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Comparative Study Between Intravenous Lignocaine & Intravenous Dexmedetomidine For Attenuation of Hemodynamic Response to Laryngoscopy and Endotracheal Intubation

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Abstract

Background:
Laryngoscopy and endotracheal intubation are essential components of General Anaesthesia but are associated with a sympathetic surge, resulting in significant hemodynamic changes such as tachycardia and hypertension. These transient changes, although often well tolerated in healthy individuals, may pose serious risks in patients with cardiovascular or cerebrovascular comorbidities. Various pharmacological agents have been used to blunt this response, among which dexmedetomidine and lignocaine are frequently employed. This study aimed to compare the efficacy of intravenous lignocaine and intravenous dexmedetomidine in attenuating the hemodynamic responses to laryngoscopy and endotracheal intubation in adult patients undergoing elective surgeries under General Anaesthesia.

Materials and Methods:
A prospective, randomized, clinical study was conducted at ICARE Institute of Medical Sciences and Research & Dr. B. C. Roy Hospital, Haldia, West Bengal, over a one-year period from July 2023 to June 2024. A total of 100 patients (ASA I & II), aged 18–60 years, were randomly divided into two groups: Group L received intravenous lignocaine (1.5 mg/kg, 3 minutes before intubation) while Group D received intravenous dexmedetomidine (1 µg/kg over 10 minutes before induction). Hemodynamic parameters such as heart rate (HR), systolic blood pressure (SBP), and mean arterial pressure (MAP) were recorded at baseline, induction, intubation, and at 1, 3, 5, and 10 minutes post-intubation.

Results:
Group D (Dexmedetomidine) showed a statistically significant attenuation of heart rate, systolic blood pressure and mean arterial pressure at induction and during the first five minutes post-intubation compared to Group L (Lignocaine), with p-values <0.001 across most time points. At intubation, the mean heart rate in Group D was 72.58 bpm versus 84.28 bpm in Group L, and mean SBP was 104.70 mmHg in Group D versus 120.16 mmHg in Group L. MAP values followed a similar trend, with Group D demonstrating consistently lower readings. No severe bradycardia, hypotension, or delayed

emergence was noted in either group. The dexmedetomidine group also showed a smoother anesthetic induction and better peri-intubation cardiovascular stability, reinforcing its efficacy and safety profile in mitigating intubation-induced stress responses.

Conclusion:

Dexmedetomidine is significantly more effective than lignocaine in attenuating the hemodynamic response to laryngoscopy and intubation, making it a safer and more reliable choice, especially in patients at cardiovascular risk.

Keywords:

Dexmedetomidine, Lignocaine, Hemodynamic response, Laryngoscopy, Endotracheal intubation, Blood pressure, Heart rate etc.

Introduction

Securing the airway through direct laryngoscopy and endotracheal intubation is a fundamental component of General Anaesthesia. However, these procedures elicit a well-documented sympathetic response, resulting in transient but significant hemodynamic changes such as tachycardia and hypertension [1]. These cardiovascular alterations are primarily mediated through reflex sympathoadrenal stimulation due to the mechanical stimulation of the oropharyngolaryngeal structures [2]. While these responses are often well tolerated by healthy individuals, they can pose substantial risks in patients with pre-existing coronary artery disease, cerebrovascular disorders, or intracranial pathology and hyperactive airway, where even brief surges in blood pressure or heart rate may precipitate myocardial ischemia, arrhythmias or cerebral haemorrhage and may often precipitate bronchospasm [3,4].

The hemodynamic response to laryngoscopy typically peaks within the first minute post-intubation and normalizes within 5–10 minutes [5]. Multiple strategies have been explored to mitigate this response, including the use of deep anaesthesia, high-dose opioids, beta blockers, vasodilators, local anaesthetics, and alpha-2 adrenergic agonists [6–9]. Among these, lignocaine and dexmedetomidine are two agents frequently used due to their ease of administration and established pharmacodynamic profiles.

Lignocaine is an amide-type local anaesthetic that also possesses antiarrhythmic properties. When administered intravenously, it stabilizes neuronal membranes and blunts the cardiovascular response to noxious stimuli [10]. Although it is widely used for this purpose, its efficacy in consistently suppressing the pressor response has shown variability across studies [11].

