Section: Anaesthesiology and Critical Care

Original Research Article

COMPARATIVE STUDY OF NEBULISED DEXMEDETOMIDINE VERSUS NEBULISED 4% LIGNOCAINE IN ATTENUATING THE HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION: A RANDOMISED CONTROLLED STUDY

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ABSTRACT

Background: Direct laryngoscopy and endotracheal intubation produce a transient but clinically important sympathetic stress response characterized by tachycardia and increases in systolic, diastolic and mean arterial blood pressure. Although several pharmacological methods have been used to attenuate this response, nebulised drug administration offers a simple, non-invasive route. Dexmedetomidine, an α2-adrenergic agonist, provides sympatholytic and sedative effects, whereas lignocaine produces topical airway anaesthesia and reduces afferent airway stimulation. This study compared nebulised dexmedetomidine with nebulised 4% lignocaine for attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation. **Materials and Methods:** This prospective, randomized, double-blind comparative study included 50 adult patients aged 18–60 years with American Society of Anesthesiologists physical status I or II undergoing elective surgery under general anaesthesia. Patients were randomly allocated into two equal groups. Group D received nebulised dexmedetomidine 1 µg/kg diluted in 5 mL of 0.9% saline, while Group L received nebulised 4% lignocaine 3 mg/kg, 15 minutes before induction. Heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were recorded before and after nebulisation, before laryngoscopy, and at 1, 5, 10 and 15 minutes after intubation. Post-extubation haemodynamic parameters and recovery were assessed using the Observer's Assessment of Alertness/Sedation Scale. **Results:** Baseline demographic characteristics were comparable between groups. Heart rate was significantly lower in Group D before laryngoscopy and at 1, 5 and 10 minutes after intubation. Systolic blood pressure, diastolic blood pressure and mean arterial pressure were also significantly lower in Group D during the period of laryngoscopy and intubation. The differences were most prominent for MAP, which remained significantly lower in Group D through 15 minutes after intubation. Haemodynamic parameters became comparable between groups by 30 minutes after extubation. Group D showed significantly deeper sedation immediately after extubation and at 30 minutes, whereas Group L demonstrated faster recovery of alertness. **Conclusion:** Nebulised dexmedetomidine 1 µg/kg provided superior attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation compared with nebulised 4% lignocaine. Its sympatholytic effect resulted in better control of heart rate and blood pressure, although lignocaine was associated with faster postoperative recovery of alertness. Nebulised dexmedetomidine may therefore be a useful pre-induction strategy when peri-intubation haemodynamic stability is a priority.

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential components of general anaesthesia but constitute potent noxious stimuli capable of producing transient sympathetic activation. The resulting cardiovascular response commonly manifests as tachycardia and increases in systolic, diastolic and mean arterial pressure. Although these changes are usually short-lived in healthy individuals, exaggerated haemodynamic responses may be clinically important in patients with cardiovascular, cerebrovascular or other significant comorbidities. Recent clinical trials continue to demonstrate the importance of attenuating this response during airway manipulation.[1,2]

Dexmedetomidine is a highly selective α2-adrenergic receptor agonist with sedative, analgesic and sympatholytic properties. Its central sympatholytic action reduces catecholamine-mediated cardiovascular responses to airway stimulation. Conventional intravenous administration can attenuate the pressor response but may be associated with bradycardia and hypotension. Consequently, alternative routes such as nebulisation have been investigated to provide effective drug delivery while potentially minimizing abrupt systemic cardiovascular effects.[2,3]

Several randomized studies have demonstrated that preoperative nebulised dexmedetomidine can attenuate increases in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure following laryngoscopy and intubation.[3,4] A systematic review and meta-analysis of randomized trials published in 2023 also reported beneficial effects of nebulised dexmedetomidine on post-intubation haemodynamic parameters, although the authors emphasized limitations in the available evidence.[5]

