e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(9); 797-801

Original Research Article

Comparison of Intravenous Lignocaine and Intravenous Dexmedetomidine for Attenuation of Hemodynamic Stress Response to Laryngoscopy and Endotracheal Intubation

Kona Sai Prakash[1], Kiran Kumar Suggala[2], T. Anusha[3]

[1]Junior Resident, Department of Anaesthesiology, Mamata Medical College, Khammam, Telangana, India.
[2]Professor & Head of Department, Department of Anaesthesiology, Mamata Medical College, Khammam, Telangana, India.
[3]Associate Professor, Department of Anaesthesiology, Mamata Medical College, Khammam, Telangana, India.

Received: 01-06-2025 / Revised: 15-07-2025 / Accepted: 21-08-2025
Corresponding author: Dr. Kiran Kumar Suggala
Conflict of interest: Nil

Abstract

Introduction: Laryngoscopy and endotracheal intubation are associated with significant sympathetic stimulation, leading to transient tachycardia, hypertension, and increased myocardial oxygen demand. Pharmacological agents such as intravenous lignocaine and dexmedetomidine are used to attenuate this hemodynamic stress response, but their comparative efficacy remains a subject of clinical interest. The objective of the study is to compare the efficacy of intravenous lignocaine and intravenous dexmedetomidine in attenuating the hemodynamic response to laryngoscopy and intubation, and to evaluate their effects on recovery characteristics and adverse events.

Materials and Methods: This prospective, randomized, comparative study was conducted at Mamata Medical College, Khammam, from January 2024 to January 2025 which included 86 adult patients (ASA I–II) undergoing elective surgery under general anesthesia. Patients were allocated into two groups: Group L received intravenous lignocaine 1.5 mg/kg, and Group D received intravenous dexmedetomidine 1 μg/kg, both administered 10 minutes before induction. Hemodynamic parameters like heart rate (HR) and mean arterial pressure (MAP) were recorded at baseline and at 1, 3, and 5 minutes after intubation. Adverse events, rescue medication use, extubation time, sedation scores, and PACU stay were also noted.

Results: Group D demonstrated significantly lower HR and MAP at 1, 3, and 5 minutes post-intubation ($p < 0.01$). Incidence of tachycardia (46.5% vs 14.0%) and hypertension (27.9% vs 7.0%) was significantly less in Group D, with fewer rescue interventions required. However, bradycardia (14.0% vs 2.3%) and hypotension (18.6% vs 4.7%) were more frequent in Group D. Recovery times were slightly prolonged in Group D but clinically acceptable.

Conclusion: Intravenous dexmedetomidine provides superior attenuation of hemodynamic response compared to lignocaine but requires vigilant monitoring for bradycardia and hypotension.

Keywords: Dexmedetomidine, Lignocaine, Hemodynamic Stress Response.

Introduction

Laryngoscopy and endotracheal intubation are essential steps in administering general anesthesia but are known to produce significant sympathetic stimulation, resulting in tachycardia, hypertension, and increased myocardial oxygen consumption [1-3]. These hemodynamic changes, though short-lived, may be clinically insignificant in healthy individuals but can precipitate adverse cardiovascular events in patients with limited cardiac reserve, cerebrovascular disease, or uncontrolled hypertension [4]. Attenuation of this stress response is therefore crucial to reduce perioperative morbidity and ensure hemodynamic stability [5]. Various pharmacological agents, including opioids, beta-blockers, vasodilators, and local anaesthetics, have been evaluated for their ability to blunt the pressor response to laryngoscopy and intubation [6]. Intravenous lignocaine is a commonly used drug due to its rapid onset, membrane-stabilizing property, and minimal side effects [7]. It reduces sympathetic outflow and helps to attenuate tachycardia and hypertension, but

its effect is often short-lived and inconsistent in achieving optimal suppression of the stress response [8]. Dexmedetomidine, a highly selective α2-adrenergic agonist, has emerged as a useful adjunct in anesthesia practice due to its sedative, sympatholytic, and analgesic properties [9]. Its use before induction has been shown to attenuate the hemodynamic response to intubation more effectively than many conventional drugs [10]. However, dexmedetomidine may be associated with bradycardia and hypotension, and its impact on recovery characteristics must also be considered, especially in short-duration surgeries [9,11]. In view of these considerations, the present study was conducted to compare the efficacy of intravenous lignocaine and intravenous dexmedetomidine in attenuating the hemodynamic stress response to laryngoscopy and endotracheal intubation, and to evaluate their effects on recovery parameters and adverse events.

