Section: Anaesthesiology

Original Research Article

# COMPARISON OF NEBULIZED 4% LIGNOCAINE AND NEBULIZED 0.25% ROPIVACAINE AND LIGNOCAINE 10% SPRAY ON PRESSOR RESPONSE IN PATIENTS UNDERGOING ELECTIVE SURGERY UNDER GENERAL ANAESTHESIA

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## ABSTRACT

**Background:** Laryngoscopy and tracheal intubation produce a transient sympathoadrenal response that may be clinically important in patients with cardiovascular or cerebrovascular disease. Topical airway local anaesthesia is a simple approach for attenuating this response. This study compared nebulised 4% lignocaine, nebulised 0.25% ropivacaine and 10% lignocaine spray with saline nebulisation.

**Materials and Methods:** This prospective randomized controlled study included 120 adults aged 18–60 years, American Society of Anaesthesiologists (ASA) physical status I–II, undergoing elective surgery under general anaesthesia. Participants were allocated equally to four groups: 5 mL of 4% lignocaine nebulization, 5 mL of 0.25% ropivacaine nebulization, 10% lignocaine spray, or 5 mL saline nebulization. Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and oxygen saturation were recorded at baseline and after induction, immediately after intubation, and at 1, 3 and 5 min.

**Results:** Among the participants who completed the study, baseline age, sex, weight, ASA physical status and Mallampati class were comparable. Heart rate did not differ significantly between groups at any measured time. Between -group differences were observed for systolic, diastolic and mean arterial pressure at 3 and 5 min after intubation, with the lowest values in the 10% lignocaine spray group and the highest values in the saline group. At 3 min, mean SBP was 110.47 mmHg in the spray group versus 120.83 mmHg in the saline group; at 5 min the corresponding values were 111.90 and 123.80 mmHg. Similar patterns were observed for DBP and MAP. Oxygen saturation remained clinically stable at 99–100%. No recorded complications occurred; none of the patients had bronchospasm or laryngospasm.

**Conclusion:** In this study, 10% lignocaine spray was associated with the smallest post-intubation blood-pressure response, particularly compared with saline nebulization. Nebulized 4% lignocaine and 0.25% ropivacaine also showed acceptable haemodynamic profiles but did not demonstrate the same degree of blood-pressure attenuation. Because baseline blood-pressure values differed between groups, the magnitude of treatment effect should be interpreted cautiously and confirmed in larger studies using adjusted repeated-measures analyses.

**Keywords:** Laryngoscopy; Tracheal intubation; Lignocaine; Ropivacaine; Hemodynamics.

## INTRODUCTION

Laryngoscopy and tracheal intubation are essential components of conventional general anaesthesia, but manipulation of the upper airway produces a characteristic transient cardiovascular response. Stimulation of the epipharyngeal and laryngopharyngeal mucosa activates afferent pathways and produces sympathetic discharge, with release of catecholamines and consequent increases in heart rate and arterial pressure. [1-3] The response usually develops rapidly after airway instrumentation and is particularly relevant in patients in whom abrupt increases in myocardial oxygen demand or arterial pressure may be poorly tolerated.[4]

Several pharmacological and non-pharmacological strategies have been investigated to attenuate this response, including deepening anaesthesia, opioids, $\alpha_2$-adrenergic agonists, β-blockers, calcium-channel blockers, vasodilators and local anaesthetics.[4-6] Intravenous lignocaine has long been used for this purpose, while topical airway anaesthesia offers a direct approach by reducing sensory input from the mucosa that is stimulated during laryngoscopy.[7] Lignocaine blocks voltage-gated sodium channels and, when applied to airway mucosa, can reduce airway reflexes and nociceptive afferent transmission.[7-9]

The route of topical administration may influence the distribution of the local anaesthetic. Nebulization can expose a relatively large mucosal surface and may provide both upper- and lower-airway anaesthesia, but part of the administered drug may be lost during aerosolization or remain in the oral cavity. [6,10] In contrast, oropharyngeal spray delivers a concentrated dose directly to the posterior pharyngeal and supraglottic regions that are contacted by the laryngoscope blade. Studies of topical lignocaine have suggested that 10% spray can reduce hemodynamic responses to airway instrumentation. [11-13]

