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Comparative Study of IV Dexmedetomidine and Lignocaine for Attenuation of Cardiovascular Stress Response to Laryngoscopy and Endotracheal Intubation

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation are essential components of general anaesthesia but are often accompanied by marked sympathetic stimulation, leading to transient increases in heart rate, blood pressure, and myocardial oxygen consumption. Such hemodynamic fluctuations may pose significant risks, particularly in patients with cardiovascular or cerebrovascular disease. **Aim**: The aim of this study was to compare the efficacy of intravenous dexmedetomidine and lignocaine in attenuating the cardiovascular stress response to laryngoscopy and endotracheal intubation in adult patients undergoing elective surgery under general anaesthesia. **Methods**: A total of 80 ASA I–II patients aged 18–60 years scheduled for elective surgical procedures under general anaesthesia were enrolled and randomly divided into two groups of 40 each. Group D received intravenous dexmedetomidine 1 $\mu g \cdot kg^{-1}$ diluted in 20 mL normal saline infused over 10 minutes before induction, while Group L received intravenous lignocaine 1.5 $mg \cdot kg^{-1}$ as a bolus 90 seconds before laryngoscopy and intubation. Hemodynamic parameters including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at baseline, pre-induction, and at 0, 1, 3, and 5 minutes post-intubation. Any adverse events such as bradycardia or hypotension were noted and appropriately managed. Data were analyzed using SPSS version 26, with a p-value < 0.05 considered statistically significant. **Results**: Both dexmedetomidine and lignocaine effectively attenuated the hemodynamic response to laryngoscopy and intubation; however, dexmedetomidine produced a more significant reduction in HR and MAP at all post-intubation intervals. The mean HR rise following intubation was significantly lower in Group D (6.4%) compared to Group L (15.8%). Similarly, the mean MAP increase was attenuated more effectively in Group D (8.6%) than in Group L (17.3%). The differences were statistically significant ($p < 0.05$). A mild incidence of bradycardia was observed in 10% of patients in the dexmedetomidine group and 2.5% in the lignocaine group, which responded to atropine administration. No significant episodes of hypotension or arrhythmia were observed in either group. Recovery profiles and sedation levels were comparable and clinically acceptable. **Conclusion**: Intravenous dexmedetomidine at a dose of 1 $\mu g \cdot kg^{-1}$ administered before induction provides superior attenuation of the cardiovascular stress response to laryngoscopy and endotracheal intubation compared to intravenous lignocaine 1.5 $mg \cdot kg^{-1}$. Dexmedetomidine offers stable peri-intubation hemodynamics with a favorable safety profile, making it a valuable agent for use in patients where cardiovascular stability is critical.

Keywords: *Dexmedetomidine; Lignocaine; Laryngoscopy; Endotracheal intubation; Hemodynamic response; Sympathetic stimulation; Anaesthesia.*

INTRODUCTION

Laryngoscopy and endotracheal intubation are potent noxious stimuli that provoke a rapid sympathetic discharge and marked catecholamine release, producing transient but sometimes clinically important increases in heart rate (HR), systolic and diastolic arterial pressures (SBP, DBP), and myocardial oxygen demand. In healthy patients these changes are usually well tolerated, but in patients with coronary artery disease, hypertension, cerebrovascular disease or intracranial pathology, the pressor and tachycardic responses may precipitate myocardial ischemia, arrhythmia, or intracranial haemodynamic complications. Because of this, attenuation of the haemodynamic response to airway instrumentation is a long-standing anesthetic objective. [1, 2].

Two pharmacologic approaches commonly used to blunt the intubation response are (a) systemic (intravenous) local anaesthetics — chiefly lignocaine (lidocaine) — which reduce airway reflexes and central sympathetic drive, and (b) agents acting on central sympathetic outflow such as α2-adrenoceptor agonists (dexmedetomidine) which provide sedation, sympatholysis and analgesia without marked respiratory depression. Intravenous lignocaine (most commonly 1–1.5 $mg \cdot kg^{-1}$ given ~60–120 seconds before laryngoscopy) has been studied extensively and appears to produce small-to-moderate reductions in SBP, DBP, MAP and HR versus placebo in pooled analyses, but the magnitude and consistency of its effect depend on dose, timing and background anesthetic regimen. [3,4].

