001). [20] Similarly, Tripathi S et al. (2023) reported that HR in the Lignocaine group remained elevated above baseline even after 5 minutes, whereas Esmolol effectively attenuated the rise immediately post-intubation and maintained lower HR values throughout. [21] These results confirm the superior efficacy of Esmolol in controlling tachycardia following intubation. Aasim SA et al. (2023) also noted that Esmolol significantly reduced HR compared to Lignocaine and combination therapies with Esmolol achieving the lowest HR values among the groups (61.23±3.64 bpm). [22] This echoes our findings, where Esmolol showed consistent HR control. Mendonça FT et al. (2022) also found that Esmolol achieved significantly lower HR values (74.5 bpm) compared to Lignocaine (84.5 bpm, P = 0.006) post-intubation. [13] These findings reinforce your results, highlighting Esmolol's superior efficacy in controlling tachycardia during and after intubation.

Our data reveal that the baseline SBP was slightly higher in the Esmolol group (130.39±4.61 mmHg) compared to the Lignocaine group (127.14±4.46 mmHg). At 1 minute post-intubation, SBP rose to 130.36±3.38 mmHg in the Esmolol group but surged to 138.17±4.42 mmHg in the Lignocaine group. By 5 minutes, SBP in the Esmolol group reduced to 123.44±7.09 mmHg, compared to 134.11±4.73 mmHg in the Lignocaine group. This demonstrates that Esmolol achieved quicker and more pronounced stabilization of SBP compared to Lignocaine, corroborating prior research. [18,23] In studies without intervention, SBP was reported to increase by 36-45% during intubation. [8,18,24] further emphasizing the efficacy of Esmolol in mitigating such changes. These results are consistent with Singh S et al. (2013), who reported percentage changes in SBP of 15.89% for Lignocaine and 10.20% for Esmolol at 1minute post-intubation. [25] Similarly, Koju RB et al. (2015) noted significantly lower SBP in the Esmolol group compared to the Lignocaine group, supporting the superiority of Esmolol in controlling SBP during intubation. [26] Jagadeesh GM et al.(2023) found that Esmolol was particularly effective in attenuating the SBP rise during intubation, producing significant suppression compared to lower doses or Lignocaine. [27] Our

study's results align with this dose-dependent efficacy. For DBP, the baseline values were 85.19±3.95 mmHg in the Esmolol group and 82.53±4.04 mmHg in the Lignocaine group. At 1 minute post-intubation, DBP increased to 85.56±4.63 mmHg in the Esmolol group and 90.53±1.86 mmHg in the Lignocaine group. By 5 minutes, DBP reduced significantly in the Esmolol group to 72.17±3.75 mmHg, while the Lignocaine group maintained higher values of 81.33±3.95 mmHg. These findings echo results from prior studies where Esmolol was superior to Lignocaine in controlling DBP fluctuations. [18,24] These findings also align with Tripathi S et al. (2023), who observed elevated DBP in the Lignocaine group throughout the study period, whereas Esmolol effectively controlled DBP. [21] Shrestha A et al. (2014) similarly found that Esmolol was more effective than Lignocaine in attenuating DBP increases following intubation. [28]

Our results demonstrated better MAP control in the Esmolol group compared to the Lignocaine group. Singh M et al. (2024) similarly observed significantly lower MAP in the Esmolol group at 1, 3 and 5 minutes post-intubation compared to Lignocaine (P<0.0001).20 Mendonça FT et al. (2022) also reported superior MAP control in the Esmolol group compared to Lignocaine. [13] These findings collectively highlight Esmolol's efficacy in reducing overall hemodynamic stress.

In terms of safety, our results indicate fewer adverse effects in the Esmolol group. For example, the incidence of hypotension was significantly lower in the Esmolol group (5.6%) compared to the Lignocaine group (19.4%) (P<0.05). Similarly, bradycardia was less frequent in the Esmolol group (5.6%) compared to the Lignocaine group (11.1%). This supports earlier findings that Esmolol is not only effective but also associated with fewer side effects. [18,19]

Our study noted lower incidences of bradycardia and hypotension in the Esmolol group compared to the Lignocaine group. This aligns with observations by Rao DS et al. (2022), where Esmolol achieved better hemodynamic control with minimal adverse effects. [29]

However, Jagadeesh GM et al. (2023) and Hatti R et al. (2016) highlighted dose-dependent side effects like bradycardia and hypotension at higher Esmolol doses, suggesting the importance of optimizing dosing. [27,30]

Conclusion

The study demonstrates that Esmolol is more effective than Lignocaine in controlling heart rate, systolic blood pressure, and diastolic blood pressure and mean arterial pressure during critical time intervals with significant differences observed. While both groups exhibited similar demographic characteristics in terms of age, gender, ASA grade and oxygen saturation, weight distribution differed significantly. Esmolol consistently maintained better stability and lower respiratory rates, especially at key intervals. Adverse effects such as hypotension were notably higher in the Lignocaine group, although other side effects were comparable. Overall, Esmolol proved to be a safer and more efficient option for hemodynamic management during the studied procedures.

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