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Comparative Study of Lignocaine Nebulization Versus Intravenous Lignocaine in Attenuating Hemodynamic Response to Laryngoscopy and Endotracheal Intubation

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ABSTRACT

Background and Aims: Laryngoscopy and tracheal intubation are associated with marked sympathetic stimulation, resulting in transient hypertension and tachycardia. While lignocaine is widely used to blunt these responses, the relative efficacy of intravenous versus nebulized administration remains uncertain. This study compared intravenous and nebulized lignocaine for attenuation of pressor responses during elective intubation.

Methods: In this randomized, single-blinded trial, 100 ASA I–II patients aged 18–55 years undergoing elective surgery under general anaesthesia were allocated into two groups (n = 50 each). Group IL received intravenous lignocaine 2% (2 mg/kg) 90 seconds before intubation, while Group NL received nebulized lignocaine 2% (2 mg/kg) 10 minutes prior to induction. Hemodynamic parameters (heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure) were recorded at baseline, at 1–5 minutes after intubation, and every 2 minutes up to 15 minutes. Statistical analysis was performed using Student's t-test and repeated measures ANOVA.

Results: Baseline demographic and hemodynamic variables were comparable between groups. Post-intubation, Group IL showed significantly attenuated rises in heart rate (mean 86.6 ± 7.4 vs. 100.0 ± 12.2 bpm at 3 min, $p < 0.001$), systolic pressure (138.9 ± 11.5 vs. 152.9 ± 12.2 mmHg at 1 min, $p < 0.001$), diastolic pressure (93.6 ± 8.2 vs. 102.5 ± 11.1 mmHg at 1 min, $p < 0.001$), and mean arterial pressure (104.3 ± 9.7 vs. 119.3 ± 10.0 mmHg at 1 min, $p < 0.001$). Significant differences persisted until 5 minutes, after which values converged. No adverse events were observed in either group.

Conclusion: Intravenous lignocaine 2 mg/kg given 90 seconds before intubation was more effective than nebulized lignocaine in attenuating the hemodynamic response to laryngoscopy and intubation. Nebulized lignocaine, though non-invasive, provided incomplete protection, particularly against peak surges in the first five minutes. Intravenous lignocaine remains the preferred method for routine elective intubations, while nebulized administration may be considered when intravenous use is not feasible.

Keywords*: Lignocaine; Intravenous; Nebulization; Laryngoscopy; Intubation; Hemodynamic Response.*

INTRODUCTION

Laryngoscopy and tracheal intubation are integral to general anaesthesia but are accompanied by significant sympathetic stimulation, leading to transient tachycardia and hypertension. These changes may be well tolerated in healthy individuals but can precipitate myocardial ischemia, arrhythmias, or cerebrovascular complications in high-risk patients

[1]. Bedford and Marshal (1984) confirmed that such cardiovascular responses occur across multiple anaesthetic techniques, with systolic pressures often rising by 20–30 mmHg [2].

The underlying mechanism involves intense stimulation of the upper airway, particularly the supraglottic and laryngeal structures, resulting in reflex sympathetic discharge and catecholamine release. Stoelting (1978) demonstrated that even brief laryngoscopy produced marked elevations in heart rate and blood pressure, highlighting the need for attenuation strategies [3]. Earlier, Nair et al. (1965) documented consistent rises in vital signs and electrocardiographic disturbances during intubation, strengthening the clinical relevance of these findings [4].

Several pharmacological interventions have been evaluated to blunt this response. Abou-Madi et al. (1977) showed that intravenous lignocaine reduced the rise in systolic blood pressure from nearly 45 mmHg in controls to less than 20 mmHg with higher doses [5]. In an earlier report, the same group recommended intravenous lignocaine premedication as a simple and effective method for preventing cardiovascular reactions to laryngoscopy [7]. Stoelting (1978) also noted that intravenous lignocaine was more effective than viscous topical lignocaine in limiting pressor responses [3].

