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Research Article

Comparison of Combination of Low Dose Intravenous Dexmedetomidine and Lidocaine Infusion with Intravenous Lidocaine Infusion to Attenuate Haemodynamic Response to Laryngoscopy and Endotracheal Intubation in Adult Patients Undergoing General Anaesthesia

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ABSTRACT

Laryngoscopy and endotracheal intubation often trigger a transient activation of the sympathoadrenal system, which can be harmful—and occasionally life-threatening—in high-risk patients. This randomized study included 70 patients, aged 18 to 60 years, classified as ASA physical status I or II, who were scheduled for elective surgery under general anesthesia. Participants were divided into two equal groups at random: Group 1 received combined low-dose lignocaine and dexmedetomidine, while Group 2 received only standard-dose lignocaine.
In Group 1, preservative-free lignocaine (1 mg/kg) was infused over 3 minutes before induction, after 0.5 mcg/kg of intravenous dexmedetomidine (diluted in 0.9% normal saline) was administered over 10 minutes. Group 2 received 1.5 mg/kg of preservative-free intravenous lignocaine over 3 minutes, along with a 10-minute infusion of 0.9% normal saline as placebo. Hemodynamic parameters monitoring, which included heart rate (HR), systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP) were recorded during several perioperative intervals: upon arrival to the theatre, 2 and 5 minutes after receiving the drug, just before and after induction, and at 1, 3, 5, and 10 minutes post intubation. Results indicated that Group 1 sustained heart rate and blood pressure values below baseline during the entire 10-minute post intubation period. It is concluded that a low dose combination of dexmedetomidine 0.5 mcg/kg and lignocaine 1 mg/kg provided greater attenuation of the pressor response to laryngoscopy and intubation, without significant hemodynamic alterations, when compared to lignocaine 1.5 mg/kg alone during general anaesthesia.

Keywords*: Dexmedetomidine; Lignocaine; Direct laryngoscopy; Pressor response; General anaesthesia; Endotracheal intubation; Hemodynamic response; Alpha-2 agonist.*

INTRODUCTION

Securing the airway through laryngoscopy and endotracheal intubation is a key component of general anaesthesia. However, these procedures can provoke significant sympathetic stimulation, leading to haemodynamic changes such as tachycardia and hypertension, which may be hazardous in vulnerable patients.[1-2] This response is mediated by reflex activation of the vagus and glossopharyngeal nerves, resulting in elevated heart rate, blood pressure, and catecholamine levels, particularly norepinephrine. A wide range of pharmacological interventions have been used to attenuate this haemodynamic response including: opioids, beta-blockers, vasodilators, alpha-2 agonists and calcium channel blockers. Of these, dexmedetomidine, a selective alpha-2 agonist, has shown beneficial properties because of its sympatholytic and sedative properties. Intravenously administered local anaesthetic, such as lidocaine, has also been shown to attenuate the haemodynamic response. Although both the agents have been studied individually and in combination, there is paucity of

data on comparison between low dose dexmedetomidine with lidocaine and lidocaine alone. Therefore, this study was designed to evaluate the effectiveness of a low-dose dexmedetomidine-lidocaine combination versus standard-dose lidocaine alone in reducing the haemodynamic response to laryngoscopy and intubation in adults undergoing general anaesthesia.

MATERIALS AND METHOD

This prospective randomized study was conducted in a tertiary care teaching hospital of North India, over a period from 1st August 2022 to 29th February 2024. A total of 70 patients, classified as ASA grade I and II and scheduled for elective surgery under general anaesthesia, were included. Patients were randomly assigned into two groups of 35 each, using a computer-generated random number table. The inclusion criteria comprised adult patients aged between 18 and 60 years, classified as ASA grade I or II, and scheduled for elective surgeries under general anaesthesia. The exclusion criteria included patients with hypertension, a history of coronary artery disease, arrhythmias, or the presence of pacemakers. Patients with known allergies to dexmedetomidine or lidocaine, those on beta-blocker therapy, individuals undergoing emergency surgery, patients with a full stomach, and pregnant or paediatric patients were also excluded from the study.
All the patients underwent a detailed pre-anaesthetic evaluation, including medical history, physical examination, baseline investigations, and airway assessment. Written informed consent was obtained, and patients were randomly assigned to two groups of 35 each: Group 1 and Group 2. On arrival in the operating room, patient identity was confirmed, standard ASA monitors were applied, and an appropriate intravenous line was secured. Baseline parameters, including heart rate, systolic and diastolic blood pressure, and mean arterial pressures, were recorded prior to induction of general anaesthesia. Following documentation of these baseline values (T0), the assigned study drug infusion was commenced.

