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Comparing Esmolol and Lignocaine for Managing Blood Pressure and Heart Rate during Intubation: Which Works Better?

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Abstract: ***Background**: Endotracheal intubation via direct laryngoscopy frequently induces significant sympathetic cardiovascular responses, posing risks especially in patients with underlying cardiovascular or cerebrovascular disease. Pharmacological agents such as Esmolol and Lignocaine have been used to mitigate these hemodynamic responses. This study compares the effectiveness of these two agents. **Aims**: To evaluate and compare intravenous Esmolol and Lignocaine in attenuating hemodynamic responses to laryngoscopy and endotracheal intubation. **Study Design**: Prospective randomized controlled study. **Material and Methods**: The study was conducted from March 2023 to February 2025 at Hi - Tech Medical College & Hospital, Bhubaneswar. Sixty ASA grade I and II patients aged 18 - 60 years undergoing elective surgery were randomized into two groups: a) Group E (n=30): Received intravenous Esmolol 3 mg/kg. b) Group L (n=30): Received intravenous Lignocaine 2 mg/kg. Hemodynamic parameters—Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and Heart Rate (HR) —were recorded pre - intubation and at 0, 1, 3, 5, 10, and 15 minutes post - intubation. Adverse effects were monitored. **Results and Discussion**: Esmolol significantly attenuated SBP, DBP, MAP, and HR compared to Lignocaine at 1, 3, 5, 10, and 15 minutes post - intubation ($p<0.05$). Esmolol maintained stable cardiovascular parameters close to baseline throughout the observation period, whereas Lignocaine demonstrated sustained elevation, especially notable at 1 and 3 minutes post - intubation. No significant adverse effects were noted in either group. The study confirms Esmolol's superior efficacy, likely due to its cardio - selective β_1 - blockade reducing catecholamine - induced cardiovascular stress. **Conclusion**: Intravenous Esmolol is more effective than intravenous Lignocaine in attenuating hemodynamic responses during laryngoscopy and intubation. Esmolol provides superior and consistent control over SBP, DBP, MAP, and HR surges, enhancing perioperative cardiovascular stability and safety.*

Keywords: Esmolol, Lignocaine, Hemodynamic Response, Laryngoscopy, Intubation, General Anaesthesia, Cardiovascular Stability

1. Introduction

Balanced anaesthesia involves rapid induction of unconsciousness using intravenous agents and muscle relaxation to facilitate intubation. However, laryngoscopy and intubation induce a sympathetic surge, increasing heart rate and blood pressure, which may be hazardous for patients with cardiovascular or cerebrovascular conditions. These hemodynamic fluctuations can exacerbate conditions like coronary artery disease, heart failure, thoracic aortic aneurysm, and intracranial vascular anomalies, leading to increased morbidity and mortality.

Non - pharmacological strategies such as optimal patient positioning, gentle airway manipulation, video laryngoscopy, and alternative airway devices like laryngeal mask airways (LMA) help reduce sympathetic stimulation. Pharmacological agents, including lignocaine, beta - blockers, calcium channel blockers, and α2 - adrenergic agonists like dexmedetomidine, have also been used to blunt pressor responses.

Esmolol, a short - acting β1 - blocker, and lignocaine, a sodium channel blocker, have demonstrated efficacy in controlling hemodynamic responses during intubation. Studies indicate that both drugs reduce increases in heart rate and blood pressure, with esmolol exhibiting a more pronounced effect. This study aims to compare the effectiveness of intravenous lignocaine and esmolol in mitigating cardiovascular responses to laryngoscopy and intubation, evaluating their impact on perioperative hemodynamic stability.

2. Methods and Methodology

This prospective randomized study was conducted in the Department of Anaesthesiology, Hi - Tech Medical College & Hospital, Bhubaneswar, from March 2023 to February 2025. Sixty ASA grade I and II patients, aged 18 - 60 years, posted for elective surgery under general anaesthesia were enrolled after obtaining informed consent. Patients were randomly assigned to two groups: Group L (Lignocaine 2 mg/kg IV) and Group E (Esmolol 3 mg/kg IV). Exclusion criteria included patients with uncontrolled diabetes, IHD, COPD, recent MI/CVA, asthma, hypertension, hepatic or renal dysfunction, pregnancy, emergency surgical intubation, anticipated difficult intubation (Mallampati III/IV), hypersensitivity to study drugs, and significantly deranged preoperative hemodynamic parameters (BP>180/100 mmHg, PR <60 bpm or >140 bpm).