Ropivacaine is an amino-amide local anaesthetic supplied as the S-enantiomer and is widely used for regional and neuraxial anaesthesia. Its use for topical airway anaesthesia has been investigated because of its longer duration and favourable cardiovascular safety profile compared with racemic bupivacaine. [8,14] A randomized trial comparing nebulized 0.25% ropivacaine with lignocaine and saline reported attenuation of intubation and extubation responses with ropivacaine, although results across studies have not been uniform.[15]

The present study was designed to compare three topical local-anaesthetic techniques—5 mL of 4% lignocaine nebulization, 5 mL of 0.25% ropivacaine nebulization and 10% lignocaine oropharyngeal spray—with saline nebulization as control. The primary outcomes were heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure at baseline and after laryngoscopy and tracheal intubation. Oxygen saturation and airway complications were assessed as additional outcomes.

## MATERIALS AND METHODS

**Study design and setting:** The study included adult patients scheduled for elective surgery under general anaesthesia requiring tracheal intubation. Institutional ethics committee approval was obtained and written informed consent was taken from all participants.

**Participants:** Patients aged 18–60 years, with ASA physical status I or II, BMI 18–30 kg/m² and planned elective procedures expected to last approximately 1.5–2 h or longer were eligible. Exclusion criteria were ASA physical status III or above, refusal to participate, known allergy to local anaesthetics, age below 18 or above 60 years, positive COVID-19 reverse-transcription polymerase chain reaction testing, pregnancy, BMI >30 kg/m², long-standing diabetes, risk of gastric aspiration, chronic obstructive pulmonary disease and recent respiratory infection.

**Sample size:** The sample size was calculated with a two-sided α of 0.05 and β of 0.20, giving 80% power. Based on the reference study used in the source protocol, the minimum calculated sample size was 28 per group; this was increased to 30 per group, giving a total sample of 120 participants.[15]

**Randomization and allocation:** Participants were randomly allocated in a 1:1:1:1 ratio using computer-generated numbers concealed in closed envelopes. Thirty participants were allocated to each study group.

**Preoperative preparation:** All patients underwent routine pre-anaesthetic assessment, including airway examination and standard laboratory investigations. Patients were kept nil by mouth for 6 hours. The patients were premedicated with ranitidine 150 mg, metoclopramide 10 mg and alprazolam 0.25 mg on the night before surgery and on the day of surgery. Written informed consent was obtained before the surgery.

**Interventions:** Group A received 5 mL of 4% lignocaine by nebulization; Group B received 5 mL of 0.25% ropivacaine by nebulization; Group C received 10% lignocaine spray; and Group D received 5 mL of normal saline by nebulization. For Group C, 10% lignocaine containing 10 mg per puff was applied to the posterior pharyngeal wall; the protocol describes two applications and a maximum of three puffs. Nebulization was completed 10 min before induction of anaesthesia and spray administration was completed 3 min before induction.

**Anaesthetic technique and monitoring:** After the topical intervention, fentanyl 2 μg/kg intravenously was given 1–2 min before induction. After preoxygenation with 100% oxygen, anaesthesia was induced with propofol 2 mg/kg. Atracurium 0.5 mg/kg was administered after 3 min of ventilation

with 100% oxygen. Tracheal intubation was performed by a laryngoscope using a Macintosh blade with an appropriately sized cuffed endotracheal tube. Anaesthesia was maintained with nitrous oxide 66%, oxygen 33%, sevoflurane 2% and atracurium. At the end of surgery, residual neuromuscular blockade was reversed with neostigmine 0.05 mg/kg and glycopyrrolate 0.01 mg/kg, followed by extubation when adequate consciousness and muscle power were present.

**Outcome assessment:** Heart rate, SBP, DBP, MAP and $SpO_2$ were recorded at baseline, after premedication, after study drug administration, after induction, immediately after laryngoscopy and intubation, and at 1, 3 and 5 min thereafter. Heart rate, SBP, DBP and MAP were additionally recorded during recovery/extubation and in the postoperative ward. The secondary outcomes specified in the protocol were cough, laryngospasm and bronchospasm during laryngoscopy and intubation.