Dexmedetomidine — a highly selective α2-adrenergic agonist — produces dose-dependent sympatholysis, bradycardia and hypotension, and has been shown in a large body of randomized controlled trials and meta-analyses to substantially blunt the haemodynamic surge at laryngoscopy and intubation when given as a pre-induction bolus (commonly 0.5–1.0 $\mu g \cdot kg^{-1}$ over 10 minutes) or as an infusion. Compared with placebo or no-dexmedetomidine, pooled estimates indicate clinically meaningful reductions in SBP, MAP and HR at the time of intubation; however, dexmedetomidine increases the risk of bradycardia and hypotension, an important safety consideration particularly in hypovolaemic or cardiac-compromised patients. [5,6].

Comparative trials directly pitting dexmedetomidine against other agents (e.g., fentanyl, esmolol, labetalol) or against local anaesthetics (lignocaine) yield useful but varied results: many randomized studies and single-centre trials report that dexmedetomidine produces a more pronounced attenuation of HR and blood pressure compared with lignocaine or placebo, and it also reduces anaesthetic/opioid requirements intraoperatively; yet the trade-off is a higher incidence of bradycardia and hypotension in some series. This heterogeneity in outcomes is influenced by differences in study populations, dexmedetomidine dose and infusion protocol, timing relative to induction, type of induction agents used, and endpoints measured. These points justify a controlled head-to-head comparison with standard lignocaine dosing (e.g., 1.5 $mg \cdot kg^{-1}$ IV bolus) in a homogeneous elective surgical cohort with ASA I–II patients. [7,8,9].

Pharmacologically, lignocaine acts by sodium-channel blockade at peripheral and central sites, reducing afferent airway reflexes and blunting the immediate pressor response, whereas dexmedetomidine's effects are mediated centrally through α2-receptor binding in the locus coeruleus and in the dorsal horn, producing sympatholysis, reduced catecholamine release and analgesia that together blunt both the HR and BP response. The timing of peak drug effect is important: lignocaine's maximal circulatory effect occurs within 1–3 minutes after intravenous bolus while dexmedetomidine's onset (when given as a bolus infusion over 10 minutes) is somewhat slower but more sustained. These pharmacodynamic differences inform both study design (timing of drug administration) and clinical decision-making in practice. [4,5,10].

Given the clinical importance of reliably attenuating the intubation response while avoiding excess bradycardia or hypotension, a randomized comparison of IV dexmedetomidine versus IV lignocaine using standard, commonly used dosing regimens is warranted. The present study therefore aims to compare the efficacy and safety of (a) dexmedetomidine 1.0 $\mu g \cdot kg^{-1}$ given over 10 minutes before induction and (b) lignocaine 1.5 $mg \cdot kg^{-1}$ IV bolus given ~90 seconds before laryngoscopy, with standardized induction and monitoring, using heart rate and blood pressure changes at intubation and during the first 5 minutes afterward as the primary haemodynamic endpoints; secondary endpoints include incidence of bradycardia (HR < 50 bpm), hypotension (>30% MAP drop from baseline), and requirement for rescue atropine or vasoactive drugs. This protocol mirrors the timing and doses used in many trials and meta-analyses and therefore allows direct comparison with the existing evidence base. [1,3,5,7].

MATERIALS AND METHODS

This prospective, randomized, double-blind, controlled clinical study was conducted in the Department of Anaesthesiology, Government Medical College, Srinagar, from January 2022 to December 2025. The study was carried out as part of an institutional research project by the investigator while serving as a Senior Resident in the Department. Ethical clearance was obtained from the Institutional Ethics Committee, and written informed consent was taken from all patients prior to inclusion in the study.

Study population
A total of 80 adult patients of either sex, aged between 18 and 60 years, belonging to ASA physical status I or II, and scheduled for elective surgical procedures under general anaesthesia requiring endotracheal intubation, were enrolled. Patients were randomly assigned into two equal groups (n = 40 each):
* Group D (Dexmedetomidine group): Received intravenous dexmedetomidine 1 $\mu g \cdot kg^{-1}$ diluted in 20 mL normal saline, administered slowly over 10 minutes before induction.
* Group L (Lignocaine group): Received intravenous lignocaine (lidocaine) 1.5 $mg \cdot kg^{-1}$ as a bolus 90 seconds before laryngoscopy and intubation.