Materials and Methods

This prospective, randomized, comparative study was conducted at Mamata Medical College, Khammam, from January 2024 to January 2025 after obtaining approval from the Institutional Ethics Committee and written informed consent from all participants. A total of 86 adult patients, aged 18–60 years, belonging to ASA physical status I or II and scheduled for elective surgeries under general anesthesia requiring endotracheal intubation were enrolled. Patients with anticipated difficult airway, significant cardiovascular disease, arrhythmias, uncontrolled hypertension, hepatic or renal dysfunction, pregnancy, or known allergy to study drugs were excluded. Participants were randomly allocated into two groups of 43 each using a computer-generated randomization table. Group L received intravenous lignocaine 1.5 mg/kg diluted in 10 mL normal saline over 10 minutes before induction, whereas Group D received intravenous dexmedetomidine 1 µg/kg diluted in 10 mL normal saline as an infusion over 10 minutes prior to induction. All patients were premedicated with injection glycopyrrolate 0.2 mg IV and midazolam 0.02 mg/kg IV. Standard ASA monitors were applied, including ECG, non-invasive blood pressure, and pulse oximetry, and baseline parameters were recorded. General anesthesia was induced with injection fentanyl 2 µg/kg IV followed by propofol 2 mg/kg IV and vecuronium 0.1 mg/kg IV to facilitate tracheal intubation. Laryngoscopy and intubation were performed under standard conditions by an experienced anaesthesiologist after 3 minutes of neuromuscular blockade. Heart rate and mean arterial pressure were recorded at baseline, 1 minute, 3 minutes, and 5 minutes after intubation. Any occurrence of tachycardia (>20% rise from baseline), hypertension (>20% rise in MAP), bradycardia (HR < 50 bpm), or hypotension (MAP < 65 mmHg) was noted, and rescue drugs were administered as required. After completion of surgery, patients were reversed with neostigmine and glycopyrrolate and extubated when standard criteria were met. Recovery characteristics including Ramsay sedation score at 10 minutes, time to extubation, and duration of stay in post-anesthesia care unit (PACU) were recorded. All data were compiled and analyzed using appropriate statistical tests. Continuous variables were expressed as mean ± SD and compared using Student's t-test, whereas categorical variables were analyzed using Chi-square or Fisher's exact test. A p-value < 0.05 was considered statistically significant.

Results

Baseline demographic and clinical variables were similar between the two groups. The mean age was 38.5 ± 8.2 years in Group L and 37.9 ± 7.9 years in Group D (p = 0.63). Male-to-female distribution was comparable (58.1% vs. 62.7% males, p = 0.62), as were mean body weights (68.2 ± 9.5 kg vs. 67.6 ± 9.1 kg, p = 0.74). Most patients belonged to ASA class I (69.7% in Group L vs. 62.7% in Group D, p = 0.64), confirming that both groups were well matched before intervention (Table 1).

Table 1: Baseline / demographic characteristics

Variable		Group L (n = 43)	Group D (n = 43)	p-value
Age (years)	**Mean ± SD**	38.5 ± 8.2	37.9 ± 7.9	0.63
Gender	**Male**	25 (58.1%)	27 (62.7%)	0.62
	Female	18 (41.8%)	16 (37.2%)	
Weight (kg)	**Mean ± SD**	68.2 ± 9.5	67.6 ± 9.1	0.74
ASA	**I**	30 (69.7%)	28 (62.7%)	0.64
	II	13 (30.2%)	15 (34.8%)	

Heart rate increased significantly after intubation in both groups, but the rise was attenuated with dexmedetomidine.

At 1 minute, mean HR rose to 95 ± 12 bpm in Group L compared with 81 ± 10 bpm in Group D (p < 0.001). At 3 minutes, HR remained elevated at 88 ± 11 bpm in Group L, while it was 78 ± 9 bpm in Group D (p < 0.001).

By 5 minutes, HR had nearly returned to baseline in both groups but remained significantly lower in the dexmedetomidine group (82 ± 10 bpm vs. 76 ± 8 bpm, p = 0.002) (Table 2).

Table 2: Heart rate (HR) at baseline and after laryngoscopy/intubation

Time point	Group L (n = 43)	Group D (n = 43)	p-value
Baseline	78 ± 9	76 ± 8	0.28
1 minute after intubation	95 ± 12	81 ± 10	< 0.001
3 minutes after intubation	88 ± 11	78 ± 9	< 0.001
5 minutes after intubation	82 ± 10	76 ± 8	0.002

Mean arterial pressure also showed a more pronounced rise in the lignocaine group. At 1 minute post-intubation, MAP increased to 112 ± 14 mmHg in Group L compared with 98 ± 12 mmHg in Group D ($p < 0.001$). Similar differences persisted at 3 minutes (104 ± 13 mmHg vs. 95 ± 11 mmHg, $p = 0.001$) and 5 minutes (98 ± 12 mmHg vs. 92 ± 10 mmHg, $p = 0.007$), indicating better attenuation of the hypertensive response with dexmedetomidine (Table 3).

Table 3: Mean arterial pressure (MAP) at baseline and after laryngoscopy/intubation

Time point	Group L (n = 43)	Group D (n = 43)	p-value
Baseline	94 ± 10	93 ± 9	0.45
1 minute after intubation	112 ± 14	98 ± 12	< 0.001
3 minutes after intubation	104 ± 13	95 ± 11	0.001
5 minutes after intubation	98 ± 12	92 ± 10	0.007

Adverse events were more frequent in the lignocaine group.

Tachycardia (>20% rise from baseline) occurred in 46.5% of patients in Group L versus 14.0% in Group D ($p = 0.001$), and hypertension in 27.9% versus 7.0% ($p = 0.008$). Conversely, bradycardia (HR < 50 bpm) was seen more often with dexmedetomidine (14.0% vs. 2.3%, $p = 0.01$) as was hypotension (18.6% vs. 4.7%, $p = 0.02$).