Group 1 patients received a combination of 0.5 mcg/kg of Intravenous dexmedetomidine diluted in 0.9 % normal saline as infusion over 10 minutes and 1 mg/kg of intravenous lidocaine (preservative free) diluted in 0.9% normal saline infusion over 3 minutes before induction of anaesthesia, while Group 2 patients received only 1.5mg/kg of intravenous lidocaine (preservative free) diluted in 0.9% normal saline as infusion over 3 minutes and intravenous 0.9% normal saline infusion over 10 minutes before induction of anaesthesia.

Following completion of the test drug infusion and recording of vital signs at predefined intervals, standard induction of general anaesthesia was carried out. Patients underwent preoxygenation for 3 minutes, followed by intravenous administration of midazolam (0.03 mg/kg), fentanyl (2 mcg/kg), propofol (2 mg/kg), and vecuronium bromide (0.1 mg/kg). After 3 minutes, tracheal intubation was performed under direct laryngoscopy using an appropriately sized cuffed endotracheal tube, with placement confirmed by bilateral equal breath sounds on auscultation. Mechanical ventilation was initiated to maintain normoxia and normocarbia, with supplemental doses of vecuronium bromide given as required to maintain muscle relaxation. Surgery commenced only 10 minutes after intubation. Vital parameters such as HR, SBP, DBP and MAP were recorded, at baseline, 2 and 5 minutes after study drug administration, after induction, 1, 3, 5 and 10 minutes after intubation.

At the end of the procedure, neuromuscular blockade was reversed with neostigmine (0.05 mg/kg) and glycopyrrolate (0.01 mg/kg). Standard extubation guidelines were followed and the patient was transferred to the recovery room for continued monitoring. Patients were monitored for adverse effects like Bradycardia and Hypotension. Bradycardia was defined if pulse rate was less than 50 beats per minute or if there was more than twenty percent decrease in heart rate from the baseline value. It was treated with intravenous inj atropine 0.6mg. Hypotension was defined if mean blood pressure decreased more than twenty percent from the baseline values. It was corrected with rapid intravenous fluid administration and intravenous Mephentermine 0.1mg/kg body weight if required.

Data analysis was conducted using SPSS for Windows, version 28.0 (IBM Corp., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as absolute numbers and percentages. Normality of continuous data was assessed prior to statistical testing. Normally distributed variables were compared using the unpaired t-test, while the Mann–Whitney U test was applied for non-normally distributed data. Categorical variables were analysed using the Chi-square test or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant.

