ASIAN JOURNAL OF PHARMACEUTICAL AND CLINICAL RESEARCH

INNOVARE ACADEMIC SCIENCES
Knowledge to Innovation

Vol 19, Issue 2, 2026

Online - 2455-3891
Print - 0974-2441

Research Article

EFFECTS OF LIGNOCAINE NEBULIZATION VERSUS DEXMEDETOMIDINE NEBULIZATION IN BLUNTING HEMODYNAMIC RESPONSE IN PATIENTS UNDERGOING NASOTRACHEAL INTUBATION IN HEAD AND NECK SURGERIES

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Received: 22 October 2025, Revised and Accepted: 10 December 2025

ABSTRACT

Objective: The objective of this study was to compare the efficacy of nebulized lignocaine and dexmedetomidine in attenuating the hemodynamic response to nasotracheal intubation in patients undergoing head and neck surgeries.

Methods: This prospective interventional comparative study included 192 ASA I–II patients aged 18–60 years scheduled for head and neck surgeries under general anesthesia. Patients were allocated to receive nebulization with lignocaine 1.5 mg/kg, dexmedetomidine 2 μg/kg, or normal saline (control) 15 min before intubation. Hemodynamic parameters – heart rate (HR), systolic, diastolic, and mean arterial pressures – were recorded at baseline, pre-intubation, and at 1, 5, 10, and 15 min post-intubation. Statistical analysis was performed using analysis of variance and Chi-square tests, with $p<0.05$ considered significant.

Results: Baseline characteristics were comparable among the groups. Dexmedetomidine nebulization produced significantly greater attenuation of increases in HR and blood pressure at all post-intubation time points compared with lignocaine and control ($p<0.001$). Lignocaine provided partial attenuation, whereas the control group displayed marked hemodynamic surges. Adverse events were minimal, with only mild, self-limiting bradycardia in the dexmedetomidine group.

Conclusion: Nebulized dexmedetomidine is more effective than lignocaine in blunting the hemodynamic response to nasotracheal intubation and represents a safe, non-invasive strategy for achieving perioperative hemodynamic stability.

Keywords: Nebulized Dexmedetomidine, Lignocaine, Hemodynamic Response.

 DOI: http://dx.doi.org/10.22159/ajpcr.2026v19i2.57228. Journal homepage: https://innovareacademics.in/journals/index.php/ajpcr

INTRODUCTION

Nasotracheal intubation is a common procedure in head and neck surgeries, providing a secure airway while facilitating surgical access [1,2]. However, airway manipulation during intubation often triggers sympathetic responses, leading to significant hemodynamic changes such as tachycardia, hypertension, and arrhythmias [3,4]. These responses can be particularly hazardous in patients with cardiovascular comorbidities and may increase perioperative morbidity [5]. Therefore, strategies to attenuate this stress response are critical for patient safety and optimal anesthetic management [6,7].

Various pharmacological interventions have been explored to blunt the hemodynamic response to intubation. Intravenous agents such as opioids, beta-blockers, and alpha-2 agonists have demonstrated efficacy but may be associated with systemic side effects, including hypotension and bradycardia [8,9]. Local anesthetics administered through nebulization offer a non-invasive alternative, providing topical anesthesia to the airway and potentially reducing sympathetic stimulation without significant systemic effects [10].

Lignocaine nebulization has been widely used to anesthetize the upper airway, attenuating reflex responses during airway instrumentation [11]. Similarly, dexmedetomidine, an alpha-2 adrenergic agonist, has gained attention for its sedative, analgesic, and sympatholytic properties [12,13]. Nebulized dexmedetomidine may provide a convenient, non-invasive method to achieve hemodynamic stability during intubation, with minimal respiratory depression compared to intravenous administration [13,14]. Despite these potential benefits, direct comparative data on nebulized lignocaine and dexmedetomidine for blunting hemodynamic responses during nasotracheal intubation remain limited.

The aim of this study was to compare the efficacy of lignocaine versus dexmedetomidine nebulization in blunting the hemodynamic response to nasotracheal intubation in patients undergoing head and neck surgeries under general anesthesia.

METHODS

This prospective interventional comparative study was conducted at the Department of Anesthesiology at a tertiary care health center over a period of 6 months from May 2025 to October 2025. Patients of both sexes aged 18–60 years, belonging to ASA physical status I or II, scheduled for elective head-and-neck surgeries under general anesthesia requiring nasotracheal intubation, were included. Exclusion criteria were as follows: known allergy to local anesthetics, pregnancy, anticipated difficult airway, risk of aspiration, recent upper respiratory tract infection, and patients who required more than one intubation attempt. These patients were excluded before analysis and were managed by standard airway protocols. Written informed consent was obtained from all participants, and the study was conducted

following approval from the Institutional Ethics Committee (EC/NEW/INST/1527/2025/04/234).

