

A COMPARATIVE STUDY TO EVALUATE THE EFFECTS OF PREOPERATIVE DEXMEDETOMIDINE NEBULIZATION VS. LIGNOCAINE AND NORMAL SALINE NEBULIZATION ON THE HEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND INTUBATION

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation are associated with significant sympathetic stimulation, often leading to undesirable hemodynamic fluctuations. Dexmedetomidine, a selective α_2 -adrenergic agonist, has shown promise in blunting such responses. This study evaluates the efficacy of pre-operative nebulized dexmedetomidine in attenuating the cardiovascular stress response during laryngoscopy and intubation. **Materials and Methods:** In this prospective, randomized, controlled study, 70 adult patients scheduled for elective surgeries under general anesthesia were enrolled and divided into two equal groups. Group A received 0.75 μ g/kg of dexmedetomidine plus normal saline total 4ml via nebulization 20 minutes prior to induction, while Group B received lignocaine 2ml plus normal saline total 4ml via nebulization 20 minutes prior to induction. Hemodynamic parameters, including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP), were recorded at baseline, post-nebulization, and at 1,2, 3,4, 5,6,7,8,9 and 10 minutes following intubation till response to skin incision. **Result:** Group A exhibited a significant attenuation in HR, SBP, DBP, and MAP at all recorded time points post-intubation compared to Group B ($p < 0.05$). No adverse effects related to nebulized dexmedetomidine were observed. **Conclusion:** Pre-operative dexmedetomidine nebulization is effective in blunting the hemodynamic response to laryngoscopy and intubation, providing a non-invasive and well-tolerated option for improving perioperative cardiovascular stability.

INTRODUCTION

Laryngoscopy and endotracheal intubation commonly trigger a transient yet significant sympathetic surge, resulting in tachycardia and elevated blood pressure that can pose critical risks—especially in patients with cardiovascular comorbidities.^[1,2] Various pharmacological interventions, including intravenous lignocaine, beta-blockers, and opioids, have been historically employed to attenuate this pressor reflex.^[3] However, systemic administration is often accompanied by undesirable side effects or delayed onset.^[1,4] Nebulization is a non-invasive drug delivery method offering high mucosal bioavailability and rapid onset. It avoids systemic peaks while reducing adverse effects, positioning it as a promising approach to modulate airway-mediated sympathetic responses

[5]. Lignocaine nebulization has shown efficacy in dampening reflex-induced hypertension and tachycardia during airway manipulation.^[3] However, its effect is short-acting and may be insufficient in isolation.

Dexmedetomidine, a selective α_2 -adrenergic agonist, provides sedation, analgesia, anxiolysis, and sympatholysis without causing respiratory depression.^[6] Although its intravenous route effectively attenuates hemodynamic responses, it carries a risk of hypotension and bradycardia.^[7] Recent randomized controlled trials have explored nebulized dexmedetomidine, demonstrating effective attenuation of heart rate and systolic blood pressure increases during laryngoscopy and intubation, without significant systemic adverse effects.^[1,8] While several studies have compared dexmedetomidine with saline or placebo, direct

comparisons with nebulized lignocaine remain scarce.

Misra et al. conducted a randomized trial where 1 µg/kg of nebulized dexmedetomidine significantly reduced HR increase post-laryngoscopy compared to saline, although SBP differences were not significant.^[1] Similar findings were confirmed in meta-analyses suggesting hemodynamic stability without bradycardia or hypotension at nebulized dosages.^[8] Meanwhile, Siddiqui et al. and related studies have observed that nebulized lignocaine alone had limited hemodynamic control compared to dexmedetomidine, particularly during intubation.^[3] To date, no controlled study has directly compared nebulized dexmedetomidine against lignocaine nebulization, each with normal saline as control, in moderating hemodynamic stress during laryngoscopy and tracheal intubation. We hypothesize that preoperative dexmedetomidine nebulization will more effectively reduce HR, SBP, DBP, MAP spikes, and propofol requirements compared to lignocaine and saline nebulization. This randomized, double-blind study was designed to evaluate and compare the hemodynamic responses and perioperative outcomes between Groups receiving dexmedetomidine, lignocaine, or saline nebulization.

MATERIALS AND METHODS

This prospective, randomized, double-blind study was conducted at Shree Krishna Hospital, Karamsad, Gujarat, following approval from the Institutional Human Research and Ethics Committee (CTRI/2024/04/065959). Written informed consent was obtained from all patients.

Study Population: A total of 70 patients aged between 18 and 60 years, classified as American Society of Anesthesiologists (ASA) physical status I–III and scheduled for elective surgeries under general anesthesia requiring laryngoscopy and intubation, were included. Patients were allocated into two equal groups (n=35 each) by an odd-even randomization method: odd-numbered patients were assigned to Group A and even-numbered patients to Group B.