Inclusion criteria
1. Age 18–60 years.
2. ASA physical status I or II.
3. Scheduled for elective surgery under general anaesthesia with orotracheal intubation.
4. Anticipated easy airway (Mallampati Grade I or II, normal mouth opening, thyromental distance > 6 cm).

Exclusion criteria
1. Known hypersensitivity or contraindication to dexmedetomidine or lignocaine.
2. Pre-existing cardiac conduction abnormalities, significant bradycardia (< 50 bpm) or uncontrolled hypertension.
3. Healthy hard working patients with high vagal tone.
3. Patients on chronic β-blockers, α2-agonists, or calcium channel blockers.
4. Anticipated difficult airway, morbid obesity (BMI > 35 kg/m^2), pregnancy or lactation.
5. Presence of hepatic, renal, neurological, or endocrine disease.
6. Patients unwilling to participate.

Randomization and blinding
Patients were randomly allocated using a computer-generated randomization list and sealed opaque envelopes for allocation concealment. The study drugs were prepared in identical 20 mL syringes by an anaesthesiologist not involved in patient management or data collection. Both the patient and the attending anaesthesiologist were blinded to group assignment, maintaining the double-blind nature of the study.

Anaesthetic technique
Upon arrival in the operating room, patients were connected to standard monitors — ECG, non-invasive blood pressure, pulse oximetry, and capnography. Baseline readings of heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded.

An intravenous line was secured, and all patients received premedication with midazolam 0.02 $mg \cdot kg^{-1}$ and glycopyrrolate 0.004 $mg \cdot kg^{-1}$ intravenously.

The study drug was administered according to group allocation. Following infusion or bolus administration, patients were preoxygenated for three minutes. Anaesthesia was induced using propofol 2 $mg \cdot kg^{-1}$ and fentanyl 2 $\mu g \cdot kg^{-1}$. Neuromuscular blockade was achieved with atracurium0.5 $mg \cdot kg^{-1}$ to facilitate tracheal intubation. Laryngoscopy and intubation were performed by a single experienced anaesthesiologist using a Macintosh laryngoscope to ensure consistency, with intubation completed within 15 seconds.

Anaesthesia was maintained using 50% nitrous oxide in oxygen, isoflurane 1%, and intermittent doses of vecuronium bromide for muscle relaxation.

Hemodynamic monitoring and data collection
The following parameters were recorded at specific time intervals:
1. Baseline (before study drug administration)
2. Pre-induction (after study drug administration, before induction)
3. Immediately after intubation (0 minute)
4. 1 minute after intubation
5. 3 minutes after intubation
6. 5 minutes after intubation

Measured variables included HR, SBP, DBP, and MAP.
Adverse events such as bradycardia (HR < 50 bpm), hypotension (MAP ↓ > 30% from baseline), or arrhythmias were noted.

Bradycardia was treated with atropine 0.6 mg IV, and hypotension with mephentermine 6 mg IV boluses and intravenous fluids as required.

Statistical analysis

All collected data were tabulated and analyzed using SPSS version 26 (IBM Corp., USA). Quantitative data were expressed as mean ± standard deviation (SD). Inter-group comparisons were made using the independent-samples t-test, and intra-group comparisons using the paired t-test. Categorical data such as the incidence of adverse events were analyzed using the chi-square test. A p-value < 0.05 was considered statistically significant.

The sample size of 80 patients (40 per group) was calculated based on earlier pilot studies showing a 20% difference in mean arterial pressure response between dexmedetomidine and lignocaine, with a power of 80% and $\alpha = 0.05$, accounting for a 10% dropout rate.

RESULTS

Both groups were comparable with respect to demographic characteristics, baseline hemodynamic parameters, and duration of laryngoscopy. No statistically significant differences were found between the two groups in any of the demographic or baseline variables ($p > 0.05$), indicating comparability [Table 1].