All patients underwent pre - anaesthetic evaluation, including history, physical examination, and investigations (CBC, coagulation profile, blood sugar, ECG, and chest X - ray). Pre - operatively, an 18G IV cannula was secured, and patients were pre - hydrated with Normal Saline (NS) or Ringer's Lactate (RL). Standard monitoring included HR, NIBP, MAP, and SpO2. Pre - medication included IV Glycopyrrolate 0.2

mg and Midazolam 1 mg, followed by pre - oxygenation for 3 minutes. Induction was performed with IV Propofol 2 mg/kg and Vecuronium 0.1 mg/kg. Study drugs were administered 60 seconds after muscle relaxation, and laryngoscopy with intubation was performed after 4 minutes. Anaesthesia was maintained with a 50: 50 O2: N2O mixture, Isoflurane, and incremental doses of Vecuronium. At the end of surgery, neuromuscular blockade was reversed using IV Neostigmine 0.05 mg/kg and Glycopyrrolate 0.01 mg/kg. Patients were observed in the post - anaesthesia care unit before transfer to the ward.

Hemodynamic parameters (HR, SBP, DBP, MAP, SpO2) were recorded at baseline, post - induction, and at 1, 3, 5, 10, and 15 minutes post - intubation. ECG monitoring and adverse effects were noted. Data was analysed using appropriate statistical methods and presented as tables and graphs for clinical interpretation.

3. Results

Table 1: Demographic characters of the patients in two groups

Demography and other parameters of patients	Group E (n=30) (Mean ± SD)	Group L (n=30) (Mean ± SD)	P - value
Age in years	41.17±12.77	41.33±12.64	0.641
Sex (M/F)	12/18	19/11	0.642
Weight in kgs	65.77±14.18	60.07±9.22	0.548
Height in meters	1.60±0.14	1.63±0.092	0.892
BMI in kg/m^2	24.29±2.74	25.28±3.36	0.980

Table 2: Baseline parameters of patients in Group E and Group L

Parameter	Group E (Mean ± SD)	Group L (Mean ± SD)	P - value
SBP in mm of Hg	124.37±8.03	126.50±10.46	0.639
DBP in mm of Hg	79.67±7.74	78.57±7.29	0.616
MAP in mm of Hg	94.50±6.73	94.3±6.69	0.556
Heart Rate in beats/min	85.27±8.61	86.90±7.85	0.057

Table 3: Depicts the changes in mean and SD of SBP at different time intervals in group E and group L.

Time	N	Esmolol Mean	SD	Lignocaine MEAN	SD	T Value	P Value
Pre – Intubation	30	106.80	9.84	113.97	11.07	2.65	0.01
After Intubation (0 Min)	30	119.30	12.28	114.03	10.54	1.78	0.07
1 Min	30	119.53	7.92	141.07	8.26	10.3	0.0001
3 Min	30	116.43	8.29	138.00	8.6	9.88	0.0001
5 Min	30	111.63	7.83	132.57	6.67	10.19	0.0001
10 Min	30	118.5	8.0	130.2	8.0	4.02	0.0003
15 Min	30	117.5	8.4	119.0	9.0	3.27	0.0028

Table 4: Depicts the changes in mean diastolic blood pressure at different time intervals between group E and group L

Time	N	Esmolol Mean	SD	Lignocaine MEAN	SD	T Value	P Value
Pre – Intubation	30	69.73	9.73	79.03	7.82	4.08	0.0001
After Intubation (0 Min)	30	76.17	8.95	78.40	8.04	1.01	0.31
1 Min	30	73.23	7.91	88.37	4.66	9.03	0.0001
3 Min	30	76.27	10.64	86.00	6.17	4.33	0.0001
5 Min	30	72.73	8.67	82.00	5.93	4.38	0.0001
10 Min	30	73.30	7.74	85.00	6.47	4.51	0.0001
15 Min	30	70.00	11.76	74.44	6.35	4.51	0.0001

Table 5: Depicts the changes in mean arterial blood pressure at different time intervals between group E and group L.

Time	N	Esmolol Mean	SD	Lignocaine MEAN	SD	T Value	P Value
Pre – Intubation	30	82.07	9.31	90.67	6.32	4.18	0.0001
After Intubation (0 Min)	30	90.43	8.14	90.37	6.71	0.03	0.97
1 Min	30	88.60	6.11	106.03	5.26	11.84	0.0001
3 Min	30	89.63	8.56	103.37	6.55	6.98	0.0001
5 Min	30	85.73	7.63	98.80	5.23	7.73	0.0001
10 Min	30	81.25	1.91	85.00	6.46	4.52	0.0001
15 Min	30	81.25	1.57	85.00	5.31	5.50	0.0001

Table 6: Depicts the comparison of changes in mean heart rate at different time intervals between group E and group L

Time	N	Esmolol Mean	SD	Lignocaine MEAN	SD	T Value	P Value
Pre – Intubation	30	80.01	9.22	80.97	8.01	0.39	0.6900
After Intubation (0 Min)	30	84.23	9.07	82.93	6.88	0.62	0.53
1 Min	30	81.93	8.96	104.00	10.67	8.67	0.0001
3 Min	30	79.27	8.04	100.60	11.74	8.21	0.0001
5 Min	30	77.73	9.65	97.57	11.88	6.38	0.0001

10 Min	30	82.00	6.80	85.00	6.46	4.52	0.0001
15 Min	30	82.00	5.75	85.00	5.83	5.01	0.001

4. Discussion

Endotracheal intubation induces significant hemodynamic disturbances due to sympathetic stimulation, resulting in increased HR, SBP, DBP, and MAP. These fluctuations pose risks, particularly for cardiac patients, increasing the likelihood of myocardial ischemia and arrhythmias. Various pharmacological agents, including lignocaine, fentanyl, magnesium sulphate, and esmolol, have been evaluated to blunt this response [1, 2].

