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Effect of Intravenous Dexmedetomidine Combined with Nebulised Lignocaine on Haemodynamic Responses to Laryngoscopy and Tracheal Intubation: A Randomized Controlled Trial

Dr Rajesh V Nagmothe[1], Dr Satishkumar Mandve[2], Dr.Tamizhaaruvi.K[3]

[1]Associate Professor and Head, Department of Anaesthesiology, KMSKGMC Chandrapur
[2]Assistant professor, Department of Anaesthesiology, KMSKGMC Chandrapur
[3]Resident, Department of Anaesthesiology, KMSKGMC Chandrapur

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Corresponding Author:

Dr Rajesh V Nagmothe
Associate Professor and Head,
Department of Anaesthesiology,
KMSKGMC Chandrapur

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ABSTRACT

Background: Laryngoscopy and tracheal intubation can provoke clinically significant tachycardia and hypertension. Dexmedetomidine and topical lignocaine may attenuate this response through complementary central and peripheral mechanisms.
Objective: To compare intravenous dexmedetomidine plus nebulised lignocaine with routine intubation practice for attenuation of haemodynamic responses to laryngoscopy and intubation.
Methods: In this prospective randomized controlled trial, 71 adults undergoing elective surgery were allocated to control or combination treatment. The combination group received intravenous dexmedetomidine 1.5 µg/kg and nebulised 4% lignocaine 3 mg/kg before induction. Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded through 30 minutes after intubation. Adverse events were also assessed.
Results: The efficacy population included 70 participants. All post-intubation haemodynamic measurements were significantly lower in the combination group after Holm adjustment (all $p<0.001$). The largest heart-rate difference was 29.86 bpm at 5 minutes, while the greatest systolic, diastolic, and mean arterial-pressure differences occurred at 15 minutes. Adverse events occurred in 13.9% of combination-treated participants and none of the controls; dry mouth was most common.
Conclusion: Intravenous dexmedetomidine with nebulised lignocaine provided strong and sustained attenuation of the haemodynamic response to laryngoscopy and tracheal intubation, with occasional bradycardia and hypotension.

Keywords*: dexmedetomidine; lignocaine; laryngoscopy; tracheal intubation; haemodynamic response.*

INTRODUCTION

Direct laryngoscopy and tracheal intubation are essential components of general anaesthesia but produce a transient sympathoadrenal response caused by mechanical stimulation of the supraglottic and tracheal structures. The resulting catecholamine release may increase heart rate, arterial pressure, myocardial oxygen demand, and the risk of dysrhythmia [1]. Although these changes are usually well tolerated by healthy patients, they may be clinically important in individuals with limited cardiovascular or cerebrovascular reserve. Several pharmacological approaches have therefore been evaluated, including opioids, β-blockers, calcium-channel blockers, local anaesthetics, vasodilators, and α2-adrenergic agonists, but no single intervention consistently provides complete attenuation without adverse effects [2].

Dexmedetomidine is a highly selective α2-adrenergic agonist with sedative, analgesic, anaesthetic-sparing, and sympatholytic properties. By reducing central sympathetic outflow and norepinephrine release, it can limit tachycardia

and hypertension associated with airway manipulation. Clinical studies have demonstrated that pre-induction dexmedetomidine attenuates haemodynamic responses to laryngoscopy and reduces perioperative anaesthetic requirements [3,4]. Its effect is dose dependent, however, and clinically relevant bradycardia, hypotension, and excessive sedation may occur, particularly when higher doses are combined with opioids or other anaesthetic agents [5].

Lignocaine acts through a complementary mechanism. Topical administration reduces afferent stimulation from the airway mucosa and may suppress coughing and cardiovascular reflexes during instrumentation. Nebulisation provides relatively uniform, non-invasive topicalisation of the oropharyngeal, laryngeal, and tracheal mucosa. However, evidence regarding nebulised lignocaine alone is inconsistent; some studies have reported incomplete attenuation of the pressor response compared with systemic agents [6]. This variability may relate to dose, timing, deposition within the airway, and the fact that laryngoscopy itself generates sympathetic stimulation that is not abolished by mucosal anaesthesia alone.