Table 1: Demographic and baseline characteristics of patients

Parameter	Group D (Dexmedetomidine) Mean ± SD	Group L (Lignocaine) Mean ± SD	p-value
Number of patients (n)	40	40	--
Age (years)	37.4 ± 10.2	36.7 ± 9.8	0.74
Gender (M/F)	22/18	21/19	0.82
Weight (kg)	63.2 ± 8.6	62.4 ± 9.1	0.69
Height (cm)	166.3 ± 7.5	167.1 ± 7.1	0.71
ASA physical status (I/II)	27/13	26/14	0.83
Baseline HR (beats/min)	79.2 ± 8.1	80.3 ± 7.7	0.54
Baseline SBP (mmHg)	121.6 ± 10.3	122.9 ± 9.8	0.63
Baseline DBP (mmHg)	77.8 ± 6.7	78.4 ± 6.3	0.72
Baseline MAP (mmHg)	92.3 ± 7.4	93.1 ± 7.2	0.68
Duration of laryngoscopy (seconds)	11.5 ± 2.2	11.8 ± 2.4	0.53

Immediately after intubation, there was a transient rise in HR in both groups. However, the increase was significantly less in the dexmedetomidine group compared to the lignocaine group. HR values in Group D returned near baseline by the 3rd minute, whereas in Group L, HR remained elevated even at 5 minutes post-intubation. Dexmedetomidine effectively blunted the tachycardic response to intubation, showing significantly lower mean HR values at all post-intubation intervals ($p < 0.05$). A similar pattern was observed in systolic blood pressure. The increase in SBP immediately after intubation was significantly lower in the dexmedetomidine group compared to the lignocaine group [Table 2].

Table 2: Changes in mean heart rate (beats per minute) at different time intervals

Time Interval	Group D Mean ± SD	Group L Mean ± SD	p-value
Baseline	79.2 ± 8.1	80.3 ± 7.7	0.54
Pre-induction	76.4 ± 7.6	79.5 ± 8.2	0.09
0 min after intubation	84.2 ± 8.9	92.8 ± 9.4	0.001
1 min after intubation	85.0 ± 9.2	93.5 ± 10.1	0.002
3 min after intubation	80.6 ± 7.8	87.9 ± 8.7	0.003
5 min after intubation	78.8 ± 7.4	84.5 ± 8.2	0.004

A similar pattern was observed in systolic blood pressure. The increase in SBP immediately after intubation was significantly lower in the dexmedetomidine group compared to the lignocaine group. The attenuation of SBP rise was more consistent and profound in patients receiving dexmedetomidine, with values returning close to baseline by 5 minutes post-intubation [Table 3].

Table 3: Changes in systolic blood pressure (mmHg) at different time intervals

Time Interval	Group D Mean ± SD	Group L Mean ± SD	p-value
Baseline	121.6 ± 10.3	122.9 ± 9.8	0.63
Pre-induction	118.5 ± 9.4	121.7 ± 10.2	0.15
0 min after intubation	130.8 ± 11.6	142.1 ± 13.4	0.001
1 min after intubation	128.5 ± 10.9	139.2 ± 12.6	0.002
3 min after intubation	123.1 ± 9.8	132.4 ± 11.1	0.003
5 min after intubation	120.7 ± 8.7	127.8 ± 9.9	0.004

Both DBP and MAP followed trends similar to HR and SBP, with dexmedetomidine showing better control of hemodynamic fluctuations. MAP increase was significantly less in Group D than Group L at all time intervals after intubation ($p < 0.05$) [Table 4].