Dexmedetomidine is a highly selective alpha-2 adrenergic receptor agonist with sedative, analgesic, and sympatholytic properties. It attenuates sympathetic outflow by acting on central presynaptic receptors, resulting in decreased norepinephrine release and more stable hemodynamics during intubation [12,13]. Unlike many other agents, it does not cause significant respiratory depression, making it a favorable option in various anaesthetic settings [14].

In light of these considerations, this study was conducted to compare the effectiveness of intravenous lignocaine (1.5 mg/kg) versus intravenous dexmedetomidine (1 µg/kg) in attenuating the hemodynamic stress response to laryngoscopy and endotracheal intubation in adult patients undergoing elective surgery under General Anaesthesia.

Materials and Methods

This prospective, randomized, clinical study was conducted in the Department of Anaesthesiology at ICARE Institute of Medical Sciences and Research and Dr. B. C. Roy Hospital, Haldia, West Bengal, after obtaining approval from the Institutional Ethics Committee (dated 8th June 2023). The study was carried out over a one-year period from July 2023 to June 2024 and included a total of 100 patients.

Eligible participants were adult patients between 18 and 60 years of age, belonging to American Society of Anesthesiologists (ASA) physical status I and II, who were scheduled for elective surgeries

under General Anaesthesia. Written informed consent was obtained from all participants after a detailed explanation of the study protocol. Patients were excluded if they had ASA grade III or higher, a history of asthma, chronic obstructive pulmonary disease, hypertension, or any known hypersensitivity to the study drugs. Additional exclusion criteria included obesity and short neck relating to difficult airway (Mallampati class >2), pregnancy, hepatic or renal impairment and prior head & neck or oral surgeries.

The enrolled patients were randomly allocated into two equal groups of 50 each using computer-generated random numbers. Group L received 100 ml of normal saline infused over 10 minutes, followed by 1.5 mg/kg of intravenous lignocaine (made up to 5.0 ml with saline), administered 3 minutes before laryngoscopy and intubation. Group D received intravenous dexmedetomidine at a dose of 1 μg/kg diluted in 100 ml of normal saline, administered over 10 minutes, completed 10 minutes prior to induction and 5.0 ml saline was administered IV 3 mins before intubation.

On the day of surgery, standard preoperative fasting guidelines were followed, and all patients underwent pre-anaesthetic evaluation including laboratory investigations (complete blood count, blood glucose, liver and kidney function tests), electrocardiogram, and chest X-ray. In the operating room, routine monitors were attached (non-invasive blood pressure, pulse oximetry, electrocardiography, and end-tidal CO_2), and intravenous access was secured. Baseline vital parameters including heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, respiratory rate, and SpO_2 were recorded.

All patients received intravenous metoclopramide 10 mg one hour before surgery. Anaesthesia was induced with intravenous propofol at 1.5 mg/kg, followed by intravenous succinylcholine 2 mg/kg to facilitate tracheal intubation. Laryngoscopy and endotracheal intubation were performed using a Macintosh laryngoscope and appropriately sized cuffed endotracheal tube. No surgical stimulus or additional analgesics were administered for the first 10 minutes following intubation to allow for accurate hemodynamic assessment. Anaesthesia was maintained with 33% oxygen, 66% nitrous oxide, isoflurane, and intermittent doses of vecuronium. Fentanyl (1 μg/kg) was administered 10 minutes after the study period. At the end of the procedure, neuromuscular blockade was reversed with neostigmine (0.05 mg/kg) and glycopyrrolate (0.008 mg/kg), and patients were extubated once they regained adequate respiratory effort and consciousness.

Hemodynamic parameters including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and SpO_2 were recorded at the following time points: baseline, pre-induction, induction, at intubation, and at 1, 3, 5, and 10 minutes post-intubation.

Data were analyzed using SPSS software version 25. Continuous variables were expressed as mean ± standard deviation and compared using the Mann–Whitney U test due to non-normal distribution. Categorical variables were compared using Pearson's Chi-square test or Fisher's exact test, as appropriate. A p-value of <0.05 was considered statistically significant.

Results

A total of 100 patients were enrolled in the study and randomly assigned into two equal groups: Group D (Dexmedetomidine) and Group L (Lignocaine), with 50 patients in each.