Combining agents that act at different points in the reflex pathway may provide more complete haemodynamic control. Dexmedetomidine primarily suppresses the central sympathetic response, whereas nebulised lignocaine reduces peripheral airway afferent input. Studies of nebulised dexmedetomidine and comparative systemic regimens support the value of α2-agonist-based approaches, while randomized evidence indicates that adding dexmedetomidine to nebulised lignocaine improves control of heart rate and arterial pressure during laryngoscopy and intubation [7–9]. Nevertheless, published protocols vary considerably in drug doses, routes, timing, accompanying anaesthetic agents, and observation periods, and safety remains relevant because enhanced attenuation may be accompanied by bradycardia, hypotension, or dry mouth.

The present randomized controlled trial was therefore undertaken to evaluate whether intravenous dexmedetomidine combined with nebulised lignocaine provides superior attenuation of haemodynamic responses to direct laryngoscopy and endotracheal intubation compared with routine intubation practice in adults undergoing elective surgery under general anaesthesia. Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were assessed serially from baseline through 30 minutes after intubation. Adverse events, including dry mouth, bradycardia, and hypotension, were also evaluated to define the clinical balance between haemodynamic stability and treatment-related risk.

METHODS

Study design and participants

This prospective, single-centre, parallel-group randomized controlled trial was conducted over two years in the Department of Anaesthesiology, Karmavir M. S. Kannamwar Government Medical College and Hospital, Chandrapur, Maharashtra, India. The protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants.

Adults aged 18–60 years with American Society of Anesthesiologists physical status I or II who were scheduled for elective surgery of less than three hours under general anaesthesia with endotracheal intubation were eligible. Exclusion criteria included emergency surgery, anticipated difficult airway, clinically significant cardiac or respiratory disease, use of drugs likely to alter the intubation response, allergy to either study drug, nasal pathology interfering with nebulisation, pregnancy or lactation, and inability to provide consent. Participants were excluded from efficacy analysis if laryngoscopy exceeded 45 seconds or more than two intubation attempts were required.

Randomization and masking

The sample size was calculated using an anticipated standardized effect size of 0.70, two-sided α of 0.05, 80% power, and equal allocation, yielding 34 participants per group; 35 were enrolled in each group to allow for attrition. Participants were assigned in a 1:1 ratio using a computer-generated random sequence with allocation concealed in sequentially numbered, opaque, sealed envelopes.

Intravenous study solutions were prepared in identical unlabelled 100-mL bags by an anaesthesiologist not involved in outcome assessment. The investigator recording haemodynamic variables was blinded to the intravenous allocation; complete masking was not feasible because nebulisation was administered only in the intervention group.

Interventions

The control group received 100 mL of 0.9% saline intravenously over 20 minutes. The combination group received nebulised 4% lignocaine 3 mg/kg, diluted to 5 mL and administered over approximately 10 minutes, together with intravenous dexmedetomidine 1.5 µg/kg diluted to 100 mL and infused over 20 minutes.

After infusion, all participants received ondansetron 4 mg, glycopyrrolate 0.2 mg, midazolam 0.02 mg/kg, and fentanyl 2 µg/kg intravenously. Following preoxygenation, anaesthesia was induced with propofol 2 mg/kg and atracurium 0.5 mg/kg. Direct laryngoscopy with a Macintosh blade was performed by an experienced anaesthesiologist, and tracheal placement was confirmed clinically and by capnography. Anaesthesia was maintained with oxygen in air and isoflurane, with ventilation adjusted to maintain end-tidal carbon dioxide at 35–40 mmHg.

Outcomes

Heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded at baseline, before and during the intervention, before induction, after induction, and at 1, 3, 5, 10, 15, and 30 minutes after intubation. Mean arterial pressure was calculated as:

$$\mathrm{MAP} = \frac{\mathrm{SBP} + 2(\mathrm{DBP})}{3}$$

Safety outcomes included dry mouth, bradycardia, hypotension, and any adverse event. Bradycardia was defined as heart rate below 60 beats/min or a decrease greater than 20% from baseline, and hypotension as a systolic blood pressure decrease greater than 20% from baseline. Symptomatic bradycardia was treated with atropine 0.6 mg, and hypotension with mephentermine 6 mg and crystalloid fluid.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation and categorical variables as n (%). Between-group comparisons used Welch independent-samples *t* tests for continuous outcomes and two-sided Fisher exact tests for categorical outcomes. Post-intubation haemodynamic comparisons were adjusted for multiple testing using the Holm method. Effect estimates were reported with 95% confidence intervals. A two-sided adjusted $p<0.05$ was considered statistically significant. Analyses were performed using IBM SPSS Statistics version 25.0.