**Statistical analysis:** We have used SPSS and one-way analysis of variance for continuous variables, with $\chi^2$ test or Fisher's exact test for categorical variables. Continuous data were summarized as mean ± standard deviation and categorical data as number and percentage. Statistical significance was set at $P \leq 0.05$. Overall ANOVA P values are reported for the four-group comparisons. Because baseline SBP, DBP and MAP differed between groups, post-intubation values are interpreted cautiously.

## RESULTS

One hundred and twenty participants were randomized, with 30 participants in each group, and all 120 were available for analysis. The master chart therefore supports a complete four-group analysis without recorded losses to follow-up.

The groups were broadly comparable in age, weight, sex, ASA physical status and Mallampati class. Mean age was 33.5±11.2 years in Group A, 33.1±9.8 years in Group B, 34.7±13.6 years in Group C and 36.3±13.4 years in Group D (P=0.726). Mean weight was 58.7±8.4, 60.4±11.5, 62.0±10.4 and 61.7±8.1 kg, respectively (P=0.545). Females comprised 60.0%, 63.3%, 76.7% and 66.7% of the four groups, respectively (P=0.552). ASA I/II distribution was 43.3/56.7%, 66.7/33.3%, 53.3/46.7% and 36.7/63.3%, respectively (P=0.105). Mallampati class distribution was also similar (P=0.919).

Heart rate remained statistically comparable among groups throughout the observation period. Baseline heart rate was 85.5±13.2, 88.6±14.1, 86.4±12.4 and 85.8±13.6 beats/min in Groups A–D, respectively (P=0.804). Immediately after intubation the corresponding values were 85.1±11.0, 85.1±12.9, 81.3±13.0 and 87.5±15.6 beats/min (P=0.345), and at 5 min they were 88.3±8.9, 85.0±10.5, 86.0±11.9 and 85.6±12.7 beats/min (P=0.668).

Systolic, diastolic and mean arterial pressures showed the clearest between-group differences. Baseline SBP, DBP and MAP were already higher in the saline group, with overall P values of 0.044, 0.040 and 0.033, respectively. After intubation, SBP at 3 min was 113.0±12.3, 113.2±13.3, 110.5±9.7 and 120.8±14.2 mmHg in Groups A–D (P=0.011); at 5 min it was 114.2±13.2, 115.4±12.3, 111.9±8.1 and 123.8±15.4 mmHg (P=0.002). DBP at 3 min was 74.8±7.4, 72.9±8.6, 72.9±7.6 and 80.8±12.0 mmHg (P=0.002), and at 5 min was 77.0±7.5, 76.4±7.1, 74.1±7.0 and 83.3±13.1 mmHg (P=0.001). MAP at 3 min was 87.4±8.7, 86.3±9.7, 85.4±7.5 and 94.2±12.5 mmHg (P=0.003), while at 5 min it was 89.2±9.0, 89.1±8.6, 86.7±6.7 and 96.7±13.6 mmHg (P<0.001). The analysis of data identified Group C versus Group D as the principal pairwise difference at 3 and 5 min.

$SpO_2$ remained clinically stable throughout the peri-intubation period. Although the ANOVA was significant at baseline and after premedication/induction, values remained between 99% and 100% at the clinically relevant post-intubation time points. No recorded complication occurred; no bronchospasm or laryngospasm was observed in any group.