The mean age in Group D (Dexmedetomidine) was 41.24 ± 12.98 years, while in Group L (Lignocaine), it was 42.48 ± 12.84 years. In terms of sex distribution, 70% of Group D and 68% of Group L were female. The male proportions were 30% in Group D and 32% in Group L. Demographic variables such as age and sex were comparable between the groups and did not show any statistically significant differences. The demographic characteristics were well matched, confirming that any observed differences in hemodynamic response can be attributed to the drugs administered, not to age or sex.

Table 1: Comparison of Age and Sex Between the Two Groups

Variable	Group D (n=50)	Group L (n=50)	p-value
Age (years), mean ± SD	41.24 ± 12.98	42.48 ± 12.84	0.537
Male, n (%)	15 (30%)	16 (32%)	0.829
Female, n (%)	35 (70%)	34 (68%)	

Table 2 and Figure 1 show heart rate trends over time. At baseline and pre-induction, heart rates in both groups were not significantly different ($p > 0.05$). However, from the point of induction onward, Group D showed consistently and significantly lower heart rates compared to Group L. At intubation, Group D had a mean HR of 72.58 bpm, while Group L had 84.28 bpm ($p < 0.001$). At 1, 3, and 5 minutes post-intubation, Group D maintained HRs around 68.8, 64.2, and 59.5 bpm, respectively, compared to 82.5, 81.0, and 80.3 bpm in Group L — all differences highly significant ($p < 0.001$). By 10 minutes post-intubation, HR values converged in both groups ($p = 0.975$), indicating recovery towards baseline. Thus Group D exhibits a consistent decline in heart rate following induction, reaching the lowest point at 5 minutes post-intubation, before partially recovering by the 10-minute mark. In contrast, Group L maintains elevated heart rates throughout, with a slight decline after intubation but remaining significantly higher than Group D.

Table 2: Heart Rate (beats per minute) at Various Time Intervals

Time Point	Group D (Mean ± SD)	Group L (Mean ± SD)	p-value
Baseline	86.06 ± 10.26	81.76 ± 14.42	0.072
Pre-induction	76.86 ± 9.30	81.56 ± 14.43	0.161
Induction	72.44 ± 9.35	84.60 ± 15.72	<0.001
Intubation	72.58 ± 10.83	84.28 ± 15.50	<0.001
1 min post	68.80 ± 9.76	82.54 ± 15.00	<0.001
3 min post	64.22 ± 9.04	81.00 ± 14.60	<0.001
5 min post	59.56 ± 7.88	80.38 ± 14.60	<0.001
10 min post	78.10 ± 8.31	79.10 ± 14.29	0.975