RESULTS

Participant disposition and baseline characteristics

Seventy-one participants were enrolled: control n=35 (49.3%) and combination n=36 (50.7%). One participant in the combination group discontinued before post-intubation efficacy assessment. The efficacy population therefore comprised n=70 participants, with control n=35 (50.0%) and combination n=35 (50.0%); all n=71 participants were included in the safety analysis.

Age, sex, and American Society of Anesthesiologists physical status were similarly distributed between groups. Baseline heart rate and systolic blood pressure were higher in the control group, whereas diastolic blood pressure and mean arterial pressure were similar (Table 1).

Table 1. Baseline demographic and haemodynamic characteristics of the efficacy population

Characteristic	Control (n=35)	Combination (n=35)	Test	Statistic	df	p-value
Age, years	39.2 ± 11.44	39.0 ± 11.14	Welch t	0.074	68.0	0.941
Male sex	20 (57.1)	21 (60.0)	Fisher exact	—	—	1.000
ASA physical status I	22 (62.9)	23 (65.7)	Fisher exact	—	—	1.000
Heart rate, bpm	86.06 ± 6.27	80.03 ± 5.93	Welch t	4.132	67.8	<0.001
Systolic BP, mmHg	128.00 ± 6.56	124.06 ± 6.27	Welch t	2.570	67.9	0.012
Diastolic BP, mmHg	84.06 ± 4.70	86.03 ± 4.90	Welch t	-1.718	67.9	0.090
Derived MAP, mmHg	98.70 ± 5.31	98.70 ± 5.21	Welch t	0.000	68.0	1.000

Values are mean ± standard deviation or n (%). Welch t tests compare control minus combination; positive t values indicate higher values in the control group. Fisher's exact test was used for categorical variables. MAP = mean arterial pressure, calculated as (SBP + 2×DBP)/3; SBP = systolic blood pressure; DBP = diastolic blood pressure.

Haemodynamic response

Mean heart rate separated after study-drug administration and remained lower with the combination regimen throughout the post-intubation observation period (Figure 1). At every assessment from 1 to 30 minutes after intubation, heart rate was lower in the combination group after multiplicity adjustment (all Holm-adjusted $p<0.001$; Table 2). The largest difference occurred 5 minutes after intubation: 29.86 bpm (95% confidence interval 26.26 to 33.45; $t(59.5)=16.611$).

Table 2. Post-intubation heart-rate comparisons between study groups

Time after intubation	Control mean (bpm)	Combination mean (bpm)	Mean difference (control - combination)	95% CI	t (df)	Holm-adjusted p-value
1 min	98.06	80.91	17.14	13.77 to 20.51	10.165 (63.8)	<0.001
3 min	101.00	81.89	19.11	15.62 to 22.61	10.936 (63.4)	<0.001
5 min	107.89	78.03	29.86	26.26 to 33.45	16.611 (59.5)	<0.001
10 min	100.09	76.06	24.03	20.71 to 27.35	14.476 (59.6)	<0.001
15 min	98.11	76.06	22.06	18.89 to 25.23	13.912 (60.6)	<0.001
30 min	92.37	75.37	17.00	14.13 to 19.87	11.813 (65.5)	<0.001

Control n=35 and combination n=35. Mean differences are control minus combination; positive values indicate lower heart rate with the combination regimen. Test statistics are from Welch independent-samples t tests. Holm adjustment was applied across the six post-intubation comparisons. CI = confidence interval.

Systolic blood pressure, diastolic blood pressure, and mean arterial pressure were lower with the combination regimen at every post-intubation timepoint (all Holm-adjusted p<0.001; Table 3 and Figure 2). The largest differences for all three pressure outcomes occurred 15 minutes after intubation: 25.14 mmHg for systolic blood pressure, 20.09 mmHg for diastolic blood pressure, and 21.77 mmHg for mean arterial pressure.