**Table 1: Baseline demographic and airway characteristics**

| Variable | Group A(n=30) | Group B(n=30) | Group C(n=30) | Group D(n=30) | P value |
|---|---|---|---|---|---|
| Age, years, mean ± SD | 33.5 ± 11.2 | 33.1 ± 9.8 | 34.7 ± 13.6 | 36.3 ± 13.4 | 0.726 |
| Weight, kg, mean ± SD | 58.7 ± 8.4 | 60.4 ± 11.5 | 62.0 ± 10.4 | 61.7 ± 8.1 | 0.545 |
| Female, n (%) | 18 (60.0) | 19 (63.3) | 23 (76.7) | 20 (66.7) | 0.552 |
| Male, n (%) | 12 (40.0) | 11 (36.7) | 7 (23.3) | 10 (33.3) | |
| ASA I, n (%) | 13 (43.3) | 20 (66.7) | 16 (53.3) | 11 (36.7) | 0.105 |
| ASA II, n (%) | 17 (56.7) | 10 (33.3) | 14 (46.7) | 19 (63.3) | |
| Mallampati I, n (%) | 11 (36.7) | 8 (26.7) | 10 (33.3) | 9 (30.0) | 0.919 |
| Mallampati II, n (%) | 15 (50.0) | 16 (53.3) | 15 (50.0) | 18 (60.0) | |
| Mallampati III, n (%) | 4 (13.3) | 6 (20.0) | 5 (16.7) | 3 (10.0) | |

**Table 2: Heart rate and oxygen saturation during peri-intubation period**

| Time point | Group A HR | Group B HR | Group C HR | Group D HR | P |
|---|---|---|---|---|---|
| Baseline | 85.5 ± 13.2 | 88.6 ± 14.1 | 86.4 ± 12.4 | 85.8 ± 13.6 | 0.804 |
| Immediately after intubation | 85.1 ± 11.0 | 85.1 ± 12.9 | 81.3 ± 13.0 | 87.5 ± 15.6 | 0.345 |
| 1 min | 84.4 ± 11.8 | 86.2 ± 12.6 | 85.7 ± 12.1 | 83.6 ± 13.7 | 0.845 |
| 3 min | 83.7 ± 12.3 | 82.7 ± 11.7 | 82.6 ± 18.7 | 81.0 ± 12.3 | 0.906 |
| 5 min | 88.3 ± 8.9 | 85.0 ± 10.5 | 86.0 ± 11.9 | 85.6 ± 12.7 | 0.668 |
| Recovery/extubation | 90.2 ± 11.5 | 90.0 ± 10.5 | 92.1 ± 7.9 | 94.6 ± 11.6 | 0.300 |
| Postoperative | 89.4 ± 8.3 | 90.5 ± 8.2 | 92.8 ± 9.3 | 89.5 ± 12.6 | 0.493 |

$SpO_2$ remained 99–100% at all clinically relevant post-intubation time points.

**Table 3: Systolic, diastolic and mean arterial pressure during peri-intubation period**

| Time/parameter | Group A | Group B | Group C | Group D | P |
|---|---|---|---|---|---|
| Baseline – SBP | 127.7 ± 18.6 | 131.8 ± 17.1 | 126.6 ± 17.8 | 140.3 ± 26.4 | 0.044 |
| Baseline – DBP | 80.9 ± 9.4 | 84.3 ± 9.5 | 83.2 ± 11.5 | 89.4 ± 15.2 | 0.040 |
| Baseline – MAP | 96.5 ± 11.5 | 100.2 ± 11.2 | 97.7 ± 13.2 | 106.3 ± 18.0 | 0.033 |
| Immediately after intubation – SBP | 113.7 ± 18.2 | 112.0 ± 14.1 | 108.8 ± 13.5 | 114.8 ± 17.1 | 0.496 |
| Immediately after intubation – DBP | 73.5 ± 9.2 | 74.5 ± 11.1 | 72.3 ± 11.7 | 75.5 ± 13.6 | 0.727 |
| Immediately after intubation – MAP | 87.2 ± 11.6 | 87.0 ± 11.6 | 84.5 ± 11.9 | 88.6 ± 14.1 | 0.629 |
| 1 min – SBP | 113.2 ± 12.3 | 110.8 ± 14.3 | 106.7 ± 10.9 | 114.2 ± 15.3 | 0.140 |
| 1 min – DBP | 71.6 ± 13.8 | 72.8 ± 9.9 | 70.6 ± 8.3 | 75.8 ± 13.0 | 0.326 |
| 1 min – MAP | 87.0 ± 8.2 | 85.5 ± 11.0 | 82.6 ± 8.6 | 88.9 ± 13.1 | 0.128 |
| 3 min – SBP | 113.0 ± 12.3 | 113.2 ± 13.3 | 110.5 ± 9.7 | 120.8 ± 14.2 | 0.011 |
| 3 min – DBP | 74.8 ± 7.4 | 72.9 ± 8.6 | 72.9 ± 7.6 | 80.8 ± 12.0 | 0.002 |
| 3 min – MAP | 87.4 ± 8.7 | 86.3 ± 9.7 | 85.4 ± 7.5 | 94.2 ± 12.5 | 0.003 |
| 5 min – SBP | 114.2 ± 13.2 | 115.4 ± 12.3 | 111.9 ± 8.1 | 123.8 ± 15.4 | 0.002 |
| 5 min – DBP | 77.0 ± 7.5 | 76.4 ± 7.1 | 74.1 ± 7.0 | 83.3 ± 13.1 | 0.001 |
| 5 min – MAP | 89.2 ± 9.0 | 89.1 ± 8.6 | 86.7 ± 6.7 | 96.7 ± 13.5 | 0.001 |