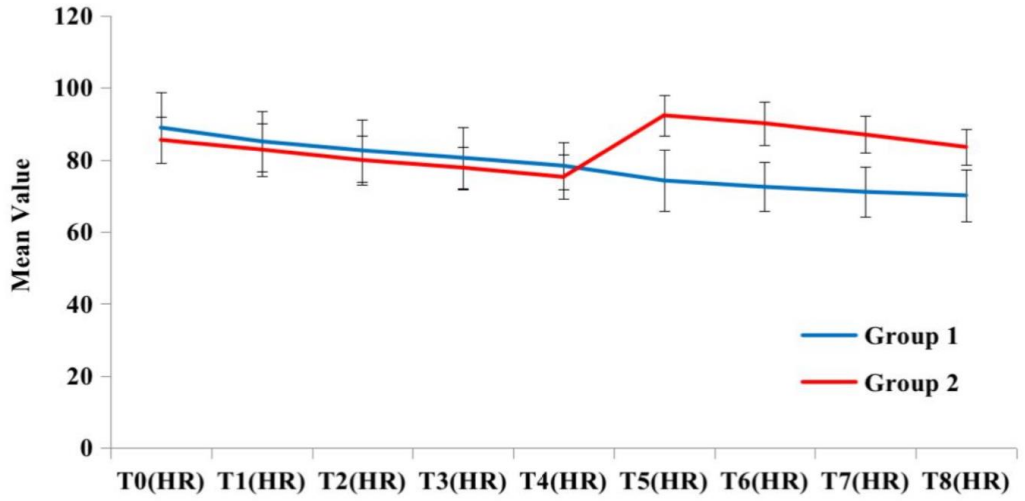
OBSERVATION AND RESULTS

The mean age of patients in Group 1 was 35.20 ± 11.74 years, while in Group 2 it was 37.49 ± 10.71 years. The difference in mean age between the two groups was not statistically significant ($p = 0.398$). It was also observed that there was no significant difference in gender distribution (p value 0.473) when patients under two groups were compared.

Table 1: Comparison of Heart Rate of the patients between the two groups

	Group 1	Group 2	p value
	Mean ± SD	Mean ± SD	
T0(HR)	88.94 ± 9.74	85.54 ± 6.36	0.129

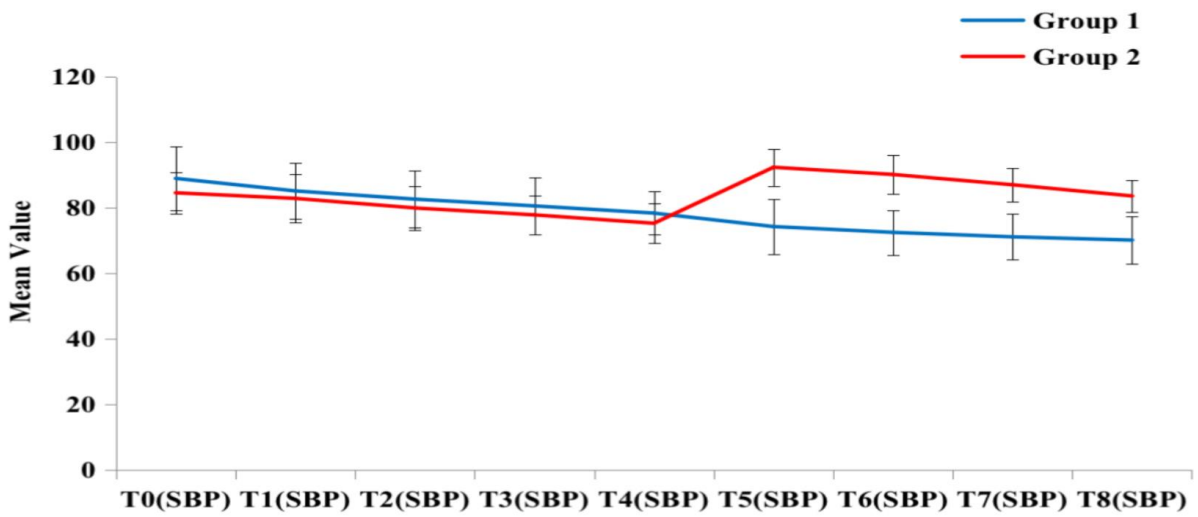
T1(HR)	85.11 ± 8.49	82.83 ± 7.32	0.232
T2(HR)	82.6 ± 8.65	79.91 ± 6.75	0.152
T3(HR)	80.54 ± 8.64	77.8 ± 5.85	0.125
T4(HR)	78.34 ± 6.61	75.23 ± 6.11	0.044*
T5(HR)	74.23 ± 8.49	92.34 ± 5.67	<0.001**
T6(HR)	72.46 ± 6.84	90.14 ± 5.93	<0.001**
T7(HR)	71.09 ± 6.93	87 ± 5.07	<0.001**
T8(HR)	70.09 ± 7.25	83.57 ± 4.87	<0.001**
*signifies significant p value<0.05			
**signifies highly significant p value<0.001			
Test used: Student's t test			