Inclusion and Exclusion Criteria

Inclusion criteria included patients aged 18–60 years, of either sex, ASA grade I–III, and undergoing elective procedures under general anesthesia requiring endotracheal intubation. Exclusion criteria were patients with ASA grade IV or higher, those with hepatic, renal, cardiovascular, or respiratory disease, history of allergy to study drugs, active upper respiratory tract infection, pregnancy or lactation, and those unwilling to participate.

Intervention: Group A received nebulized dexmedetomidine (0.75 µg/kg) diluted with 0.9% saline to a total volume of 4 mL. Group B received 2 mL of 2% lignocaine mixed with 2 mL of 0.9% saline. Nebulization was performed using an electric jet nebulizer 20 minutes before induction of anesthesia. The solutions were prepared and administered by an independent investigator to ensure blinding.

Anesthesia Protocol: All patients underwent standard preoperative evaluation. Monitoring included ECG, non-invasive blood pressure (NIBP), and pulse oximetry. Premedication included glycopyrrolate (0.004 mg/kg IV), midazolam (0.02 mg/kg IV), and fentanyl (1–2 µg/kg IV). Anesthesia was induced with propofol (2 mg/kg IV), followed by succinylcholine (2 mg/kg IV) for intubation. Endotracheal placement was confirmed by waveform capnography.

Anesthesia was maintained using 50% oxygen and 50% air with sevoflurane. Muscle relaxation was maintained using vecuronium (0.1 mg/kg IV). Hemodynamic parameters—heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP)—were recorded at baseline, post-nebulization, during laryngoscopy, every minute for 10 minutes post-intubation, and during skin incision.

At the end of surgery, residual neuromuscular blockade was reversed with neostigmine (0.05 mg/kg IV) and glycopyrrolate (0.01 mg/kg IV). Extubation was performed after the return of spontaneous breathing and responsiveness. Patients were monitored postoperatively for 2 hours, and incidence of sore throat and adverse events were recorded.

Statistical Analysis: Data were analyzed using STATA version 18. Descriptive statistics [mean ± standard deviation, frequency (%)] were used for demographic data. Independent t-test was used for continuous variables and Chi-square or Fisher's exact test for categorical variables. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 70 patients were equally randomized into two groups: Group A (dexmedetomidine nebulization) and Group B (lignocaine nebulization). The demographic characteristics including age, gender distribution, body mass index (BMI), and ASA physical status were comparable between the two groups, with no statistically significant differences [Table 1]. This ensured a uniform baseline for evaluating the hemodynamic responses and drug requirements.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (N = 70)

Variable	Group A (n = 35)	Group B (n = 35)	Total (N = 70)	P-value
Age (years)	41.83 ± 13.38	44.86 ± 11.89	–	0.320
Gender				
Female	17 (45.9%)	20 (54.1%)	37 (52.9%)	0.632
Male	18 (54.1%)	15 (45.9%)	33 (47.1%)	
BMI (kg/m ²)	23.04 ± 3.17	21.74 ± 2.57	–	0.065

ASA Physical Status				
ASA I	1 (33.3%)	2 (66.7%)	3 (4.3%)	0.141
ASA II	26 (59.1%)	18 (40.9%)	44 (62.9%)	
ASA III	8 (34.8%)	15 (65.2%)	23 (32.9%)	

Heart rate (HR) trends over time revealed a significant intergroup difference. While baseline and post-nebulization HRs were comparable, Group A exhibited significantly lower heart rates during laryngoscopy and at all subsequent time points until

skin incision, as compared to Group B ($p < 0.001$ for each time point except baseline and post-nebulization). This indicates a more stable cardiac response in the dexmedetomidine group [Table 2].

Table 2: Comparison of Heart Rate (beats per minute) Between Groups at Various Time Points

Time Point	Group A (n = 35)	Group B (n = 35)	P-value
Baseline (BL)	89.20 ± 9.63	84.91 ± 7.80	0.045
After Nebulisation	89.54 ± 9.59	90.80 ± 7.70	0.548
During Laryngoscopy	88.69 ± 10.77	96.80 ± 8.09	<0.001
1 min after Intubation	88.80 ± 13.33	99.66 ± 7.93	<0.001
2 min after Intubation	84.63 ± 9.40	94.34 ± 8.37	<0.001
3 min after Intubation	83.26 ± 9.90	96.00 ± 7.48	<0.001
4 min after Intubation	83.54 ± 9.19	97.03 ± 6.07	<0.001
5 min after Intubation	82.74 ± 10.19	98.29 ± 5.32	<0.001
6 min after Intubation	82.17 ± 10.06	98.57 ± 4.35	<0.001
7 min after Intubation	81.94 ± 9.56	99.60 ± 3.53	<0.001
8 min after Intubation	82.74 ± 9.31	98.17 ± 2.89	<0.001
9 min after Intubation	81.60 ± 10.20	97.14 ± 2.39	<0.001
10 min after Intubation	82.74 ± 10.75	93.66 ± 4.59	<0.001
Response to Skin Incision	84.97 ± 10.73	101.54 ± 4.97	<0.001