This study compared intravenous esmolol, a β1 - adrenergic blocker with an ultrashort half - life (~9 min) and rapid onset (1 - 2 min), to intravenous lignocaine, a local anaesthetic acting via sodium channel blockade. Both study groups were demographically comparable, eliminating baseline variability. Additionally, preoperative baseline hemodynamic parameters (SBP, DBP, MAP, HR) were similar ($P > 0.05$), ensuring unbiased cardiovascular status before drug administration.

Both groups exhibited SBP increases at 1, 3, and 5 minutes post - intubation, but the esmolol group demonstrated significantly lower SBP elevations compared to the lignocaine group ($P < 0.05$), suggesting superior attenuation [3, 4]. By 10 minutes, SBP declined, stabilizing near baseline at 15 minutes. This aligns with the study by Ugur B and Ogurlu M [5], which found lignocaine alone to be insufficient in fully blunting SBP surges post - intubation. Helfman et al. [6] further confirmed that esmolol effectively controls both HR and BP when administered as a bolus dose or infusion, preventing hypertensive surges compared to lignocaine.

Diastolic Blood Pressure (DBP) Response: Post - intubation, DBP increased significantly in both groups. However, esmolol exhibited significantly lower DBP at 1, 3, and 5 minutes ($P < 0.05$) [7]. By 10 minutes, DBP declined, stabilizing at 15 minutes. Studies by Aleem et al. [8] and Bostana & Eroglu [9] confirmed esmolol's efficacy in reducing BP fluctuations post - intubation. Additionally, Bostan and Eroglu demonstrated that IV esmolol (1 mg/kg) effectively suppressed HR and arterial BP rise during intubation, aligning with the findings of this study.

Mean Arterial Pressure (MAP) Response: Both groups exhibited significant MAP increases at 1, 3, and 5 minutes, but the esmolol group maintained significantly lower MAP values ($P < 0.05$) [10]. By 10 minutes, MAP declined, stabilizing at 15 minutes. This corroborates studies by Abou - Madi et al. [11], which found that while lignocaine effectively reduced arrhythmias, it provided only partial MAP control. Singh et al. [12] further demonstrated esmolol's superiority over lignocaine in blunting the pressor response.

Heart Rate (HR) Response: HR increased in both groups at 1, 3, and 5 minutes post - intubation, but esmolol significantly reduced HR spikes ($P < 0.05$) [13]. By 5 minutes, HR returned to near baseline in the esmolol group, whereas the lignocaine group exhibited a prolonged tachycardic response. Studies by Helfman et al. [14] and Sharma et al. [15] confirmed esmolol's effectiveness in controlling HR, demonstrating that esmolol significantly attenuated tachycardic surges post - intubation compared to lignocaine. Additionally, research by Gupta & Tank [16] and Bensky et al. [17] found esmolol to effectively suppress HR and BP surges during intubation.

This study confirms that esmolol is significantly more effective than lignocaine in attenuating hemodynamic responses to laryngoscopy and intubation. Its β1 - blocking action minimizes HR and BP surges, making it the preferred agent for perioperative cardiovascular stability, particularly in high - risk patients. Findings also align with prior studies by Singh et al. [18], reinforcing esmolol's clinical advantage. Lignocaine, while beneficial in some aspects, remains a secondary choice when β - blockers are contraindicated. Further studies with larger sample sizes and alternative dosing regimens are warranted to optimize management strategies.

5. Limitations

1) **Lack of plasma drug concentration measurement:** The study did not measure plasma concentrations of esmolol and lignocaine, which could have provided more precise pharmacokinetic correlations with their hemodynamic effects. This may have influenced the accuracy of clinical parameter interpretations.
2) **Absence of stress hormone analysis:** The study did not assess plasma catecholamine or stress hormone levels in response to laryngoscopy and intubation. Measuring these biomarkers could have offered additional insights into the sympathetic activation and the efficacy of drug interventions.
3) **Premedication timing as a limiting factor:** The duration of premedication administration could not be standardised, potentially affecting the depth of sedation and altering baseline hemodynamic parameters before intubation. A universal premedication period could have contributed to better hemodynamic evaluation.
4) **Limited sample size:** The study was conducted on a relatively small sample size (n = 60), which may limit the generalizability of the findings. A larger study population could provide more robust statistical validation and improve the reliability of the conclusions

6. Conclusion

This study validates intravenous Esmolol as a superior agent over intravenous Lignocaine for attenuating sympathetic cardiovascular responses during laryngoscopy and intubation, recommending its preferential use in clinical practice for enhanced patient safety.

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Conflicts of Interest
None declared.

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