Table 3. Post-intubation arterial-pressure comparisons between study groups

Outcome and time	Control mean (mmHg)	Combination mean (mmHg)	Mean difference (control - combination)	95% CI	t (df)	Holm-adjusted p-value
SBP, 1 min	134.06	121.97	12.09	8.71 to 15.46	7.155 (63.9)	<0.001
SBP, 3 min	138.00	120.06	17.94	14.55 to 21.34	10.553 (63.4)	<0.001
SBP, 5 min	138.31	118.03	20.29	17.07 to 23.50	12.596 (64.7)	<0.001
SBP, 10 min	133.94	115.94	18.00	14.82 to 21.18	11.330 (61.9)	<0.001
SBP, 15 min	135.20	110.06	25.14	22.10 to 28.18	16.523 (62.6)	<0.001
SBP, 30 min	133.29	110.14	23.14	20.27 to 26.02	16.072 (64.6)	<0.001
DBP, 1 min	92.06	76.06	16.00	13.74 to 18.26	14.130 (60.8)	<0.001
DBP, 3 min	96.03	76.06	19.97	17.60 to 22.35	16.834 (57.5)	<0.001
DBP, 5 min	94.03	74.09	19.94	17.69 to 22.20	17.717 (57.3)	<0.001
DBP, 10 min	90.03	72.00	18.03	15.92 to 20.14	17.099 (55.8)	<0.001
DBP, 15 min	90.14	70.06	20.09	18.01 to 22.16	19.381 (54.8)	<0.001
DBP, 30 min	80.06	72.03	8.03	6.11 to 9.94	8.385 (59.6)	<0.001
MAP, 1 min	106.06	91.36	14.70	12.06 to 17.33	11.164 (62.2)	<0.001
MAP, 3 min	110.02	90.72	19.30	16.58 to 22.01	14.237 (60.1)	<0.001
MAP, 5 min	108.79	88.73	20.06	17.57 to 22.55	16.109 (62.2)	<0.001
MAP, 10 min	104.67	86.65	18.02	15.56 to 20.48	14.655 (58.4)	<0.001
MAP, 15 min	105.16	83.39	21.77	19.40 to 24.15	18.345 (58.6)	<0.001
MAP, 30 min	97.80	84.73	13.07	10.86 to 15.28	11.817 (62.4)	<0.001

Control n=35 and combination n=35. Mean differences are control minus combination; positive values indicate lower arterial pressure with the combination regimen. Test statistics are from Welch independent-samples t tests. Holm adjustment was applied separately across the six post-intubation comparisons for each outcome. MAP = mean arterial pressure, calculated as (SBP + 2×DBP)/3; SBP = systolic blood pressure; DBP = diastolic blood pressure; CI = confidence interval.

Safety outcomes

Any adverse event occurred in control n=0 (0.0%) and combination n=5 (13.9%) (Fisher's exact p=0.054). Dry mouth was the most frequent event. One participant developed both bradycardia and hypotension and discontinued the intervention (Table 4).

Table 4. Adverse events in the safety population

Event	Control (n=35)	Combination (n=36)	Risk difference, percentage points (95% CI)	Test	p-value
Any adverse event	0 (0.0)	5 (13.9)	13.9 (1.3 to 28.7)	Fisher exact	0.054
Dry mouth	0 (0.0)	4 (11.1)	11.1 (-0.8 to 25.3)	Fisher exact	0.115
Bradycardia	0 (0.0)	1 (2.8)	2.8 (-7.4 to 14.2)	Fisher exact	1.000
Hypotension	0 (0.0)	1 (2.8)	2.8 (-7.4 to 14.2)	Fisher exact	1.000

Values are n (%). Risk differences are combination minus control and are presented with Newcombe-Wilson 95% confidence intervals. Two-sided Fisher's exact tests were used. Bradycardia and hypotension occurred in the same participant.