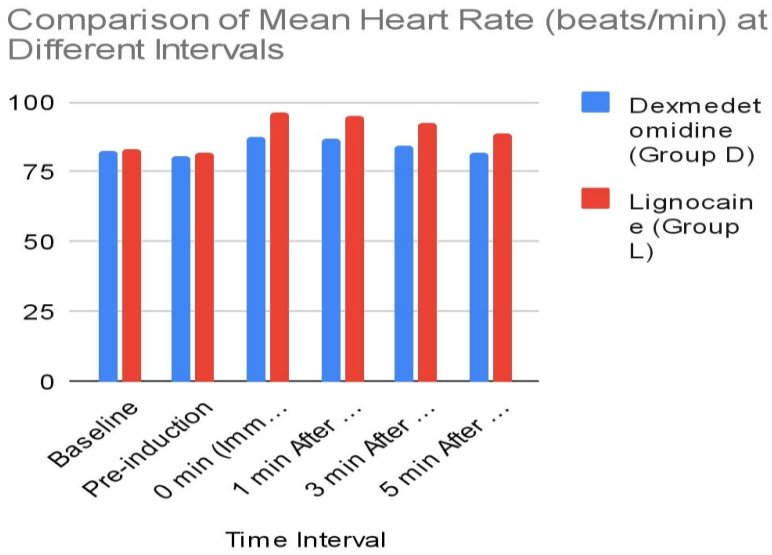
Table 4: Changes in mean arterial pressure (mmHg) at different time intervals

Time Interval	Group D Mean ± SD	Group L Mean ± SD	p-value
Baseline	92.3 ± 7.4	93.1 ± 7.2	0.68
Pre-induction	89.4 ± 6.9	91.6 ± 7.1	0.22
0 min after intubation	100.2 ± 8.7	109.2 ± 9.8	0.001
1 min after intubation	99.1 ± 8.4	107.8 ± 9.3	0.002
3 min after intubation	93.8 ± 7.1	101.4 ± 8.2	0.003
5 min after intubation	91.7 ± 6.6	97.6 ± 7.3	0.004

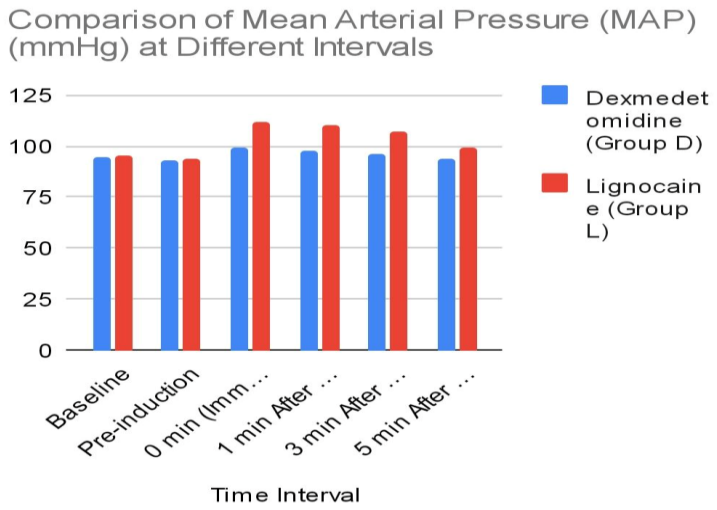
Adverse events such as bradycardia and hypotension were infrequent. Mild bradycardia occurred in 4 patients (10%) in Group D and 1 patient (2.5%) in Group L, which responded to atropine 0.6 mg IV. No significant hypotension or arrhythmias were observed. Sedation levels and recovery times were comparable between groups. There was no statistically significant difference in the incidence of complications between the two groups ($p > 0.05$). Both agents were well tolerated [Table 5].

Table 5: Incidence of adverse events and rescue interventions

Parameter	Group D (n=40)	Group L (n=40)	p-value
Bradycardia (HR < 50 bpm)	4 (10%)	1 (2.5%)	0.17
Hypotension (MAP ↓ > 30%)	2 (5%)	1 (2.5%)	0.56
Arrhythmias	0	0	--
Use of atropine	4 (10%)	1 (2.5%)	0.17
Use of vasopressor	2 (5%)	1 (2.5%)	0.56
Sedation score > 3	2 (5%)	1 (2.5%)	0.56
Delayed recovery (>10 min)	3 (7.5%)	2 (5%)	0.64



Bar graph: Comparison of Mean Heart Rate at Different intervals.



Bar graph 2: Comparison of Mean Arterial Pressure at Different Intervals.

DISCUSSION

The present study demonstrated that intravenous dexmedetomidine administered at a dose of 1 µg/kg over 10 minutes prior to induction produced a more effective attenuation of the cardiovascular stress response to laryngoscopy and tracheal intubation compared to intravenous lignocaine 1.5 mg/kg given 90 seconds before laryngoscopy. The mean heart rate and mean arterial pressure increases at 0, 1, and 3 minutes post-intubation were significantly lower in the dexmedetomidine group, highlighting its superior efficacy in blunting the pressor response.