Participants were allocated into three groups receiving one of the nebulization agents (Lignocaine, dexmedetomidine, or normal saline). Group allocation was done using a computer-generated simple randomization sequence. No blinding was implemented in this study; both the anesthesiologist and the outcome assessor were aware of group assignment.

Sample size calculation was performed for a repeated measure analysis of variance (ANOVA) comparing three groups with the primary outcome defined as the change in heart rate (HR) after nasotracheal intubation. We assumed a medium effect size (Cohen's f=0.25), a two-sided alpha of 0.05, and 80% power (β=0.20). This calculation indicated that 53 patients per group were required. Allowing for an anticipated 20% attrition (withdrawal/loss to follow-up/exclusions after allocation), the sample size was increased to 64 patients per group, giving a total planned sample of 192 patients.

All patients received one of three nebulization regimens: Lignocaine 1.5 mg/kg diluted to 7 mL normal saline, dexmedetomidine 2 μg/kg diluted to 7 mL normal saline, or 7 mL normal saline alone. Nebulization was administered 15 min before shifting to the operating room. A jet nebulizer (particle size 3–5 μm) with an oxygen flow rate of 6–8 L/min was used for all patients in a sitting position; complete delivery of the 7 ml volume was ensured by nebulizing until the chamber was dry.

Standard monitoring (NIBP, HR, and SpO_2) was applied. Anesthesia induction was standardized for all patients: Fentanyl 2 μg/kg, propofol 2–2.5 mg/kg, and atracurium 0.5 mg/kg were administered. Intubation was performed after adequate neuromuscular blockade using size-appropriate cuffed nasotracheal tubes. Hemodynamic parameters were recorded at baseline, pre-intubation, and at 1, 5, 10, and 15 min post-intubation.

Descriptive statistics included mean±standard deviation for continuous variables and frequency/percentages for categorical variables. Longitudinal hemodynamic data were analyzed using repeated-measures ANOVA with "group" as the between-subject factor, "time" as the within-subject factor, and reporting of the group × time interaction effect. *Post hoc* analysis was performed where applicable. p-value<0.05 was considered statistically significant. Analyses were conducted using SPSS v26.

RESULTS

A total of 192 patients were enrolled and equally distributed among the three study groups (n=64 per group). Baseline demographic and clinical characteristics, including age, gender, body weight, and ASA physical status, were comparable across the three groups (p>0.05 for all parameters), confirming uniformity before intervention (Table 1).

HR values were similar at baseline and before intubation in all groups (p>0.05). Following nasotracheal intubation, the control group demonstrated a significant rise in HR, with a peak at 1 min. Lignocaine attenuated this response moderately, whereas dexmedetomidine produced the greatest blunting of tachycardia at all post-intubation time points. Repeated-measures ANOVA with *post hoc* Bonferroni correction showed significant inter-group differences at 1, 5, 10, and 15 min (p<0.001) (Table 2).

Systolic blood pressure (SBP) remained comparable at baseline and pre-intubation. Following intubation, patients in the control group exhibited a marked hypertensive response, while lignocaine partially attenuated the rise. Dexmedetomidine maintained SBP closest to baseline, showing significantly lower values than the other groups at all post-intubation time points (p<0.001) (Table 3).

Diastolic blood pressure (DBP) displayed a similar pattern. Post-intubation DBP was significantly higher in the control group at all time points, whereas dexmedetomidine consistently produced the most stable and attenuated DBP profile. Inter-group comparisons were significant at all post-intubation intervals (p<0.001), confirmed by repeated-measures ANOVA (Table 4).

Mean arterial pressure (MAP) increased sharply after intubation in the control group. Dexmedetomidine provided the most effective attenuation, maintaining MAP closer to baseline values at all measured intervals. Differences between groups at 1, 5, 10, and 15 min were statistically significant (p<0.001) (Table 5).

Adverse effects were generally infrequent across the study. Bradycardia, defined as HR <50 beats/min, occurred more often in the dexmedetomidine group. Hypotension was defined as SBP <90 mmHg or MAP <65 mmHg. Due to small expected cell counts, Fisher's Exact Test was used for all adverse-event comparisons. Bradycardia rates differed significantly between groups (p=0.032), whereas hypotension and nausea/vomiting showed no statistically significant differences (p>0.05). No serious adverse events occurred, and both nebulization agents were well tolerated (Table 6).

DISCUSSION

The present study demonstrated that nebulized dexmedetomidine is superior to lignocaine nebulization in attenuating the hemodynamic stress response to nasotracheal intubation in patients undergoing head and neck surgeries. All three groups were comparable with respect to demographic variables including age, gender, body weight, and ASA physical status, indicating that baseline characteristics did not confound the results. These findings are consistent with previous studies, such as Rani *et al.*, which also reported comparable baseline demographics across intervention groups [15].