Nebulized and topical preparations have also been studied. Williams et al. (2005) described a combination of nebulization and "spray-as-you-go" topical lignocaine, which improved airway tolerance and reduced circulatory perturbations during awake fibreoptic intubations [6]. However, the reliability of nebulized lignocaine in routine laryngoscopy remains debated, with some studies suggesting incomplete attenuation of hemodynamic responses.

Against this background, lignocaine remains one of the most widely used and investigated agents for attenuating the intubation response. However, the comparative efficacy of intravenous versus nebulized lignocaine remains incompletely defined, with conflicting evidence in the literature. The present study was therefore designed to compare the efficacy of intravenous and nebulized lignocaine in attenuating the pressor response to laryngoscopy and tracheal intubation in ASA I–II patients undergoing elective surgery.

OBJECTIVES

The primary objective of this study was to compare the efficacy of intravenous lignocaine (2 mg/kg administered 90 seconds prior to intubation) with nebulized lignocaine (2 mg/kg administered 10 minutes prior to induction) in attenuating the hemodynamic responses to laryngoscopy and tracheal intubation.
The specific objectives were:

1. To evaluate and compare changes in heart rate between the two groups at baseline and at defined time intervals following intubation.
2. To assess and compare changes in systolic blood pressure, diastolic blood pressure, and mean arterial pressure during the same period.
3. To observe and record any adverse events, such as hypotension, bradycardia, or arrhythmias, associated with either route of lignocaine administration.

METHODS

Study Design and Setting

This was a prospective, randomized, single-blinded, comparative clinical study conducted in the Department of Anaesthesiology, Mysore Medical College and Research Institute. Ethical committee approval was obtained, and written informed consent was taken from all participants. The study included 100 patients scheduled for elective surgeries under general anaesthesia requiring endotracheal intubation.

Participants

Patients of either sex, aged 18–55 years, belonging to American Society of Anaesthesiologists (ASA) physical status I or II, and with Mallampati class I–II airways were eligible for inclusion.

Exclusion criteria were: ASA grade III or IV, anticipated difficult airway, obesity (BMI > 30 kg/m²), cervical spine instability, endocrine or cardiovascular disorders, known allergy to lignocaine, pregnancy, and lactation.

Randomization and Group Allocation

Using a computer-generated random sequence, patients were assigned into two equal groups (n = 50 each):

- **Group IL**: Received intravenous lignocaine 2% at a dose of 2 mg/kg, administered 90 seconds prior to laryngoscopy and intubation.
- **Group NL**: Received nebulized lignocaine 2% at a dose of 2 mg/kg via face mask, administered 10 minutes before induction.

The anaesthesiologist recording hemodynamic data was blinded to group allocation.

Anaesthesia Technique
All patients were premedicated with midazolam (0.02 mg/kg IV) and fentanyl (2 µg/kg IV). Standard monitoring (ECG, non-invasive blood pressure, and pulse oximetry) was applied. Anaesthesia was induced with propofol (2 mg/kg IV) and muscle relaxation achieved with vecuronium (0.1 mg/kg IV). Laryngoscopy and tracheal intubation were performed using a Macintosh laryngoscope by experienced anaesthesiologists within 15 seconds. Anaesthesia was maintained with isoflurane in 50% oxygen and nitrous oxide.

Hemodynamic Monitoring
Hemodynamic parameters recorded included heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP). Measurements were obtained at:

- Baseline (before induction),
- 1, 2, 3, 4, and 5 minutes after intubation,
- Every 2 minutes thereafter up to 15 minutes post-intubation.

Adverse events such as hypotension, bradycardia, or arrhythmias were noted.

Statistical Analysis
Sample size was estimated based on previous studies to detect a 20% difference in mean arterial pressure with 80% power and $\alpha = 0.05$, requiring 45 patients per group; 50 were recruited per group to account for possible dropouts. Data were analyzed using SPSS software version 20.0. Continuous variables were expressed as mean ± standard deviation (SD) and compared using independent Student's t-test. Repeated measures analysis of variance (ANOVA) was used for intra-group comparisons. Categorical variables were analyzed using the chi-square test. A p-value < 0.05 was considered statistically significant.