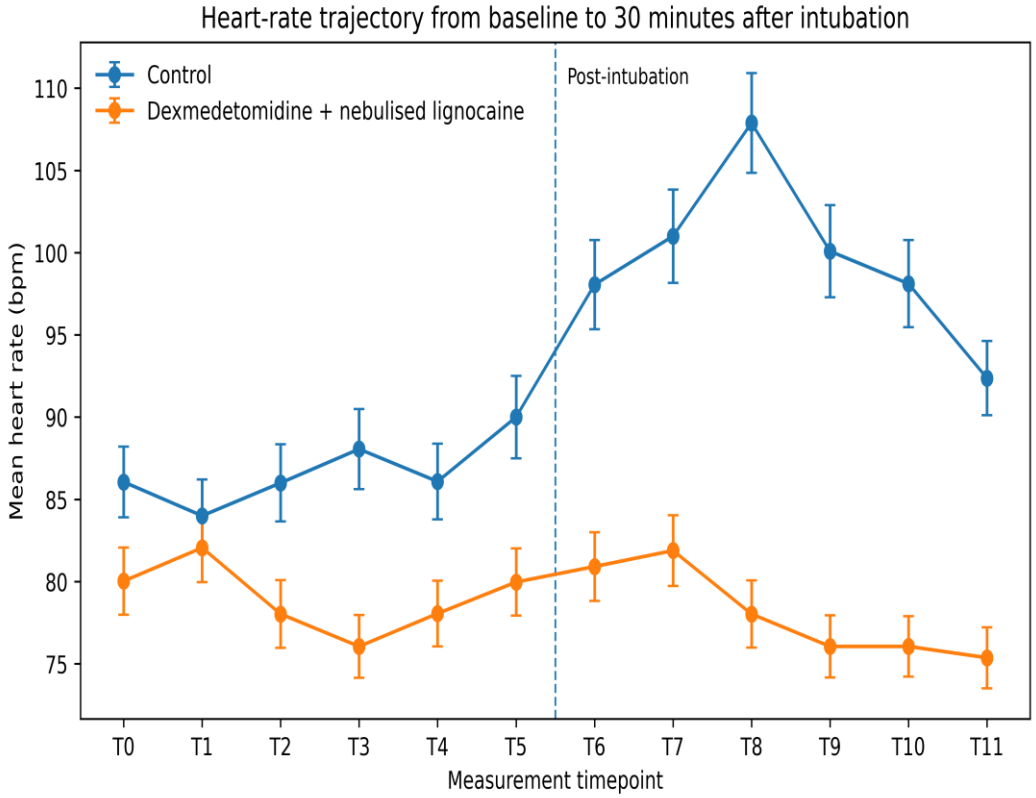

Figure 1. Heart-rate trajectory from baseline to 30 minutes after intubation. Points show group means and error bars show 95% confidence intervals (control n=35; combination n=35). T0, baseline on operating-room arrival; T1, before study intervention; T2, 10 minutes after intervention began; T3, 20 minutes after intravenous infusion began; T4, immediately before induction; T5, after induction and before laryngoscopy; T6-T11, 1, 3, 5, 10, 15, and 30 minutes after intubation, respectively.