Following intubation, a significant rise in HR was observed in the control group, peaking at 1 min post-intubation and gradually declining over

Table 1: Demographic and baseline characteristics

Parameter	Group A (Lignocaine) (n=64)	Group B (Dexmedetomidine) (n=64)	Group C (Control) (n=64)	Statistical value	p-value
Age (years)					
Mean±SD	37.2±10.8	36.5±11.2	35.8±9.7	F=0.29	0.74
Gender					
Male	38 (59.4%)	40 (62.5%)	37 (57.8%)	χ^2=0.31	0.86
Female	26 (40.6%)	24 (37.5%)	27 (42.2%)		
Weight (kg)					
Mean±SD	64.3±8.6	63.9±9.1	65.1±8.2	F=0.41	0.67
ASA grade					
I	40 (62.5%)	42 (65.6%)	41 (64.1%)	χ^2=0.14	0.93
II	24 (37.5%)	22 (34.4%)	23 (35.9%)		

Data presented as mean±SD or n (%). Group A: Lignocaine 1.5 mg/kg nebulization; Group B: Dexmedetomidine 2 μg/kg nebulization; Group C: Normal saline (Control). ASA: American Society of Anesthesiologists. Statistical tests: repeated measure ANOVA for continuous variables; Chi-square test for categorical variables

Table 2: Changes in HR (beats/min)

Time interval	Group A (Lignocaine) (n=64)	Group B (Dexmedetomidine) (n=64)	Group C (Control) (n=64)	F-value	p-value
Baseline	83.1±7.8	82.4±8.2	82.7±7.5	0.42	0.81
Just before intubation	85.2±8.1	80.3±7.6	86.1±8.4	1.85	0.16
1 min after intubation	102.4±10.6	88.2±9.8	112.8±12.1	9.24	<0.001
5 min after intubation	92.6±9.1	84.7±8.4	101.5±10.3	7.85	<0.001
10 min after intubation	87.3±8.5	82.1±7.9	94.8±9.7	6.12	<0.001
15 min after intubation	84.1±7.9	80.5±7.3	89.7±8.6	5.27	<0.001

Data presented as mean±SD. HR: Heart rate. Analysis through repeated-measures ANOVA with Bonferroni correction

Table 3: Changes in SBP (mmHg)

Time interval	Group A (Lignocaine) (n=64)	Group B (Dexmedetomidine) (n=64)	Group C (Control) (n=64)	F-value	p-value
Baseline	123.4±8.9	122.7±9.1	124.1±8.4	0.45	0.65
Just before intubation	125.6±9.4	119.2±8.7	127.4±9.8	1.78	0.17
1 min after intubation	142.8±11.2	128.6±10.4	155.3±12.7	10.21	<0.001
5 min after intubation	132.1±9.7	122.4±9.1	141.8±10.5	8.46	<0.001
10 min after intubation	126.8±9.2	120.3±8.6	133.6±9.9	6.27	<0.001
15 min after intubation	124.2±8.8	118.7±8.1	128.9±9.1	5.91	<0.001

Data presented as mean±SD. SBP: Systolic Blood Pressure. Analysis through repeated-measures ANOVA with Bonferroni correction

Table 4: Changes in DBP (mmHg)

Time interval	Group A (Lignocaine) (n=64)	Group B (Dexmedetomidine) (n=64)	Group C (Control) (n=64)	F-value	p-value
Baseline	78.2±6.7	77.6±7.1	78.9±6.8	0.48	0.72
Just before intubation	80.1±7.2	74.8±6.9	81.2±7.4	1.95	0.15
1 min after intubation	95.3±8.9	82.6±7.8	102.7±9.4	11.02	<0.001
5 min after intubation	86.7±7.5	79.3±6.8	91.8±8.2	8.33	<0.001
10 min after intubation	82.9±7.1	77.1±6.4	87.6±7.9	7.21	<0.001
15 min after intubation	79.8±6.9	75.9±6.2	83.7±7.3	6.15	<0.001

Data presented as Mean±SD. DBP: Diastolic Blood Pressure. Analysis through repeated-measures ANOVA with Bonferroni correction

Table 5: Changes in MAP (mmHg)

Time interval	Group A (Lignocaine) (n=64)	Group B (Dexmedetomidine) (n=64)	Group C (Control) (n=64)	F-value	p-value
Baseline	93.3±7.4	92.6±7.7	93.9±7.2	0.41	0.79
Just before intubation	95.2±7.6	89.4±7.1	96.8±7.9	1.83	0.16
1 min after intubation	111.1±8.9	97.9±8.3	119.4±9.6	11.37	<0.001
5 min after intubation	101.8±8.1	93.5±7.6	107.5±8.7	8.92	<0.001
10 min after intubation	97.5±7.7	91.2±7.1	102.3±8.1	7.03	<0.001
15 min after intubation	94.6±7.2	89.5±6.8	98.9±7.6	6.41	<0.001