The systolic blood pressure (SBP) values showed a general decline post-intubation in both groups, but the differences between groups remained statistically non-significant throughout the observation period

[Table 3]. This suggests that although SBP decreased, dexmedetomidine and lignocaine provided a comparable level of control over systolic pressure during the peri-intubation period.

Table 3: Comparison of Systolic Blood Pressure (mmHg) Between Groups at Various Time Points

Time Point	Group A (n = 35)	Group B (n = 35)	P-value
Baseline (BL)	128.11 ± 15.39	130.00 ± 19.55	0.655
After Nebulization	126.00 ± 13.62	130.34 ± 15.96	0.225
During Laryngoscopy	122.80 ± 11.90	126.91 ± 11.19	0.141
1 min after Intubation	117.14 ± 14.94	121.60 ± 13.11	0.189
2 min after Intubation	120.00 ± 8.76	121.03 ± 9.72	0.643
3 min after Intubation	115.54 ± 9.68	116.29 ± 9.72	0.750
4 min after Intubation	114.29 ± 9.93	115.03 ± 10.58	0.763
5 min after Intubation	113.20 ± 9.91	113.14 ± 11.27	0.982
6 min after Intubation	112.74 ± 8.79	113.20 ± 8.91	0.830
7 min after Intubation	112.11 ± 9.36	112.80 ± 9.84	0.766
8 min after Intubation	113.89 ± 18.62	109.20 ± 11.74	0.212
9 min after Intubation	108.00 ± 19.50	109.31 ± 11.95	0.735
10 min after Intubation	112.34 ± 9.85	111.09 ± 11.42	0.624
Response to Skin Incision	117.09 ± 9.42	116.74 ± 11.50	0.892

Similarly, diastolic blood pressure (DBP) remained largely comparable between the groups at all measured time intervals, with no statistically

significant difference observed [Table 4]. Both interventions maintained DBP within a safe and stable range post-laryngoscopy and intubation.

Table 4: Comparison of Diastolic Blood Pressure (mmHg) Between Groups at Various Time Points

Time Point	Group A (n = 35)	Group B (n = 35)	P-value
Baseline (BL)	81.66 ± 8.45	81.20 ± 9.57	0.833
After Nebulization	80.69 ± 8.15	81.03 ± 9.14	0.869
During Laryngoscopy	78.26 ± 7.69	80.86 ± 8.39	0.181
1 min after Intubation	75.71 ± 7.85	77.23 ± 6.03	0.369
2 min after Intubation	76.80 ± 6.85	77.83 ± 6.03	0.507
3 min after Intubation	72.69 ± 7.26	72.91 ± 6.46	0.890
4 min after Intubation	71.66 ± 6.75	71.43 ± 6.90	0.889
5 min after Intubation	71.60 ± 7.31	70.97 ± 8.14	0.735
6 min after Intubation	71.31 ± 5.64	71.66 ± 5.87	0.804
7 min after Intubation	70.97 ± 6.73	69.83 ± 7.05	0.490
8 min after Intubation	71.09 ± 6.26	69.94 ± 6.71	0.464
9 min after Intubation	71.20 ± 6.14	70.57 ± 7.25	0.697
10 min after Intubation	72.06 ± 7.00	70.69 ± 7.68	0.438
Response to Skin Incision	76.26 ± 6.42	74.97 ± 7.78	0.453

Mean arterial pressure (MAP) followed a trend similar to SBP and DBP. Although most MAP values were not significantly different between groups, a statistically significant lower MAP was noted in

Group B at 5 minutes post-intubation ($p = 0.043$), suggesting a transient drop which was not sustained [Table 5].