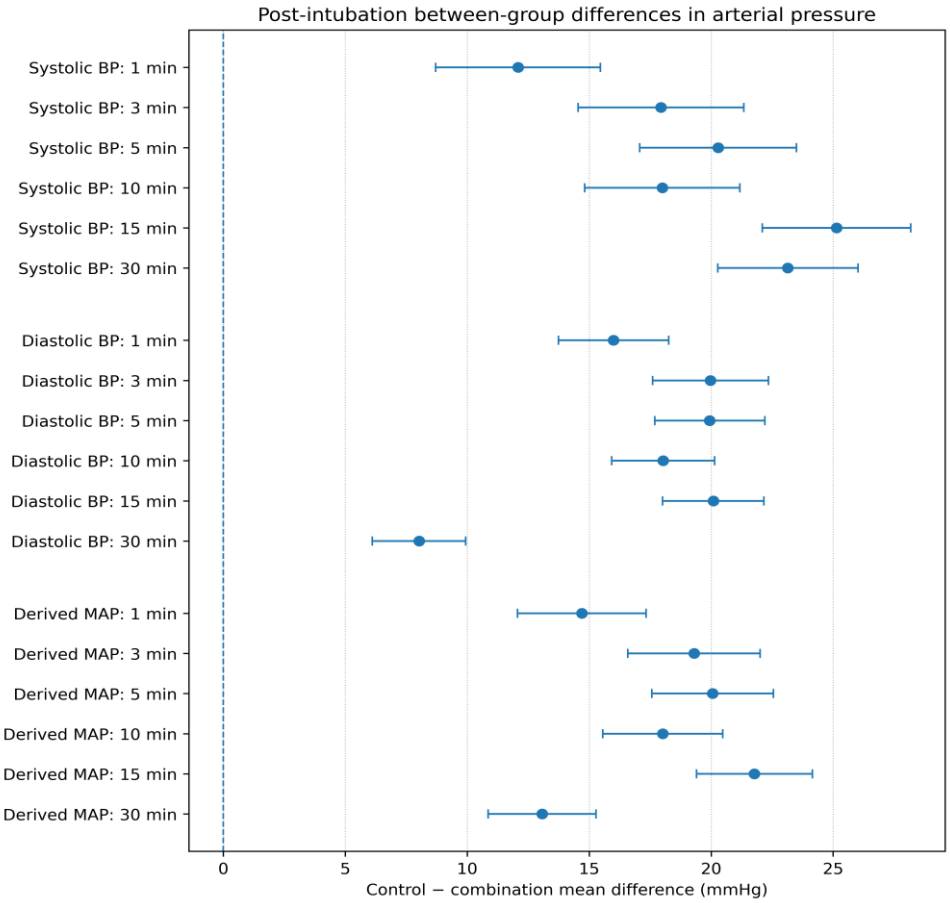

Figure 2. Post-intubation between-group differences in arterial pressure. Points show control-minus-combination mean differences and horizontal bars show 95% confidence intervals (n=35 per group). Positive values indicate lower arterial pressure with the combination regimen. MAP = mean arterial pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure.

DISCUSSION

The present randomized trial found that intravenous dexmedetomidine combined with nebulised lignocaine markedly attenuated the haemodynamic response to laryngoscopy and tracheal intubation. Heart rate and all arterial-pressure measures remained lower in the combination group from 1 to 30 minutes after intubation. The largest between-group heart-rate difference occurred at 5 minutes, while the greatest systolic, diastolic, and mean arterial-pressure differences occurred at 15 minutes. This haemodynamic benefit was accompanied by a modest increase in adverse events, principally dry mouth; one participant developed both bradycardia and hypotension.

The magnitude of attenuation is consistent with dose-ranging evidence for intravenous dexmedetomidine. Sebastian et al. compared 0.5 and 0.75 µg/kg dexmedetomidine with saline and found that both doses reduced heart-rate and blood-pressure responses, while 0.75 µg/kg produced more complete attenuation. Neither bradycardia nor hypotension occurred, oxygen saturation remained above 95%, and sedation was greater with dexmedetomidine than with placebo [10]. Our study used a higher dose of 1.5 µg/kg and demonstrated sustained suppression through 30 minutes, but the occurrence of bradycardia and hypotension in one participant suggests that increasing the dose may improve attenuation at the expense of cardiovascular tolerability.

Route of administration also influences onset and haemodynamic stability. Niyogi et al. compared intravenous and intranasal dexmedetomidine and found that both routes reduced the stress response to intubation, with broadly comparable control of heart rate and mean arterial pressure. Intravenous administration produced greater pre-induction sedation and a faster clinical effect, whereas the intranasal route offered a non-invasive alternative with less abrupt haemodynamic change [11]. The rapid and sustained separation between groups in our trial is compatible with the predictable onset of intravenous dexmedetomidine, while nebulised lignocaine may have simultaneously reduced airway afferent stimulation.

Evidence concerning lignocaine alone has been less consistent. Jokar et al. randomized 192 patients to intravenous lignocaine, nebulised lignocaine, or control treatment. Both lignocaine routes reduced post-intubation haemodynamic

changes compared with control, and nebulised lignocaine provided better overall blood-pressure control than intravenous administration. The study used 4% nebulised lignocaine, assessed serial heart rate and arterial pressures, and reported no major treatment-related complications [12]. In contrast to lignocaine-only protocols, our combination regimen produced large reductions in both heart rate and pressure variables, supporting the hypothesis that peripheral airway anaesthesia alone may be insufficient and that central sympatholysis adds clinically important benefit.

Mahjoubifard et al. compared dexmedetomidine, lignocaine, and fentanyl in cardiac-surgery patients. Dexmedetomidine was more effective than fentanyl in preventing tachycardia, lignocaine permitted an increase in mean arterial pressure during the first post-intubation minute, and dexmedetomidine produced lower mean arterial pressure at 5 and 10 minutes. Bradycardia and hypotension were more frequent with dexmedetomidine, highlighting its stronger sympatholytic effect [13]. Our findings follow the same pattern: the combination provided superior haemodynamic suppression but produced a small safety signal. The addition of nebulised lignocaine may have reinforced attenuation without requiring further opioid escalation, although the present design cannot separate the individual contributions of the two agents.