It was observed that under Group 1, mean HR at T0 was 88.94 ± 9.74, mean HR at T1 was 85.11 ± 8.49, mean HR at T2 was 82.6 ± 8.65, mean HR at T3 was 80.54 ± 8.64, men HR at T4 was 78.34 ± 6.61, mean HR at T5 was 74.23 ± 8.49, mean HR at T6 was 72.46 ± 6.84, mean HR at T7 was 71.09 ± 6.93 and mean HR at T8 was 70.09 ± 7.25 while under Group 2, mean HR at T0 was 85.54 ± 6.36, mean HR at T1 was 82.83 ± 7.32, mean HR at T2 was 79.91 ± 6.75, mean HR at T3 was 77.8 ± 5.85, mean HR at T4 was 75.23 ± 6.11, mean HR at T5 was 92.34 ± 5.67, mean HR at T6 was 90.14 ± 5.93, mean HR at T7 was 87 ± 5.07 and mean HR at T8 was 83.57 ± 4.87. Further, it was observed that there was no significant difference in mean HR at T0 to T3 while a significant difference was observed at other time intervals (p value <0.001) when patients under two groups were compared.

Table 2: Comparison of Systolic Blood Pressure of the patients between the two groups

	Group 1	**Group 2**	**p value**
	Mean ± SD	**Mean ± SD**	
T0(SBP)	88.94 ± 9.74	84.54 ± 6.36	0.517
T1(SBP)	85.11 ± 8.49	82.83 ± 7.32	0.712
T2(SBP)	82.6 ± 8.65	79.91 ± 6.75	0.169
T3(SBP)	80.54 ± 8.64	77.8 ± 5.85	0.033*
T4(SBP)	78.34 ± 6.61	75.23 ± 6.11	0.011*
T5(SBP)	74.23 ± 8.49	92.34 ± 5.67	<0.001**
T6(SBP)	72.46 ± 6.84	90.14 ± 5.93	<0.001**
T7(SBP)	71.09 ± 6.93	87 ± 5.07	<0.001**
T8(SBP)	70.09 ± 7.25	83.57 ± 4.87	<0.001**
*signifies significant p value<0.05			
**signifies highly significant p value<0.001			
Test used: Student's t test			



It was observed that under Group 1, mean SBP at T0 was 88.94 ± 9.74, mean SBP at T1 was 85.11 ± 8.49, mean SBP at T2 was 82.6 ± 8.65, mean SBP at T3 was 80.54 ± 8.64, men SBP at T4 was 78.34 ± 6.61, mean SBP at T5 was 74.23 ± 8.49, mean SBP at T6 was 72.46 ± 6.84, mean SBP at T7 was 71.09 ± 6.93 and mean SBP at T8 was 70.09 ± 7.25 while under Group 2, mean SBP at T0 was 84.54 ± 6.36, mean SBP at T1 was 82.83 ± 7.32, mean SBP at T2 was 79.91 ± 6.75, mean SBP at T3 was 77.8 ± 5.85, mean SBP at T4 was 75.23 ± 6.11, mean SBP at T5 was 92.34 ± 5.67, mean SBP at T6 was 90.14 ± 5.93, mean SBP at T7 was 87 ± 5.07 and mean SBP at T8 was 83.57 ± 4.87. Furthermore, when patients in the two groups were examined, it was shown that there was no significant difference in mean SBP from T0 to T3, although there was at other times (p value <0.001).