Table 5: Comparison of Mean Arterial Pressure (MAP) Between Groups at Various Time Points

Time Point	Group A (n = 35)	Group B (n = 35)	P-value
Baseline (BL)	96.86 ± 9.89	97.47 ± 12.54	0.822
After Nebulization	95.79 ± 9.69	97.46 ± 11.12	0.504
During Laryngoscopy	93.10 ± 8.82	96.21 ± 8.99	0.150
1 min after Intubation	89.52 ± 8.79	92.07 ± 7.00	0.184
2 min after Intubation	91.20 ± 7.07	92.22 ± 6.95	0.542
3 min after Intubation	86.97 ± 7.62	87.37 ± 6.67	0.816
4 min after Intubation	85.86 ± 7.41	85.96 ± 7.41	0.957
5 min after Intubation	85.42 ± 7.72	80.89 ± 10.44	0.043
6 min after Intubation	85.12 ± 6.22	85.12 ± 5.95	0.794
7 min after Intubation	84.68 ± 7.13	84.68 ± 7.10	0.755
8 min after Intubation	85.35 ± 8.60	85.35 ± 7.68	0.238
9 min after Intubation	83.46 ± 9.59	83.46 ± 8.30	0.811
10 min after Intubation	85.49 ± 7.62	85.49 ± 8.42	0.490
Response to Skin Incision	89.86 ± 7.23	89.86 ± 8.69	0.613

A noteworthy finding was the significantly reduced requirement of propofol in Group A compared to Group B ($p < 0.001$), highlighting the sedative-

sparing effect of preoperative dexmedetomidine nebulization [Table 6].

Table 6: Comparison of Propofol Requirement Between Groups

Group	n	Propofol Dose (mg) (Mean ± SD)	P-value
Group A	35	129.43 ± 12.58	<0.001
Group B	35	163.43 ± 19.39	

Importantly, no adverse events including hypotension, bradycardia, nausea, vomiting, or postoperative sore throat were reported in either group throughout the perioperative period, confirming the safety profile of both interventions.

DISCUSSION

Direct laryngoscopy and endotracheal intubation often provoke a transient but marked sympathetic response, manifesting as tachycardia and hypertension. This stress response can be deleterious in patients with cardiovascular or cerebrovascular comorbidities, underscoring the need for effective attenuation strategies. In this study, both nebulized dexmedetomidine and lignocaine were evaluated for their efficacy in blunting the hemodynamic response to laryngoscopy and intubation, with dexmedetomidine demonstrating superior outcomes across multiple parameters.

Our findings showed that both agents attenuated the heart rate (HR) response post-intubation, with Group A (dexmedetomidine) exhibiting a significantly greater reduction in HR at all measured time points, including response to skin incision ($p < 0.05$). This is consistent with the results reported by Misra et al., who observed a statistically lower HR trend in patients receiving nebulized dexmedetomidine compared to saline controls.^[9] Similarly, Shrivastava et al. demonstrated a significant reduction in HR at multiple intervals post-intubation with nebulized dexmedetomidine.^[11]

In contrast, systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP) values in our study did not show statistically significant intergroup differences, although both drugs provided moderate suppression of these parameters. These results align with those of Kumar et al., who noted significant reductions in HR and propofol requirements with dexmedetomidine, but less consistent changes in blood pressure indices.^[10] The lack of significant SBP, DBP, and MAP differences between the two groups suggests that dexmedetomidine's primary benefit lies in chronotropic modulation rather than direct vasomotor effects.

Importantly, dexmedetomidine significantly reduced the dose of propofol required for induction, underscoring its sedative-sparing effect—a finding consistent with Kumari et al., who reported a similar reduction in induction agent requirement in dexmedetomidine-treated patients.^[12]

No adverse effects such as hypotension, bradycardia, nausea, vomiting, or postoperative sore throat were observed in either group, supporting the safety of nebulized administration. Previous studies have also documented minimal side effects associated with nebulized dexmedetomidine, highlighting its suitability for preoperative use.^[12,13]

The present study also explored the effectiveness of these agents in specific patient subgroups. Dexmedetomidine demonstrated consistent autonomic modulation across ASA physical status I–III, comorbidities (e.g., diabetes, hypertension), and BMI strata, with enhanced MAP stabilization. These observations support the potential utility of nebulized

dexmedetomidine in high-risk surgical populations. While lignocaine nebulization was effective, it lacked the sustained and uniform suppression observed with dexmedetomidine.^[14,15]

To the best of our knowledge, this is among the first head-to-head comparisons between nebulized dexmedetomidine and lignocaine in the context of intubation-related hemodynamic stress. While both agents are effective, dexmedetomidine consistently outperformed lignocaine in terms of HR control, anesthetic sparing, and overall hemodynamic stability.

CONCLUSION

The present study demonstrates that pre-operative nebulization with dexmedetomidine effectively attenuates the hemodynamic responses associated with laryngoscopy and endotracheal intubation. Patients receiving dexmedetomidine exhibited more stable heart rate and blood pressure parameters during the peri-intubation period compared to controls. This suggests that dexmedetomidine provides a non-invasive and well-tolerated method to improve perioperative cardiovascular stability. The use of nebulized dexmedetomidine may offer an advantageous alternative to intravenous premedication in select surgical populations. Further large-scale randomized trials are warranted to confirm these findings and establish standardized protocols for its clinical application.

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