RESULTS
Patient Characteristics
A total of 100 patients (ASA I–II, aged 18–55) were enrolled and randomized equally into two groups: Group IL (intravenous lignocaine) and Group NL (nebulized lignocaine). Both groups were statistically comparable with respect to demographic variables including age, sex, and body weight. The mean age in Group IL was 33.1 ± 9.4 years and in Group NL was 31.8 ± 9.4 years ($p = 0.51$). There were slightly more females in both groups (62% in IL vs. 54% in NL), though this was not statistically significant ($p = 0.41$). Body weights were similar across groups ($p = 0.75$). No significant baseline differences were identified (Table 1).

Table 1. Baseline Characteristics of the Study Population

Parameter	Group IL (n = 50)	Group NL (n = 50)	p-value
Age (years)	33.1 ± 9.4	31.8 ± 9.4	0.51
Sex (M/F)	19 / 31	23 / 27	0.41
Weight (kg)	54.2 ± 6.3	53.7 ± 8.3	0.75

Hemodynamic Parameters
Baseline hemodynamic parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were comparable between the two groups, with no statistically significant differences observed prior to induction.

Following laryngoscopy and intubation, patients in Group IL (intravenous lignocaine) demonstrated significantly attenuated increases in all four hemodynamic variables compared to Group NL (nebulized lignocaine) during the first five minutes. These differences diminished progressively and were not statistically significant beyond the fifth minute.

Heart Rate
At baseline, mean heart rate (HR) values were comparable between the two groups (Group IL: 87.4 ± 10.4 bpm; Group NL: 85.9 ± 11.3 bpm; $p = 0.49$). Following laryngoscopy and intubation, both groups exhibited an increase in HR. However, the magnitude of this rise was significantly attenuated in the intravenous lignocaine group.

Group IL had significantly lower HR at 1 minute (101.0 ± 9.1 vs. 105.4 ± 11.3 bpm; $p = 0.03$), 2 minutes (97.3 ± 7.0 vs. 102.9 ± 9.7 bpm; $p = 0.001$), 3 minutes (86.6 ± 7.4 vs. 100.0 ± 12.2 bpm; $p < 0.001$), 4 minutes (84.2 ± 8.7 vs. 93.9 ± 10.8 bpm; $p < 0.001$), and 5 minutes (88.9 ± 7.9 vs. 94.3 ± 12.7 bpm; $p = 0.012$). No statistically significant differences in HR were observed between the groups after 5 minutes. These findings are summarized in **Table 2** and visualized in **Figure 1**.

Table 2. Comparison of Mean Heart Rate (bpm) Between Groups at Different Time Points

Time Point	Group IL (Mean ± SD)	Group NL (Mean ± SD)	p-value
Baseline	87.4 ± 10.4	85.9 ± 11.3	0.49

1 minute	101.0 ± 9.1	105.4 ± 11.3	0.03
2 minutes	97.3 ± 7.0	102.9 ± 9.7	0.001
3 minutes	86.6 ± 7.4	100.0 ± 12.2	<0.001
4 minutes	84.2 ± 8.7	93.9 ± 10.8	<0.001
5 minutes	88.9 ± 7.9	94.3 ± 12.7	0.012
7 minutes	90.0 ± 9.0	90.5 ± 12.1	0.78
9 minutes	88.5 ± 8.0	90.3 ± 12.1	0.37
11 minutes	87.4 ± 10.5	89.1 ± 12.7	0.46
13 minutes	84.9 ± 7.8	86.6 ± 12.0	0.41
15 minutes	84.6 ± 10.0	86.5 ± 11.8	0.38