Lignocaine is a commonly used topical local anaesthetic that reduces airway afferent stimulation by blocking voltage-gated sodium channels. Nebulised lignocaine provides topical anaesthesia extending from the oral cavity to the laryngeal and tracheal mucosa and has been investigated as a non-invasive method for reducing the cardiovascular response to airway instrumentation. Studies have demonstrated that nebulised lignocaine can attenuate increases in heart rate and blood pressure associated with laryngoscopy and intubation.[6]

Recent evidence has increasingly focused on direct comparisons between nebulised dexmedetomidine and lignocaine. A 2024 randomized comparative study specifically evaluated these two agents and reported that both could attenuate the haemodynamic response, with dexmedetomidine demonstrating favourable haemodynamic control.[7] However, differences in dosage, timing, patient population and anaesthetic technique make further comparative studies relevant.

The present study was therefore undertaken to compare nebulised dexmedetomidine 1 µg/kg with nebulised 4% lignocaine 3 mg/kg in adult patients undergoing elective surgery under general anaesthesia, with particular emphasis on heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure and postoperative recovery.

Aim and Objectives

To compare the efficacy of nebulised dexmedetomidine and nebulised 4% lignocaine in attenuating changes in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure following laryngoscopy and endotracheal intubation.

MATERIALS AND METHODS

Study design

This was a prospective, randomized, double-blind comparative study.

Study setting

The study was conducted in the Main General Surgery Operating Room, Rajiv Gandhi Government General Hospital, Institute of Anaesthesiology and Critical Care, Madras Medical College, Chennai. The study period was January 2025 to August 2025.

Study population

Adult patients aged 18–60 years belonging to ASA physical status I or II and scheduled for elective surgery under general anaesthesia with endotracheal intubation were considered eligible.

Inclusion criteria

- Age 18–60 years.
- ASA physical status I or II.
- Elective surgery requiring general anaesthesia and tracheal intubation.
- Written informed consent.

Exclusion criteria

Patients who declined participation, ASA physical status III or above, known hypersensitivity to dexmedetomidine or lignocaine, surgery requiring prone positioning, procedures involving the oral cavity, nasopharynx, larynx or neck, and patients with modified Mallampati score >III were excluded.

Sample size

The sample size was determined with reference to previously published studies evaluating the propofol-sparing effect of preoperative nebulised dexmedetomidine during induction of general anaesthesia. Suryawanshi et al. reported a significantly lower propofol requirement in patients receiving nebulised dexmedetomidine compared with controls (73.0 ± 19.509 mg vs. 105.2 ± 14.741 mg; $p < 0.001$).[8] Considering a 95% confidence level and 80% power, and allowing for a clinically meaningful difference in the study outcome, a sample size of 25 patients per group was selected. Thus, the total sample size was 50 patients.

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Randomization and blinding

Patients were randomized by computer-generated allocation using sealed envelopes into two groups of 25 patients each.

Group D

Patients received nebulised dexmedetomidine 1 µg/kg diluted to 5 mL with 0.9% saline, 15 minutes before induction.

Group L

Patients received nebulised 4% lignocaine at a dose of 3 mg/kg, 15 minutes before induction.

These interventions and their timing correspond to the study protocol in the dissertation.

Anaesthetic technique

After arrival in the operating room, intravenous access was established and standard ASA monitoring including ECG, non-invasive blood pressure and pulse oximetry was applied. Baseline HR, SBP, DBP, MAP and SpO_2 were recorded.

Following nebulisation, haemodynamic parameters were reassessed. Patients received glycopyrrolate 2 mgkg iv stat fentanyl 2 µg/kg and propofol 2 mg/kg following three minutes of preoxygenation. Atracurium 0.5 mg/kg was administered to facilitate tracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen in a 2:2 L mixture with sevoflurane at approximately MAC 1.0 and intermittent atracurium.

Data Collection

After obtaining Institutional Ethics Committee approval and written informed consent, eligible patients undergoing elective surgery under general anaesthesia were enrolled. Demographic and baseline clinical details, including age, sex, ASA physical status and relevant preoperative findings, were recorded using a structured data collection proforma. Patients were randomized into two groups of 25 each. Group D received nebulised dexmedetomidine 1 µg/kg diluted to 5 mL with 0.9% saline, while Group L received nebulised 4% lignocaine 3 mg/kg. Both interventions were administered 15 minutes before induction of anaesthesia.