Rescue therapy for tachycardia was required in 23.3% of Group L compared with only 4.7% of Group D ($p = 0.01$), further underscoring dexmedetomidine's superior hemodynamic control (Table 4).

Table 4: Incidence of Adverse Events

Adverse Event / Intervention	Group L (n = 43)	Group D (n = 43)	p-value
Tachycardia	20 (46.5%)	6 (14.0%)	0.001
Hypertension	12 (27.9%)	3 (7.0%)	0.008
Bradycardia	1 (2.3%)	6 (14.0%)	0.010
Hypotension	2 (4.7%)	8 (18.6%)	0.020

Recovery parameters revealed a trade-off with dexmedetomidine use. The mean Ramsay sedation score at 10 minutes was higher with dexmedetomidine (3.5 ± 0.6 vs. 2.1 ± 0.4, $p < 0.001$), reflecting deeper sedation. Extubation time was prolonged (11.8 ± 3.4 min vs. 9.2 ± 2.1 min, $p < 0.001$), and PACU stay was longer (55 ± 10 min vs. 45 ± 8 min, $p < 0.001$) compared to lignocaine, suggesting slower recovery despite better stress response attenuation (Table 5).

Table 5: Recovery and Sedation Parameters

Parameter	Group L (n = 43)	Group D (n = 43)	p-value
Ramsay Sedation Score at 10 min	2.1 ± 0.4	3.5 ± 0.6	< 0.001
Time to Extubation (min)	9.2 ± 2.1	11.8 ± 3.4	< 0.001
PACU Stay	45 ± 8	55 ± 10	< 0.001

Discussion

The present study compared intravenous lignocaine and dexmedetomidine for attenuation of the hemodynamic stress response to laryngoscopy and endotracheal intubation in 86 ASA I–II patients. Baseline demographic and clinical variables were comparable, ensuring that observed differences were attributable to the study drugs rather than confounders. Both drugs attenuated the rise in heart rate (HR) and mean arterial pressure (MAP) following laryngoscopy and intubation, but dexmedetomidine was significantly more effective at all measured time points. HR rose to 95 ± 12 bpm at 1 minute in the lignocaine group compared to only 81 ± 10 bpm in the dexmedetomidine group ($p < 0.001$), and MAP increased by 13.4% versus 4% from baseline, respectively.

These findings are consistent with Gangappa RC et al., who also demonstrated that dexmedetomidine (1 μg/kg) was superior to lignocaine (1.5 mg/kg) in attenuating HR and BP responses during laryngoscopy and intubation [12]. Our results align

with earlier work by Raval DL et al., who reported that dexmedetomidine at 1 µg/kg produced greater blunting of pressor responses than lower doses, highlighting the importance of adequate dosing to achieve hemodynamic stability [13]. Similarly, Keniya VM et al. showed that dexmedetomidine reduced the maximal rise in systolic and diastolic BP to only 8% and 11% respectively, compared to 40% and 25% in the control group [14]. The findings of Singh G et al. and Samala S et al. further corroborate the present results, supporting the conclusion that dexmedetomidine provides more consistent and prolonged suppression of sympathetic activation compared to lignocaine, which may have a shorter duration of effect [15,16].

While dexmedetomidine demonstrated superior control of tachycardia and hypertension, it was associated with higher rates of bradycardia (14% vs. 2.3%) and hypotension (18.6% vs. 4.7%). These events were manageable with standard interventions and did not result in major complications. Similar adverse effect profiles have been reported by Surabathuni S et al. and Kalakeri et al., emphasizing that careful titration and vigilant monitoring are essential when using dexmedetomidine [17,18].

An additional trade-off observed in our study was the prolonged extubation time (11.8 ± 3.4 min vs. 9.2 ± 2.1 min) and PACU stay, which may be attributable to the sedative properties of dexmedetomidine, as also noted by Panchal M et al.

Overall, our findings support the use of dexmedetomidine as a preferred agent for blunting the hemodynamic response to laryngoscopy and intubation, particularly in patients were exaggerated sympathetic stimulation may pose a risk. However, it's bradycardic and hypotensive effects, along with delayed recovery, should be considered in clinical decision-making. Further studies in high-risk populations and with dose optimization strategies may help refine its use and balance efficacy with safety.

Conclusion

Intravenous dexmedetomidine at 1 µg/kg was more effective than lignocaine 1.5 mg/kg in attenuating the hemodynamic stress response to laryngoscopy and endotracheal intubation, resulting in significantly lower heart rate and mean arterial pressure at all measured intervals.

It also reduced the incidence of tachycardia and hypertension and decreased the requirement for rescue medications. However, its use was associated with a higher incidence of bradycardia, hypotension, and a modest prolongation of extubation time and PACU stay. These findings suggest that dexmedetomidine provides superior hemodynamic stability compared to lignocaine, making it a preferred option in patients where blunting of the sympathetic response is critical, though careful monitoring for bradycardia and hypotension is recommended.

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