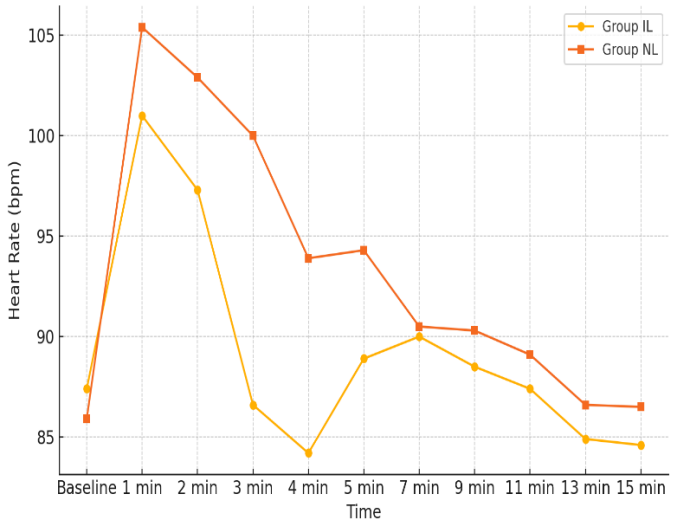

Figure1. Heart Rate Over Time

Systolic Blood Pressure

Baseline systolic blood pressure (SBP) values were comparable between the two groups (Group IL: 121.8 ± 10.6 mmHg; Group NL: 122.4 ± 10.6 mmHg; p = 0.76). Following intubation, both groups exhibited an increase in SBP, but the rise was more pronounced in the nebulized lignocaine group.

At 1-minute post-intubation, Group NL showed a significantly higher SBP (152.9 ± 12.2 mmHg) compared to Group IL (138.9 ± 11.5 mmHg; p < 0.001). This difference remained statistically significant at 2 minutes (145.7 ± 16.3 vs. 137.3 ± 16.4 mmHg; p = 0.013), 3 minutes (138.8 ± 18.3 vs. 129.6 ± 14.3 mmHg; p = 0.007), 4 minutes (133.6 ± 16.6 vs. 128.6 ± 11.7 mmHg; p = 0.008), and 5 minutes (132.5 ± 11.2 vs. 125.2 ± 13.8 mmHg; p = 0.005). Beyond the 5-minute mark, the differences were not statistically significant. These values are detailed in **Table 3** and visualized in **Figure 2**.

Table 3. Comparison of Mean Systolic Blood Pressure (mmHg) Between Groups

Time Point	Group IL (Mean ± SD)	Group NL (Mean ± SD)	p-value
Baseline	121.8 ± 10.6	122.4 ± 10.6	0.76
1 minute	138.9 ± 11.5	152.9 ± 12.2	<0.001
2 minutes	137.3 ± 16.4	145.7 ± 16.3	0.013
3 minutes	129.6 ± 14.3	138.8 ± 18.3	0.007
4 minutes	128.6 ± 11.7	133.6 ± 16.6	0.008
5 minutes	125.2 ± 13.8	132.5 ± 11.2	0.005
7 minutes	127.3 ± 10.1	128.5 ± 12.9	0.58
9 minutes	125.1 ± 9.5	127.6 ± 12.0	0.25
11 minutes	124.1 ± 9.1	125.9 ± 10.8	0.37
13 minutes	124.0 ± 12.0	128.1 ± 11.3	0.08
15 minutes	125.0 ± 12.7	129.5 ± 11.0	0.06

Diastolic blood pressure

Baseline diastolic blood pressure (DBP) was comparable between the groups (Group IL: 78.0 ± 8.5 mmHg; Group NL: 78.8 ± 7.6 mmHg; p = 0.60). Following intubation, both groups experienced a rise in DBP; however, the increase was significantly lower in the intravenous lignocaine group during the first five minutes.