Standard ASA monitoring was instituted, and baseline heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and oxygen saturation (SpO_2) were recorded before nebulisation. The same parameters were subsequently documented after nebulisation, before laryngoscopy, and at 1, 5, 10 and 15 minutes following endotracheal intubation. Haemodynamic parameters were also recorded immediately after extubation and at 30 minutes and 1 hour postoperatively.

The Observer's Assessment of Alertness/Sedation Scale (OASS) was used to assess postoperative recovery immediately after extubation, at 30 minutes and at 1 hour. Any episodes of bradycardia, hypotension, hypertension, desaturation or other adverse events were recorded. All observations were entered into the study proforma and subsequently transferred to Microsoft Excel and SPSS version 28 for statistical analysis.

Statistical analysis

Data were analysed using Microsoft Excel and SPSS version 28. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequencies and percentages. Between-group comparisons were performed using appropriate parametric and non-parametric tests, including independent-samples t-test, chi-square test and Friedman test where appropriate. A p value <0.05 was considered statistically significant.

Ethical approval

Institutional Ethics Committee approval was obtained before commencement of the study. The thesis records IEC registration numbers ECR/270/Inst/TN/2013/RR-20 and EC/New/INST/2021/1618, with approval dated 17.08.2025. Written informed consent was obtained from all participants.

RESULTS

A total of 50 patients were included, with 25 patients in each group.

Table 1: Comparison of demographic and baseline characteristics

Parameter	Group D Mean ± SD / n (%)	Group L Mean ± SD / n (%)	p value
Age (years)	39.32 ± 10.94	40.16 ± 12.10	0.790
Height (cm)	160.80 ± 4.12	161.00 ± 3.57	0.850
Weight (kg)	64.80 ± 6.70	64.56 ± 6.22	0.890
BMI (kg/m^2)	25.06 ± 2.38	24.92 ± 2.47	0.840
Male	19 (76%)	16 (64%)	0.530
Female	6 (24%)	9 (36%)	
ASA I	17 (68%)	16 (64%)	1.000
ASA II	8 (32%)	9 (36%)	

There were no statistically significant differences in age, height, weight, BMI, sex distribution or ASA physical status between the groups, indicating adequate baseline comparability. [Table 1]

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Table 2: Comparison of heart rate at different time points

Time point	Group D Mean ± SD	Group L Mean ± SD	p value
Pre-nebulisation	80.84 ± 4.09	81.32 ± 3.39	0.690
Post-nebulisation	81.76 ± 4.28	80.16 ± 3.61	0.280
Before laryngoscopy	81.24 ± 4.34	84.80 ± 3.04	**0.004***
1 min post-intubation	81.04 ± 4.05	84.76 ± 3.64	**0.003***
5 min post-intubation	80.44 ± 4.24	84.92 ± 3.39	**0.001***
10 min post-intubation	80.76 ± 4.35	84.60 ± 3.67	**0.006***
15 min post-intubation	80.60 ± 4.04	81.16 ± 3.90	0.747
Post-extubation	81.92 ± 4.17	80.92 ± 3.70	0.495
30 min post-extubation	80.68 ± 3.79	81.08 ± 3.72	0.364
1 h post-extubation	82.92 ± 3.26	81.52 ± 4.20	0.264

p value <0.05 significant using Student T Test

Heart rate was comparable at baseline and after nebulisation. However, Group D demonstrated significantly lower heart rates before laryngoscopy and at 1, 5 and 10 minutes following intubation. By 15 minutes, the difference was no longer statistically significant. [Table 2; Figure 1]