Pairwise comparison of Group C versus Group D at 3 min showed mean differences of −10.4 mmHg for SBP (95% CI −16.7 to −4.1; P=0.002), −7.9 mmHg for DBP (95% CI −13.1 to −2.7; P=0.003) and −8.8 mmHg for MAP (95% CI −14.2 to −3.5; P=0.002). At 5 min, corresponding differences were −11.9 mmHg (95% CI −18.3 to −5.5; P<0.001), −9.2 mmHg (95% CI −14.7 to −3.8; P=0.001) and −10.0 mmHg (95% CI −15.5 to −4.4; P<0.001). These pairwise calculations are unadjusted and should be interpreted in light of baseline differences.

**Table 4: Recorded peri-intubation complications**

| Outcome | Group A | Group B | Group C | Group D |
|---|---|---|---|---|
| Any complication recorded in master chart | 0 (0) | 0 (0) | 0 (0) | 0 (0) |
| Bronchospasm | None reported | None reported | None reported | None reported |
| Laryngospasm | None reported | None reported | None reported | None reported |

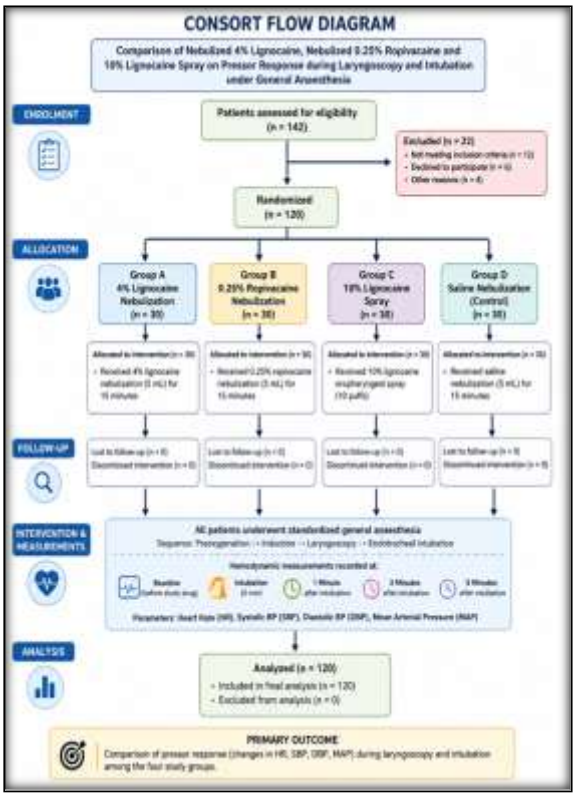


**Figure 1: CONSORT-style participant flow diagram content.**

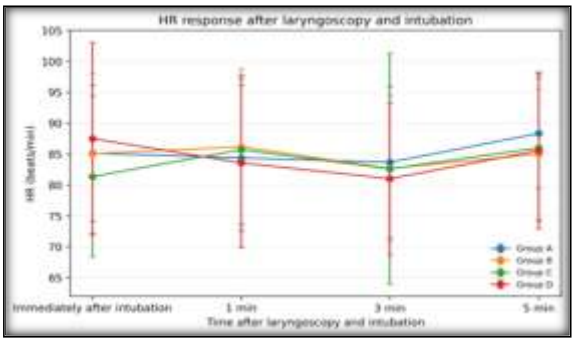


**Figure 2: Heart-rate response after laryngoscopy and tracheal intubation. Values are mean ± SD.**

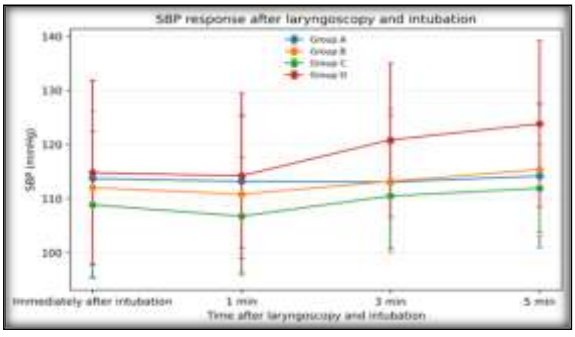


**Figure 3: Systolic blood-pressure response after laryngoscopy and tracheal intubation. Values are mean ± SD.**

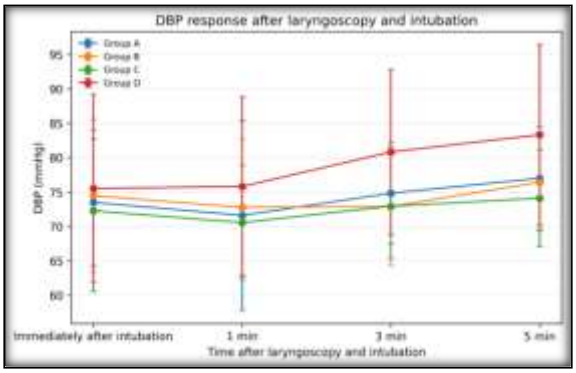


**Figure 4: Diastolic blood-pressure response after laryngoscopy and tracheal intubation.Values are mean ± SD.**

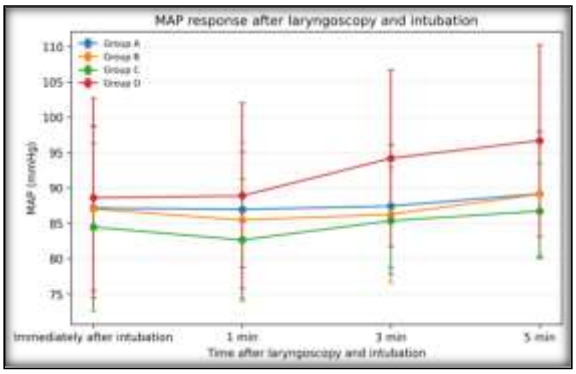


**Figure 5: Mean arterial pressure response after laryngoscopy and tracheal intubation. Values are mean ± SD.**

## DISCUSSION

**Principal findings:** This randomized study compared three topical local-anaesthetic approaches with saline during conventional laryngoscopy and tracheal intubation. The principal finding was that 10% lignocaine spray was associated with the lowest SBP, DBP and MAP values at 3 and 5 min after intubation, whereas saline nebulization had the highest values. Nebulized 4% lignocaine and 0.25% ropivacaine produced intermediate hemodynamic values. Heart rate did not differ significantly between groups at any measured time point. These findings broadly support the hypothesis that targeted topical airway anaesthesia can reduce the hemodynamic response to airway instrumentation, although the present analysis also revealed baseline blood-pressure imbalance that limits causal interpretation.

**Physiological basis:** The pressor response to laryngoscopy and intubation is mediated predominantly by sympathetic activation following stimulation of pharyngeal and laryngeal sensory receptors. [1-3] Lignocaine can reduce this afferent input by blocking sodium channels in sensory nerve fibres. [7,8] Direct topicalization of the regions contacted by the laryngoscope blade therefore provides a plausible mechanism for attenuation of the response. The rapidity of the response also explains why the timing of topical administration is important.