The appropriate dexmedetomidine dose remains clinically important. Sharma and Mehta compared loading doses of 0.5 and 1.0 µg/kg with placebo and found that both active doses reduced the propofol requirement, improved intubating conditions, and attenuated heart-rate and blood-pressure responses. The lower dose was therapeutically comparable to 1.0 µg/kg for these outcomes but produced fewer episodes of hypotension and bradycardia [14]. These results suggest that the 1.5 µg/kg dose used in our study may be greater than necessary for many ASA I–II patients. A lower dexmedetomidine dose combined with nebulised lignocaine could potentially preserve efficacy while improving safety and merits direct evaluation.

Kumari et al. evaluated preoperative dexmedetomidine 1 µg/kg in 60 elective-surgery patients and reported lower heart rate, systolic pressure, diastolic pressure, and mean arterial pressure during and after intubation than in controls. Dexmedetomidine also reduced anaesthetic requirements and maintained haemodynamic control beyond the immediate post-intubation period, although bradycardia and hypotension remained recognised concerns [15]. Our sustained between-group differences through 30 minutes are concordant with this prolonged sympatholytic profile. However, the marked late pressure differences in our data may also reflect the combined effects of dexmedetomidine, fentanyl, propofol, and volatile anaesthesia rather than attenuation of the intubation response alone.

Comparisons with short-acting opioids further clarify the clinical trade-off. Lee et al. randomized patients to dexmedetomidine or remifentanil during induction and found that both agents limited haemodynamic responses to laryngoscopy. Remifentanil provided stronger suppression immediately after intubation, whereas dexmedetomidine produced lower heart rates and a more prolonged effect. Hypotension was more closely associated with remifentanil, while dexmedetomidine was associated with bradycardic tendencies [16]. In our trial, the combination regimen similarly maintained a prolonged reduction in heart rate and arterial pressure, but without opioid-related respiratory depression being identified as a study outcome.

The safety findings should be interpreted alongside the efficacy results. Adverse events occurred in 13.9% of combination-treated participants and none of the controls, although the overall comparison narrowly missed conventional statistical significance. Dry mouth was the predominant event and is consistent with α2-adrenergic agonism. Bradycardia and hypotension occurred together in one participant, reinforcing the need for slow infusion, close monitoring, and readiness to administer atropine, fluids, or vasopressors. The study population was limited to relatively healthy adults; haemodynamic suppression of this magnitude may have different implications in elderly patients, those receiving rate-limiting drugs, or patients with autonomic dysfunction.

Several strengths support the findings. Allocation was randomized and concealed, intravenous study solutions were prepared identically, outcome assessment was partially masked, and laryngoscopy was standardized. Haemodynamic measurements were collected repeatedly through 30 minutes, confidence intervals were reported, and multiplicity was addressed using Holm adjustment. Safety analysis included all treated participants.

The study also has limitations. Baseline heart rate and systolic pressure were significantly higher in the control group, which may have exaggerated some unadjusted post-intubation differences. Analysis relied on separate timepoint comparisons rather than a longitudinal model adjusting for baseline values and within-participant correlation. Complete masking was not possible because nebulisation was administered only to the combination group. The trial did not include dexmedetomidine-only or lignocaine-only arms, so synergistic or additive effects cannot be distinguished. Plasma lignocaine concentrations, sedation scores, intubating conditions, catecholamine levels, and recovery outcomes were not assessed. Finally, the use of 1.5 µg/kg dexmedetomidine limits direct comparison with commonly studied doses of 0.5–1.0 µg/kg.

CONCLUSION

Intravenous dexmedetomidine combined with nebulised lignocaine provided strong and sustained attenuation of tachycardia and arterial-pressure responses to laryngoscopy and tracheal intubation. The regimen was generally tolerated but was associated with dry mouth and occasional bradycardia and hypotension. Future trials should compare lower dexmedetomidine doses, include single-agent groups, and use baseline-adjusted longitudinal analyses to define the minimum effective and safest combination regimen.

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