Table 3: Comparison of systolic blood pressure at different time points

Time point	Group D Mean ± SD	Group L Mean ± SD	p value
Pre-nebulisation	124.32 ± 5.12	125.20 ± 7.43	0.670
Post-nebulisation	119.12 ± 5.55	120.60 ± 7.50	0.430
Before laryngoscopy	124.12 ± 4.39	135.04 ± 7.45	**<0.001**
1 min post-intubation	122.92 ± 5.16	129.84 ± 7.51	**0.003**
5 min post-intubation	119.96 ± 5.42	129.16 ± 7.36	**<0.001**
10 min post-intubation	121.04 ± 4.76	129.16 ± 7.50	**0.001**
15 min post-intubation	120.16 ± 5.14	130.36 ± 7.66	**<0.001**
Post-extubation	124.16 ± 5.31	128.60 ± 7.57	**0.035**
30 min post-extubation	125.24 ± 5.49	125.04 ± 7.29	0.917
1 h post-extubation	124.88 ± 5.64	125.48 ± 7.47	0.821

p value <0.05 significant using Student T Test

SBP was comparable before and after nebulisation. Group L subsequently demonstrated significantly higher SBP before laryngoscopy and through 15 minutes after intubation. The difference diminished following extubation and disappeared by 30 minutes. [Table 3; Figure 2]

Table 4: Comparison of diastolic blood pressure at different time points

Time point	Group D Mean ± SD	Group L Mean ± SD	p value
Pre-nebulisation	79.60 ± 6.56	79.96 ± 8.58	0.846
Post-nebulisation	74.92 ± 6.23	75.68 ± 7.31	0.640
Before laryngoscopy	79.28 ± 5.45	90.48 ± 7.96	**<0.001**
1 min post-intubation	77.72 ± 6.15	84.00 ± 8.19	**0.012**
5 min post-intubation	74.40 ± 6.15	84.00 ± 7.47	**<0.001**
10 min post-intubation	77.40 ± 5.92	84.16 ± 7.68	**0.003**
15 min post-intubation	74.48 ± 5.24	85.68 ± 9.10	**<0.001**
Post-extubation	77.84 ± 6.09	84.20 ± 8.38	**0.017**
30 min post-extubation	79.48 ± 7.21	80.32 ± 7.74	0.735
1 h post-extubation	80.24 ± 5.41	81.12 ± 7.42	0.643

p value <0.05 significant using Student T Test

DBP remained significantly lower in Group D from the pre-laryngoscopy period through 15 minutes after intubation and immediately after extubation. The groups became comparable at 30 minutes and 1 hour after extubation. [Table 4, Figure 3]

Table 5: Comparison of mean arterial pressure at different time points

Time point	Group D Mean ± SD	Group L Mean ± SD	p value
Pre-nebulisation	94.51 ± 5.88	95.04 ± 8.05	0.810
Post-nebulisation	89.65 ± 5.70	90.65 ± 7.22	0.640
Before laryngoscopy	94.23 ± 4.83	105.33 ± 7.69	**<0.001**
1 min post-intubation	92.79 ± 5.64	99.28 ± 7.83	**0.002**
5 min post-intubation	89.59 ± 5.71	99.05 ± 7.28	**<0.001**
10 min post-intubation	91.95 ± 5.39	99.16 ± 7.42	**0.001**
15 min post-intubation	89.71 ± 5.02	100.57 ± 8.51	**<0.001**
Post-extubation	93.28 ± 5.67	99.00 ± 7.96	**0.003**
30 min post-extubation	94.73 ± 6.54	95.23 ± 7.39	0.800
1 h post-extubation	95.12 ± 5.27	95.91 ± 7.30	0.690

p value <0.05 significant using Student T Test

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MAP was comparable between groups before and after nebulisation. A marked and statistically significant difference developed before laryngoscopy and persisted throughout the first 15 minutes after intubation. Group D maintained consistently lower MAP values, suggesting superior attenuation of the pressor response. [Table 5; Figure 4]

Table 6: Comparison of postoperative OASS scores

Time point	Group D Mean ± SD	Group L Mean ± SD	p value
Immediately post-extubation	2.56 ± 0.51	4.48 ± 0.51	**<0.001**
30 min post-extubation	3.44 ± 0.51	4.80 ± 0.41	**<0.001**
1 h post-extubation	4.48 ± 0.51	4.96 ± 0.20	**0.001**

p value <0.05 significant using Student T Test

Group D demonstrated significantly lower OASS scores immediately after extubation and at 30 minutes, indicating deeper residual sedation. Group L had significantly higher scores and therefore faster recovery of alertness. At 1 hour, both groups approached full alertness, although the difference remained statistically significant. [Table 6]