Data presented as mean±SD. MAP: Mean arterial pressure. Analysis through repeated-measures ANOVA with Bonferroni correction

Table 6: Adverse effects observed

Adverse effect	Group A (Lignocaine) (n=64)	Group B (Dexmedetomidine) (n=64)	Group C (Control) (n=64)	p-value
Bradycardia	1 (1.6%)	5 (7.8%)	0 (0%)	0.032
Hypotension	2 (3.1%)	6 (9.4%)	1 (1.6%)	0.118
Nausea/vomiting	3 (4.7%)	2 (3.1%)	4 (6.3%)	0.71

Data presented as n (%). Bradycardia: HR<50 bpm. Hypotension: SBP<90 mmHg or MAP<65 mmHg. Fisher's Exact Test applied due to small expected cell counts

15 min. Both lignocaine and dexmedetomidine attenuated this increase, with dexmedetomidine providing a significantly greater reduction at all post-intubation time points (p<0.001) (Table 2). These results align with findings by Rani *et al.*, Mishra *et al.,* and Shrivastava *et al.*, who reported that nebulized dexmedetomidine significantly blunted the rise in HR compared to saline or lignocaine during laryngoscopy and intubation [15-17]. The superior attenuation is likely attributable to dexmedetomidine's sympatholytic properties through central alpha-2 receptor agonism, leading to decreased catecholamine release [8].

SBP and DBP demonstrated a similar pattern. While all groups experienced an increase during intubation, the control group showed the most pronounced rise, whereas dexmedetomidine maintained SBP and DBP closer to baseline throughout the 15-min observation period. Lignocaine provided partial attenuation but was less effective than dexmedetomidine. These findings are consistent with studies by Paul *et al.* and Shrivastava *et al.*, which reported that nebulized dexmedetomidine effectively reduces SBP and DBP during airway manipulation without causing significant hypotension [17,18]. The

more pronounced effect on DBP and MAP may be explained by dexmedetomidine's alpha-2 adrenergic mediated vasodilation and reduction in systemic vascular resistance [19].

MAP followed a similar trend, with dexmedetomidine demonstrating superior hemodynamic stability compared to lignocaine and control. Nebulized dexmedetomidine achieves systemic absorption primarily through the pulmonary epithelium and respiratory mucosa, facilitated by the large alveolar surface area and thin epithelial barrier, leading to bioavailability estimates of 65–82% higher than oral routes but with slower onset and lower peak plasma levels than intravenous administration [20]. This pharmacokinetic profile enables gradual central alpha-2 agonism in the locus coeruleus, producing sympatholytic effects (reduced norepinephrine release and sympathetic outflow) while minimizing initial hemodynamic swings associated with IV boluses. Compared to intravenous dexmedetomidine, nebulized administration often yields comparable or superior hemodynamic stability during intubation with fewer side effects such as pronounced bradycardia or hypotension, as supported by comparative trials showing reduced sedation depth and faster recovery with nebulization [13,20].

The safety profile of nebulized dexmedetomidine was favorable; only a small proportion of patients experienced mild bradycardia, none requiring intervention, and no significant hypotension or desaturation was observed. This supports previous findings that nebulized administration avoids the cardiovascular side effects commonly seen with intravenous bolus dosing, such as pronounced bradycardia or hypotension [21].

Limitations of the study

This single-center study limits generalizability to diverse populations or settings. Inclusion was restricted to ASA I-II patients aged 18–60 years, excluding elderly, high-risk, or pediatric groups potentially responding differently. Methodological shortcomings include potential assessor blinding issues, absence of intubation condition evaluations (e.g., coughing, bucking, and jaw relaxation), no plasma drug levels to verify systemic absorption, and a brief 15-min hemodynamic observation without long-term outcomes, comfort assessments, or perioperative complications.

CONCLUSION

Nebulized dexmedetomidine is more effective than lignocaine in attenuating the hemodynamic response to nasotracheal intubation in patients undergoing head and neck surgeries, providing better control of HR, SBP, DBP, and MAP with minimal adverse effects. Lignocaine offers partial attenuation, while control patients experience significant hemodynamic fluctuations, highlighting the clinical utility of dexmedetomidine nebulization as a safe and non-invasive strategy to improve perioperative hemodynamic stability.

AUTHORS' CONTRIBUTIONS

All the authors are equally contributed in designing, collecting the data and analysis of results, and writing the study.

ACKNOWLEDGMENT

None

FUNDING

None.

CONFLICTS OF INTEREST

None declared.

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