The observed hemodynamic stability with dexmedetomidine can be attributed to its highly selective α2-adrenergic agonist property that leads to central sympatholysis, sedation, and inhibition of norepinephrine release. This results in reduced sympathetic outflow and lower catecholamine surge during airway manipulation [11]. Lignocaine, on the other hand, primarily suppresses airway reflexes and attenuates afferent impulses but fails to effectively inhibit the sympathetic discharge responsible for tachycardia and hypertension [2]. Consequently, its action remains limited and inconsistent in preventing the full stress response.

Our results corroborate several previous investigations. Menda et al. Observed that dexmedetomidine administered prior to induction effectively prevented the rise in heart rate and blood pressure following intubation [6]. Similarly, Keniya et al. Reported that dexmedetomidine significantly reduced the sympathoadrenal response to laryngoscopy compared with control [5]. A more recent randomized study by Jain demonstrated that dexmedetomidine 1 µg/kg was superior to lignocaine 1.5 mg/kg in maintaining stable hemodynamics during intubation [8]. These findings align with our observations, confirming the consistency of dexmedetomidine's attenuating effects across different clinical settings.

Although dexmedetomidine provided better hemodynamic control, a mild degree of bradycardia and hypotension was more frequently observed compared to lignocaine. This is consistent with earlier reports such as that of Seangrung et al., who found that patients receiving dexmedetomidine experienced a higher incidence of bradycardia (about 18%) and hypotension (around 50%) relative to those receiving lidocaine [12]. In our study, these effects were clinically manageable and did not require major interventions, suggesting that careful titration and monitoring can minimize such adverse events.

The clinical importance of blunting the hemodynamic response to intubation is well recognized, particularly in patients with limited cardiovascular reserve, coronary artery disease, or raised intracranial pressure [5]. Sudden surges in heart rate and blood pressure can provoke myocardial ischemia, arrhythmias, or cerebral complications. Dexmedetomidine's central sympatholytic effect therefore provides a valuable protective mechanism, especially in high-risk groups. However, its potential for bradycardia necessitates vigilance, particularly in patients with baseline low heart rate or those on β-blockers.

Our results also reinforce that lignocaine remains a useful, safe, and easily available agent with modest efficacy. Studies by Qi et al. And Zou et al. Showed lignocaine's partial effectiveness in reducing pressor responses, though its impact was smaller and shorter lasting than dexmedetomidine [4,3]. Lignocaine's short duration and limited central action explain why, in our findings, hemodynamic parameters returned toward baseline faster in its group.

The study's methodology ensured homogeneity between groups in baseline characteristics such as age, sex, ASA physical status, and intubation duration, thus minimizing confounding. The fixed-dose design and controlled induction conditions provide reliable internal validity. However, some limitations should be acknowledged. The study included only ASA I–II

patients undergoing elective procedures; results may not directly extrapolate to emergency or high-risk cardiac cases. Also, we did not assess plasma catecholamine levels, which might have provided biochemical corroboration of stress attenuation.

Future research should explore the use of dexmedetomidine in varying doses (for example, 0.5 μg/kg) or combined with lower induction doses of propofol or opioids to balance efficacy with cardiovascular safety. Comparative trials incorporating other adjuncts such as esmolol or magnesium sulfate could identify optimal multimodal regimens. Further evaluation in cardiac, neurosurgical, and elderly populations would enhance the generalizability of results.

CONCLUSION

The present prospective, randomized study comparing intravenous dexmedetomidine and lignocaine for attenuation of the cardiovascular stress response to laryngoscopy and endotracheal intubation demonstrated that both agents effectively blunted hemodynamic fluctuations, but dexmedetomidine was significantly superior in maintaining stable peri-intubation heart rate and arterial pressure. Dexmedetomidine 1 μg/kg administered slowly before induction provided consistent attenuation of sympathetic stimulation, resulting in lower post-intubation surges in heart rate and blood pressure compared with lignocaine 1.5 mg/kg.

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