At 1 minute, Group IL recorded a DBP of 93.6 ± 8.2 mmHg, significantly lower than Group NL (102.5 ± 11.1 mmHg; $p < 0.001$). This pattern persisted at 2 minutes (89.9 ± 8.6 vs. 94.4 ± 9.1 mmHg; $p = 0.01$), 3 minutes (84.1 ± 6.0 vs. 88.3 ± 9.8 mmHg; $p = 0.01$), 4 minutes (81.1 ± 5.0 vs. 85.1 ± 11.8 mmHg; $p = 0.02$), and 5 minutes (79.6 ± 8.8 vs. 83.4 ± 10.5 mmHg; $p = 0.04$). No significant differences were observed from 7 minutes onward. Complete data are shown in **Table 4**

Table 4. Comparison of Mean Diastolic Blood Pressure (mmHg) Between Groups

Time Point	Group IL (Mean ± SD)	Group NL (Mean ± SD)	p-value
Baseline	78.0 ± 8.5	78.8 ± 7.6	0.60
1 minute	93.6 ± 8.2	102.5 ± 11.1	<0.001
2 minutes	89.9 ± 8.6	94.4 ± 9.1	0.01
3 minutes	84.1 ± 6.0	88.3 ± 9.8	0.01
4 minutes	81.1 ± 5.0	85.1 ± 11.8	0.02
5 minutes	79.6 ± 8.8	83.4 ± 10.5	0.04
7 minutes	82.1 ± 12.0	83.0 ± 11.1	0.71
9 minutes	82.5 ± 10.3	85.9 ± 8.0	0.06
11 minutes	81.9 ± 8.2	83.1 ± 7.9	0.45
13 minutes	83.5 ± 10.6	83.8 ± 7.7	0.84
15 minutes	81.6 ± 11.2	84.2 ± 7.5	0.18

Mean Arterial Pressure

Baseline mean arterial pressure (MAP) was statistically similar between the two groups (Group IL: 91.7 ± 8.5 mmHg; Group NL: 93.4 ± 7.8 mmHg; $p = 0.32$). However, following intubation, Group IL demonstrated significantly attenuated MAP responses during the first five minutes.

At 1 minute, MAP in Group NL increased to 119.3 ± 10.0 mmHg, significantly higher than Group IL (104.3 ± 9.7 mmHg; $p < 0.001$). This significant difference persisted at 2 minutes (111.5 ± 9.3 vs. 105.7 ± 10.3 mmHg; $p = 0.004$), 3 minutes (105.1 ± 11.3 vs. 99.3 ± 7.2 mmHg; $p = 0.003$), 4 minutes (101.2 ± 12.5 vs. 96.9 ± 5.9 mmHg; $p = 0.02$), and 5 minutes (99.9 ± 9.9 vs. 94.9 ± 9.5 mmHg; $p = 0.01$). Beyond this interval, MAP values converged and no longer differed significantly. These results are detailed in **Table 5** and graphically represented in **Figure 3**.

Table 5. Comparison of Mean Arterial Pressure (mmHg) Between Groups

Time Point	Group IL (Mean ± SD)	Group NL (Mean ± SD)	p-value
Baseline	91.7 ± 8.5	93.4 ± 7.8	0.32
1 minute	104.3 ± 9.7	119.3 ± 10.0	<0.001
2 minutes	105.7 ± 10.3	111.5 ± 9.3	0.004
3 minutes	99.3 ± 7.2	105.1 ± 11.3	0.003
4 minutes	96.9 ± 5.9	101.2 ± 12.5	0.02
5 minutes	94.9 ± 9.5	99.9 ± 9.9	0.01
7 minutes	97.2 ± 9.8	98.1 ± 10.7	0.64
9 minutes	96.7 ± 8.2	99.8 ± 8.7	0.06
11 minutes	95.8 ± 6.9	97.3 ± 8.0	0.33
13 minutes	97.0 ± 9.6	98.6 ± 8.0	0.36
15 minutes	96.1 ± 10.3	99.4 ± 7.5	0.07