**Why spray may have performed better:** In the present study, the spray was directed to the posterior pharyngeal wall and glossoepiglottic region immediately before induction. This produces a concentrated topical exposure at the sites subjected to mechanical stimulation during direct laryngoscopy. In contrast, nebulization depends on aerosol deposition and patient breathing pattern; some of the dose may be lost to the environment or retained in the mouth. The source thesis itself proposes this explanation for the relatively greater hemodynamic changes seen with nebulized lignocaine. Similar observations have been reported by Kumar et al., in whose randomized trial nebulized lignocaine was less effective than intravenous fentanyl for attenuating the hemodynamic response.[6]

**Comparison with previous evidence:** The present findings are consistent with reports supporting topical lignocaine spray. Jain et al. found that 10% lignocaine spray administered before induction attenuated the pressor response in controlled hypertensive patients.[11] Lee and Park also reported lower arterial-pressure responses with 10% lidocaine spray during suspension laryngoscopy.[10] Mahajan et al. compared intravenous lignocaine with oropharyngeal 10% xylocaine spray and found significant differences in hemodynamic parameters after intubation.[12] These findings are directionally consistent with the lower blood-pressure values observed in Group C in the present study.

**Evidence for nebulized lignocaine and ropivacaine:** Evidence concerning nebulized lignocaine is mixed. Kumar et al. reported that nebulized lignocaine was the least effective of their study interventions for blunting intubation responses.[6] Jokar et al. found that inhaled nebulized lignocaine could attenuate hemodynamic changes compared with a control group, although their clinical setting and dosing differed from the present study.[13] The difference may reflect dose, deposition, anaesthetic technique and the population studied. Ropivacaine has also shown potential as a topical airway anaesthetic. Thangavelu et al. directly compared 0.25% ropivacaine, 2% lignocaine and saline nebulization and found beneficial effects of ropivacaine on intubation and extubation responses.[15] The current study similarly showed that 0.25% ropivacaine nebulization produced acceptable hemodynamic values, although it did not outperform 10% spray.

**Heart rate and oxygenation:** The absence of a significant difference in heart rate is noteworthy. Heart rate is influenced by multiple factors during induction, including fentanyl, propofol, depth of anaesthesia, volatile-agent concentration and individual sympathetic responsiveness. The standardized fentanyl and induction protocol may therefore have reduced between-group differences. Oxygen saturation remained 99–100% after intubation, indicating that none of the topical techniques produced clinically evident deterioration in oxygenation in this cohort.

**Safety:** None of the participants developed either bronchospasm or laryngospasm. This is reassuring but should not be interpreted as evidence that these events cannot occur. Topical local anaesthetics can be systemically absorbed, particularly from highly vascular airway mucosa, and dose and delivery technique remain important determinants of safety. The present study did not measure plasma local anaesthetic concentrations, which was also identified as a limitation in the original work.

**Clinical relevance:** From a practical perspective, 10% lignocaine spray is inexpensive, simple and readily applicable to the mucosa immediately before induction. The study suggests that it may be a useful adjunct when a transient pressor response is undesirable.

## CONCLUSION

Among adults undergoing elective surgery under general anaesthesia, 10% lignocaine spray was associated with the lowest observed SBP, DBP and MAP values at 3 and 5 min after laryngoscopy and tracheal intubation, particularly when compared with saline nebulization. Nebulized 4% lignocaine and 0.25% ropivacaine also provided clinically acceptable hemodynamic profiles. However, because baseline arterial pressures differed between randomized groups and the source analysis did not include baseline-adjusted repeated-measures modelling, the findings should be interpreted as hypothesis-generating rather than definitive evidence of superiority. No bronchospasm or laryngospasm was reported.

**Limitations**

- The study was conducted at a single centre with 30 participants per group, limiting precision and generalizability.
- The study included only ASA I–II adults aged 18–60 years with specified BMI and respiratory-risk exclusions; therefore, the findings cannot be directly extrapolated to high risk cardiovascular, respiratory or difficult airway populations.
- Only one concentration of nebulized ropivacaine (0.25%) was studied, so the results cannot be extrapolated to higher concentrations.
- Plasma concentrations of lignocaine or ropivacaine were not measured and aerosol drug deposition or wastage was not quantified.

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