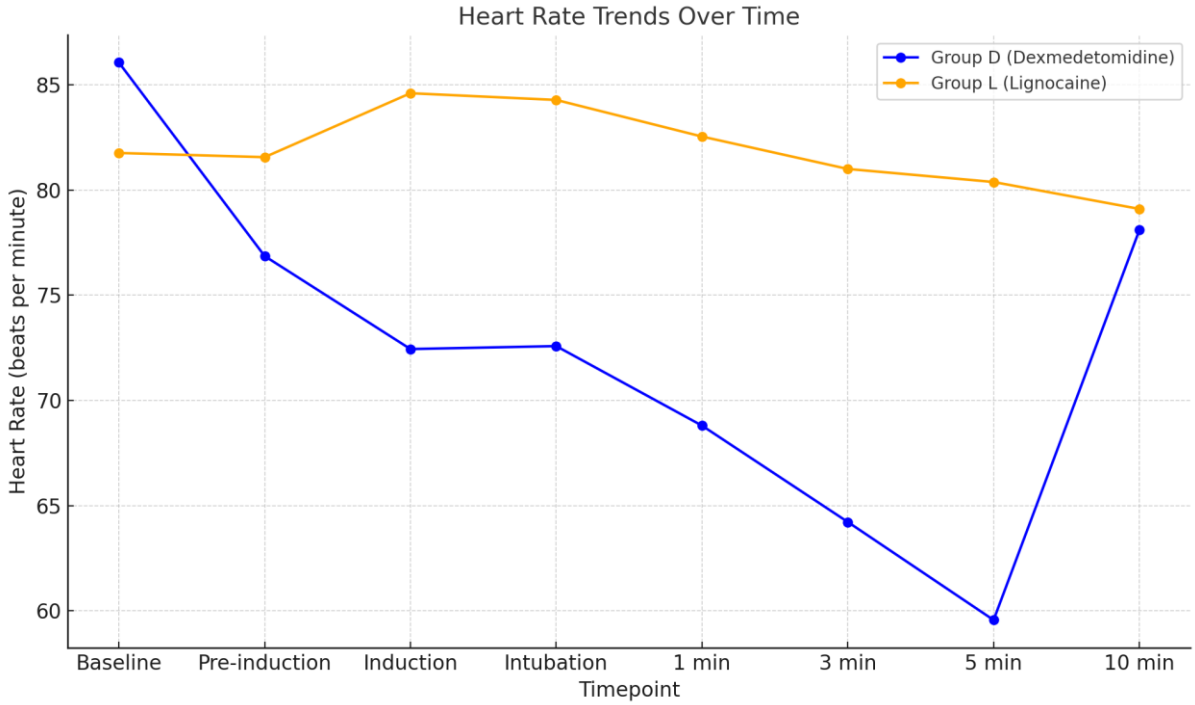

Figure 1: Comparison of heart rate changes over time in Group D (Dexmedetomidine) and Group L (Lignocaine).

Table 3 compares systolic blood pressure between the two groups. Baseline SBP values were slightly higher in Group D (119.48 mmHg) than Group L (115.76 mmHg), but the difference was not statistically significant ($p = 0.069$). At induction, Group D maintained a stable SBP (104.20 mmHg), whereas Group L's SBP rose to 112.24 mmHg ($p < 0.001$). At intubation, Group D recorded an SBP of 104.70 mmHg, while Group L spiked to 120.16 mmHg, indicating a significant pressor response in the lignocaine group ($p < 0.001$). Similar patterns continued at 1, 3, and 5 minutes, where Group D maintained significantly lower SBP than Group L. By 10 minutes, SBP values between both groups were comparable again, showing return to baseline hemodynamics. Dexmedetomidine was significantly more effective than lignocaine in blunting the systolic pressor response induced by laryngoscopy and intubation.

Table 3: Systolic Blood Pressure (SBP in mmHg)

Time Point	Group D (Mean ± SD)	Group L (Mean ± SD)	p-value
Baseline	119.48 ± 12.48	115.76 ± 16.01	0.069
Pre-induction	104.86 ± 12.09	106.12 ± 12.21	0.926
Induction	104.20 ± 9.64	112.24 ± 11.85	<0.001
Intubation	104.70 ± 10.62	120.16 ± 18.60	<0.001
1 min post	102.10 ± 18.06	111.66 ± 11.32	<0.001
3 min post	96.54 ± 11.27	103.14 ± 11.90	<0.001
5 min post	93.18 ± 9.40	96.22 ± 9.25	0.001
10 min post	95.72 ± 12.39	96.10 ± 8.71	0.302

Table 4 highlights mean arterial pressure (MAP) variations. Baseline MAP was statistically similar in both groups (92.04 mmHg in Group D vs. 90.12 mmHg in Group L, $p = 0.488$). At intubation, Group D had a MAP of 85.70 mmHg, whereas Group L had a significantly elevated MAP of 98.62 mmHg ($p < 0.001$). At 1, 3, and 5 minutes post-intubation, MAP in Group D continued to decrease progressively (82.60, 78.30, and 75.80 mmHg, respectively), while Group L exhibited higher MAP values (94.14, 90.06, and 88.42 mmHg, respectively). All differences were statistically significant ($p < 0.001$).

Table 4: Mean Arterial Pressure (MAP in mmHg)

Time Point	Group D (Mean ± SD)	Group L (Mean ± SD)	p-value
Baseline	92.04 ± 10.45	90.12 ± 12.84	0.488
Intubation	85.70 ± 8.90	98.62 ± 10.14	<0.001
1 min post	82.60 ± 10.26	94.14 ± 9.65	<0.001
3 min post	78.30 ± 9.12	90.06 ± 9.88	<0.001
5 min post	75.80 ± 8.67	88.42 ± 9.02	<0.001

Discussion:

Laryngoscopy and endotracheal intubation, although routine, are among the most stimulating procedures in anaesthetic practice, provoking a surge in sympathetic outflow that can lead to significant tachycardia and hypertension. This response, though brief, may result in myocardial ischemia, arrhythmias, or cerebrovascular complications, especially in high-risk patients [15,16]. Hence, blunting this hemodynamic response remains a critical objective in perioperative anaesthesia management.