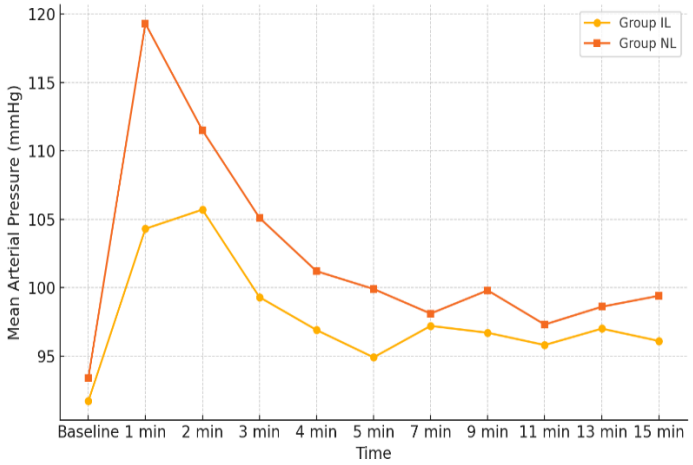

Figure 3. Mean Arterial Pressure (mmHg) Over Time

Mean arterial pressure trends between the two groups over time. Group IL exhibited significantly lower MAP values from 1 to 5 minutes post-intubation.

Adverse Events

No adverse events, including hypotension, bradycardia, or arrhythmias, were observed in any patient in either group throughout the study period. Both interventions were well tolerated.

Summary of Key Findings

The comparative effects of intravenous and nebulized lignocaine on hemodynamic responses to laryngoscopy and intubation are summarized in **Table 6**. Intravenous lignocaine consistently attenuated heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure during the first five minutes post-intubation, whereas no significant differences were observed beyond this interval.

Table 6. Summary of Key Hemodynamic Findings

Parameter	Time Frame	Finding
Heart Rate	1–5 min post-intubation	Significantly lower in IL group ($p < 0.05$)
SBP	1–5 min post-intubation	Significantly lower in IL group ($p < 0.05$)
DBP	1–5 min post-intubation	Significantly lower in IL group ($p < 0.05$)
MAP	1–5 min post-intubation	Significantly lower in IL group ($p < 0.05$)

Note: Beyond 5 minutes, all hemodynamic variables were comparable between the two groups, with no statistically significant differences.

DISCUSSION

The present study demonstrated that intravenous lignocaine (2 mg/kg administered 90 seconds before intubation) significantly attenuated the pressor response to laryngoscopy and tracheal intubation compared with nebulized lignocaine (2 mg/kg administered 10 minutes before induction). Specifically, patients in the intravenous group had consistently lower heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure during the first five minutes post-intubation, whereas beyond this interval the groups showed comparable hemodynamic trends.

These findings are consistent with the observations of Selvambika and Shetty (2019), who reported that intravenous lignocaine blunted the intubation-induced rise in systolic pressure by approximately 12–15 mmHg compared with inhaled lignocaine, while nebulized lignocaine produced only partial attenuation of the heart rate response [8]. Similarly, Puntambekar and Deshpande (2019) found that intravenous lignocaine produced a mean reduction of 8 beats/min in heart rate and 10 mmHg in mean arterial pressure at 3 minutes post-intubation compared with nebulized delivery [9]. In contrast, Jokar et al. (2018) suggested that both intravenous and nebulized routes were equally effective, with mean heart rates of 94 bpm and 96 bpm respectively at 3 minutes, although their cohort involved younger ASA I patients with shorter laryngoscopy times, which may partly explain the divergence from our results [10].

The superiority of intravenous lignocaine in our study also aligns with the findings of Ahmed et al. (2016), who reported a significant attenuation of systolic pressure rise (mean increase 12 mmHg vs. 22 mmHg in controls) and a concurrent reduction in hospital morbidity when intravenous lignocaine was used [11]. Saravanan et al. (2016) likewise observed that intravenous lignocaine limited heart rate to below 95 bpm in the first 3 minutes post-intubation, whereas nebulized

lignocaine was less effective, with peak rates approaching 105 bpm [12]. Our results fall within these ranges, with mean heart rates of 86.6 bpm versus 100.0 bpm at 3 minutes for intravenous and nebulized groups respectively.