No episodes of Bradycardia,hypotension , hypertension , desaturation or other adverse events were recorded

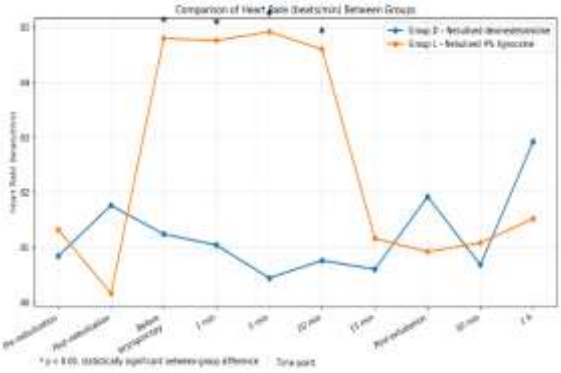

Figure 1: Comparison of heart rate at different peri-intubation and postoperative time points between Group D and Group L

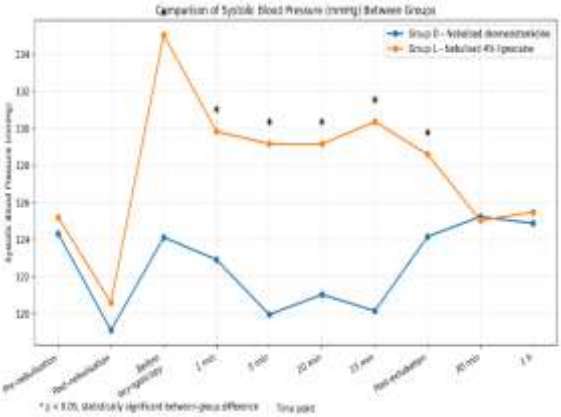

Figure 2: Comparison of systolic blood pressure at different peri-intubation and postoperative time points between Group D and Group L

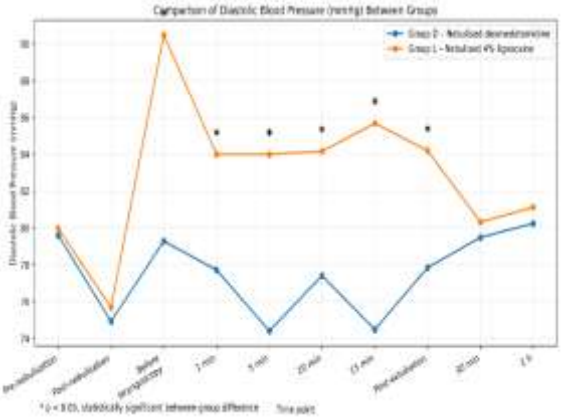

Figure 3: Comparison of diastolic blood pressure at different peri-intubation and postoperative time points between Group D and Group L

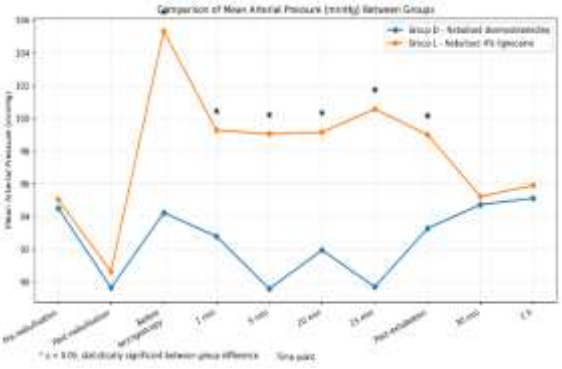

Figure 4: Comparison of mean arterial pressure at different peri-intubation and postoperative time points between Group D and Group L

DISCUSSION

The present prospective randomized comparative study evaluated the effects of nebulised dexmedetomidine and nebulised 4% lignocaine on the haemodynamic response to laryngoscopy and endotracheal intubation. Fifty ASA I and II patients were randomized equally into two groups. The principal finding was that nebulised dexmedetomidine provided significantly better control of HR, SBP, DBP and MAP during the peri-intubation period, whereas nebulised lignocaine was associated with faster recovery of alertness after extubation.