In our study, intravenous dexmedetomidine at 1 μg/kg significantly attenuated the rise in heart rate, systolic blood pressure (SBP), and mean arterial pressure (MAP) from the time of induction to five minutes post-intubation, compared to lignocaine (1.5 mg/kg). These effects can be attributed to dexmedetomidine's selective α2-adrenergic receptor agonism, which reduces sympathetic tone and norepinephrine release, leading to a stable hemodynamic profile [17,18].

Our findings are consistent with results from numerous other studies. Bajwa et al. demonstrated that dexmedetomidine effectively reduces the sympathoadrenal stress response during laryngoscopy and

also provides perioperative analgesic benefits [19]. Patel et al. showed that pre-induction dexmedetomidine significantly reduced heart rate and MAP without inducing significant bradycardia or hypotension [20]. Similarly, Tanskanen et al. reported that dexmedetomidine premedication resulted in lower intraoperative BP and HR values and improved recovery scores [21].

In contrast, lignocaine, although long used for its membrane-stabilizing and sodium-channel blocking effects, has shown variable efficacy in attenuating intubation responses. In our study, while lignocaine caused a modest reduction in hemodynamic values, it was clearly inferior to dexmedetomidine. This aligns with observations by Yavascaoglu et al., who concluded that lignocaine blunted but did not prevent the hypertensive and tachycardic responses to airway manipulation [22]. Other trials, such as that by Sharma et al., reported that lignocaine is only moderately effective, with outcomes depending heavily on timing and mode of administration [23].

Another study by Talke et al. confirmed that dexmedetomidine produces a dose-dependent attenuation of cardiovascular responses during surgery, without significant respiratory depression [24]. Moreover, Kumari et al. found that dexmedetomidine reduced intraoperative anesthetic and analgesic requirements while maintaining better control over peri-intubation hemodynamics [25]. Dexmedetomidine's unique profile — sedative, anxiolytic, analgesic, and sympatholytic — makes it particularly suitable for attenuating intubation responses in diverse surgical populations [26,27].

Interestingly, in our study, both groups returned to baseline hemodynamic values by 10 minutes post-intubation, suggesting that while the stress response is transient, the critical window for attenuation is the first 3–5 minutes post-laryngoscopy. Thus, dexmedetomidine's action is particularly well-suited for this timeframe. Furthermore, we observed no adverse effects such as severe bradycardia, hypotension, or delayed recovery, which are occasionally reported with dexmedetomidine at higher doses [28,29].

The study was strengthened by its randomized, comparative design and standardized protocols. However, limitations include its single-center scope, lack of plasma catecholamine measurements, and absence of long-term recovery or postoperative pain outcomes. Future studies should explore dexmedetomidine's effects on recovery profile, postoperative analgesia, and patient satisfaction.

In conclusion, dexmedetomidine (1 μg/kg IV) is significantly more effective than lignocaine (1.5 mg/kg IV) in attenuating the hemodynamic response to laryngoscopy and intubation. Its safety, reliability, and predictable effects make it an ideal choice for use in patients undergoing General Anaesthesia, especially in those with cardiovascular risks.

Conclusion:

This study demonstrates that intravenous dexmedetomidine at a dose of 1 μg/kg is significantly more effective than intravenous lignocaine 1.5 mg/kg in attenuating the hemodynamic stress response to laryngoscopy and endotracheal intubation. Dexmedetomidine consistently maintained lower heart rate, systolic blood pressure, and mean arterial pressure during the critical peri-intubation period, especially within the first 5 minutes, without any significant adverse effects. These findings highlight dexmedetomidine's superior sympatholytic properties and suggest its clinical utility in patients where hemodynamic stability is crucial. Therefore, dexmedetomidine may be considered a more reliable agent for blunting the cardiovascular responses to airway manipulation during General Anaesthesia.

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