Variation in nebulized lignocaine efficacy across studies may be attributed to differences in drug concentration and nebulization technique. Patil et al. (2015) compared 2% and 4% lignocaine nebulization and found that higher concentrations achieved reductions in systolic pressure comparable to intravenous lignocaine, suggesting dose dependency [13]. Kocamanoglu et al. (2015) also demonstrated that lignocaine nebulization significantly blunted the pressor response during rigid suspension laryngoscopy, although their study used a larger pre-intubation dose and topical airway deposition, which differs methodologically from our protocol [14].

Meta-analytical data further support the role of intravenous lignocaine. Qi et al. (2013), in a systematic review of randomized controlled trials, concluded that intravenous lignocaine reduced mean arterial pressure rise by an average of 14 mmHg compared with placebo, particularly when given 90 seconds before intubation [15]. The importance of timing was highlighted earlier by Tarn et al. (1987), who showed maximal efficacy when lignocaine was administered 90 seconds pre-intubation, consistent with our protocol [16].

The concept of aerosolized lignocaine for blunting intubation responses is not novel. Venus et al. (1984) demonstrated some attenuation of circulatory responses with aerosolized lidocaine, although the reduction was modest, with heart rate still exceeding 110 bpm in many patients [17]. Early observations by Gibbs (1967) similarly documented consistent tachycardia despite nebulized lignocaine use, raising questions about the reliability of this route [18]. Our results support these earlier conclusions, showing that nebulization does not adequately prevent the peak hemodynamic surges following intubation.

Other pharmacological agents have been studied for the same purpose. Chen et al. (1986) reported that fentanyl 5 μg/kg reduced systolic pressure rise more effectively than lignocaine alone [20], while Stoelting (1979) demonstrated profound blunting of systolic and diastolic responses with sodium nitroprusside infusion [21]. More recently, Narayanan and Deepa (2017) observed that esmolol at doses of 1–2 mg/kg attenuated serum cortisol responses as well as hemodynamic surges more effectively than lignocaine [22]. Leslie et al. (1989) also showed that labetalol premedication maintained both heart rate and mean arterial pressure within 10% of baseline during intubation [24]. While these alternatives may be more potent, lignocaine remains attractive due to its dual role as a local anaesthetic and antiarrhythmic, ease of administration, and favourable safety profile.

The differences across published studies underscore the influence of regional practice and methodological variation. Factors such as lignoscopy duration, pre-induction medications, patient comorbidities, and even nebulizer efficiency can alter the observed efficacy of lignocaine. For instance, Supriya Saravanan et al. (2016) included longer laryngoscopy times and found exaggerated responses despite lignocaine use [12], whereas our study excluded anticipated difficult intubations, thereby minimizing variability.

In summary, our findings corroborate much of the existing literature indicating that intravenous lignocaine at 2 mg/kg administered 90 seconds before intubation provides reliable attenuation of pressor responses, limiting rises in heart rate and blood pressure to within 10–15% of baseline. Nebulized lignocaine, though non-invasive, was less effective, particularly in blunting peak responses in the critical first 5 minutes. This suggests that intravenous lignocaine remains the preferred modality for attenuating hemodynamic responses during routine elective intubations, while nebulized lignocaine may serve as an adjunct in select cases where intravenous administration is contraindicated.

Limitations

This was a single-centre study with a modest sample size, limited to ASA I–II patients undergoing elective surgeries, which may restrict generalizability to higher-risk populations. Only a single dose of lignocaine was tested, and no placebo or active comparator arms (e.g., opioids, beta-blockers, alpha-2 agonists) were included. These factors should be considered when interpreting the results.

CONCLUSION

Intravenous lignocaine at a dose of 2 mg/kg administered 90 seconds before intubation was more effective than nebulized lignocaine in attenuating the hemodynamic responses to laryngoscopy and tracheal intubation. Significant reductions in heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were observed in the intravenous group during the first five minutes post-intubation, whereas both groups showed comparable values thereafter. Given its consistent efficacy, ease of administration, and safety, intravenous lignocaine remains the preferred method for blunting pressor responses in routine elective intubations, while nebulized lignocaine may serve as an alternative in select clinical scenarios where intravenous administration is undesirable.

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