Laryngoscopy and tracheal intubation produce a predictable sympathetic response due to stimulation of the upper airway and laryngeal structures. In the

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present study, baseline haemodynamic characteristics were comparable between groups, indicating adequate randomization. The significant differences developed predominantly around laryngoscopy and intubation, supporting an intervention-related effect.

Heart rate

Heart rate was significantly lower in Group D before laryngoscopy and at 1, 5 and 10 minutes after intubation. This finding is consistent with the sympatholytic properties of dexmedetomidine. Misra et al. demonstrated a significantly attenuated increase in HR following laryngoscopy after nebulised dexmedetomidine administration.[9] Similarly, Ghodki et al. reported effective attenuation of the haemodynamic response after preoperative nebulised dexmedetomidine.[4]

The findings of the present study are also consistent with the systematic review and meta-analysis by Gupta et al., which demonstrated reductions in post-intubation HR following nebulised dexmedetomidine.[5] A prospective randomized study by Srivastava et al. also reported effective attenuation of the HR response following nebulised dexmedetomidine.[10] These findings support the role of nebulised dexmedetomidine in controlling the chronotropic component of the intubation response.

Systolic blood pressure

The most prominent SBP differences occurred from the pre-laryngoscopy period through 15 minutes following intubation. Group L showed higher SBP values, whereas Group D maintained values closer to baseline. This suggests that dexmedetomidine provided more sustained attenuation of the pressor response.

Ghodki et al. reported significant attenuation of SBP following nebulised dexmedetomidine compared with control.[4] Another prospective randomized study by Nair et al. demonstrated that nebulised dexmedetomidine significantly reduced the rise in SBP following laryngoscopy and intubation.[11] These observations are consistent with the present findings.

Nebulised lignocaine also has a role in reducing airway-related cardiovascular stimulation. Kumar et al. evaluated 4% lignocaine nebulization at 3 mg/kg and found that lignocaine alone attenuated the haemodynamic response, although it was less effective than fentanyl-based strategies.[12] Similarly, Jokar et al. demonstrated an effect of inhaled nebulised lignocaine on haemodynamic responses during endotracheal intubation.[6]

Diastolic blood pressure and mean arterial pressure

The differences in DBP and MAP were particularly prominent in the present study. Group D consistently demonstrated lower values than Group L from before laryngoscopy through 15 minutes after intubation. At 15 minutes, MAP was 89.71 ± 5.02 mmHg in Group D compared with 100.57 ± 8.51 mmHg in Group L ($p<0.001$).

These findings are clinically relevant because MAP represents an integrated measure of arterial pressure and is an important determinant of organ perfusion. Avoidance of abrupt peri-intubation increases may be particularly beneficial in patients vulnerable to myocardial or cerebrovascular complications.

Previous randomized studies have demonstrated similar effects of nebulised dexmedetomidine on DBP and MAP. Ghodki et al. observed significantly lower DBP and MAP following airway manipulation in patients receiving nebulised dexmedetomidine.[4] Kaur et al. also reported attenuation of MAP and DBP responses following dexmedetomidine administration.[13]

Evidence concerning nebulised lignocaine is somewhat variable. Kumar et al. reported that nebulised lignocaine was less effective than fentanyl in suppressing the haemodynamic response.[12] However, other studies have demonstrated beneficial attenuation of cardiovascular responses with topical or nebulised lignocaine.[14,15] Differences in dose, route of administration, timing and patient population may account for the variability between studies.

Comparison with direct comparative evidence

Direct comparison between nebulised dexmedetomidine and nebulised lignocaine remains relatively limited. The 2024 randomized study by Rani et al. directly compared these agents and reported favourable haemodynamic attenuation with nebulised dexmedetomidine.[7] The results of the present study are broadly consistent with these findings and provide further evidence that dexmedetomidine may offer more sustained haemodynamic control.

A possible explanation is the difference in pharmacological mechanisms. Lignocaine primarily reduces afferent sensory stimulation of the airway through sodium-channel blockade, whereas dexmedetomidine produces central sympatholysis through α2-adrenergic receptor activation.[3] This sympatholytic effect may explain the more persistent reduction in HR, SBP, DBP and MAP observed with dexmedetomidine.

Recent evidence also supports the efficacy of nebulised lignocaine itself. A 2025 randomized study comparing nebulised lignocaine with lignocaine spray found that nebulised lignocaine significantly reduced HR, SBP and DBP at selected post-intubation time points.[15] Thus, lignocaine remains an effective airway-directed intervention, although the present findings suggest that dexmedetomidine may provide greater overall haemodynamic stability.

Post-extubation sedation and recovery

An important secondary finding was the difference in postoperative alertness. Group D had significantly lower OASS scores immediately after extubation and at 30 minutes, indicating greater residual sedation. Group L had higher scores and therefore faster recovery of alertness. By 1 hour, both groups approached full alertness, although the difference remained statistically significant.

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This finding is pharmacologically plausible because dexmedetomidine produces sedation in addition to sympatholysis. Its sedative effect is mediated predominantly through α2-adrenergic receptors and is generally characterized by preserved respiratory drive.[3,16] The residual sedative effect observed in the present study may therefore represent an expected consequence of dexmedetomidine administration rather than an adverse effect.

Previous studies of nebulised dexmedetomidine have also demonstrated clinically useful sedative effects. Studies evaluating preoperative nebulised dexmedetomidine have reported satisfactory sedation and acceptable perioperative haemodynamic stability.[17,18] However, the greater residual sedation should be considered when rapid postoperative neurological assessment or early discharge from the recovery area is required.

Clinical implications

The findings suggest that nebulised dexmedetomidine may be particularly useful when prevention of peri-intubation tachycardia and hypertension is the principal objective. This may be clinically relevant in patients in whom abrupt cardiovascular changes are undesirable. However, the greater early postoperative sedation associated with dexmedetomidine should be considered when rapid recovery and immediate neurological assessment are priorities.

The choice between the two agents may therefore depend on the primary clinical objective. Dexmedetomidine appears advantageous when haemodynamic stability is prioritized, whereas lignocaine may be preferable when a shorter sedative effect and faster postoperative alertness are desired.

Strengths and limitations

The principal strengths of the study include randomized allocation, double blinding, standardized anaesthetic management, equal group sizes and serial assessment of multiple haemodynamic variables. The study also evaluated postoperative recovery rather than restricting assessment to the immediate intubation period.

The major limitation is the relatively small sample size of 50 patients from a single centre. Only ASA I and II patients were included, limiting extrapolation to patients with significant cardiovascular disease. The study also evaluated only one dose of each drug and did not include a placebo control group. Larger multicentre randomized controlled trials involving higher-risk populations and different doses are required to establish the optimal role of nebulised dexmedetomidine and lignocaine.

CONCLUSION

Nebulised dexmedetomidine 1 µg/kg was more effective than nebulised 4% lignocaine 3 mg/kg in attenuating the haemodynamic response to laryngoscopy and endotracheal intubation in adult ASA I and II patients undergoing elective surgery. Dexmedetomidine produced significantly better control of heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure during the critical peri-intubation period.

However, this improved haemodynamic control was accompanied by deeper early postoperative sedation and slower recovery of alertness compared with nebulised lignocaine. Nebulised lignocaine, although less effective in controlling the haemodynamic response, was associated with faster recovery.

Thus, nebulised dexmedetomidine may be preferred when peri-intubation cardiovascular stability is the primary concern, while nebulised lignocaine may remain useful when rapid postoperative recovery is particularly important.

Declarations

Ethical approval

Institutional Ethics Committee approval was obtained before commencement of the study.

Informed consent

Written informed consent was obtained from all participants.

Funding

No external funding was reported for the study.

Conflict of interest

The authors declare no conflict of interest.

Data availability

The data supporting the findings are available from the corresponding author subject to institutional and ethical requirements.

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