In Group 1, the mean diastolic blood pressure (DBP) was 79.91 ± 6.75 at T0, 75.46 ± 7.56 at T1, 72.20 ± 7.01 at T2, 69.63 ± 6.27 at T3, 68.11 ± 7.11 at T4, 62.80 ± 5.38 at T5, 59.91 ± 4.46 at T6, 60.29 ± 5.72 at T7, and 60.91 ± 3.99 at T8. In Group 2, the corresponding values were 80.26 ± 4.90, 77.46 ± 4.03, 74.74 ± 4.29, 72.34 ± 3.89, 71.43 ± 3.89, 85.37 ± 4.80, 82.40 ± 4.95, 79.46 ± 4.26, and 76.86 ± 4.03. No statistically significant difference in mean DBP was found between the two groups from T0 to T4. However, the difference was statistically significant at T5, T6, T7, and T8 ($p < 0.001$).

In Group 1, the mean mean arterial pressure (MBP) was 95.03 ± 5.80 at T0, 91.71 ± 6.96 at T1, 84.37 ± 8.24 at T2, 84.11 ± 5.26 at T3, 83.11 ± 7.67 at T4, 76.43 ± 4.24 at T5, 74.03 ± 3.43 at T6, 74.40 ± 5.87 at T7, and 73.94 ± 2.96 at T8. In Group 2, corresponding values were 94.46 ± 4.13, 92.14 ± 4.16, 90.97 ± 4.82, 87.06 ± 3.70, 85.66 ± 3.08, 101.54 ± 4.23, 98.23 ± 4.15, 95.66 ± 3.67, and 92.46 ± 3.31, respectively. No statistically significant difference in MBP was observed between the two groups at T0, T1, T2, and T4. However, the difference was statistically significant at T3, T5, T6, T7, and T8 ($p < 0.001$).

DISCUSSION

Laryngoscopy and tracheal intubation are routine procedures for most patients undergoing surgery under general anaesthesia. However, stimulation of laryngeal and tracheal structures during these maneuvers triggers a reflex sympathetic surge, leading to elevations in blood pressure, heart rate, and serum catecholamine levels. Various pharmacological and non-pharmacological strategies have been employed to attenuate this haemodynamic response. Pharmacological options include inhalational agents, local anaesthetics (topical and intravenous), calcium channel blockers, opioids, and vasodilators, though none have consistently proven to be ideal. Dexmedetomidine [6], owing to its sedative, analgesic, amnestic, and neuroprotective properties, is commonly used as a premedication in such situations. Lidocaine also demonstrates sedative, analgesic, and neuroprotective effects, making it another useful adjuvant. Both agents are known to blunt the haemodynamic response to laryngoscopy and intubation.

Dexmedetomidine acts primarily through alpha-2A adrenoceptor activation in the locus coeruleus, inhibiting norepinephrine release and producing sedation, hypnosis, and analgesia. Stimulation of these receptors also dampens the descending nociceptive pathways, while postsynaptic activation in the CNS reduces sympathetic outflow, causing hypotension and bradycardia.

Lignocaine acts by binding to specific sites within-gated sodium ion channels. This binding effectively blocks the flow of sodium ions, thereby reducing the excitability of neuronal, cardiac, or central nervous system tissue. This results in direct myocardial depression, peripheral vasodilation, suppression of cough and strain during tracheal manipulation, inhibition of afferent C-fibre activity from the larynx, and possible central nervous system action that enhances anaesthetic depth.

In a study by **Dube S et al (2021)** [7], Baseline HR and SBP were comparable across groups. After study drug administration, HR and BP remained stable in Group L, decreased significantly in Group D, and moderately in Group DL. Post-induction, Group L showed marked increases in HR and BP after laryngoscopy, persisting above baseline, while Groups D and DL remained below baseline with fewer fluctuations. Bradycardia occurred in 6 patients in Group D, and hypotension in 3, both absent in Group DL. DBP and MAP trends mirrored SBP changes.Our findings align with **Mehta et al. (2023)**[8], who studied 150 ASA I patients to compare lignocaine, dexmedetomidine, and their combination for attenuating the intubation response. They reported stable HR in the lignocaine group, significant HR reduction in dexmedetomidine and combination groups ($p < 0.001$), with the effect persisting post-induction. The lignocaine group showed a mild post-intubation HR rise, while the combination group maintained HR below baseline without bradycardia or tachycardia.In a comparative study between doses of 1.0 μg/kg and 0.5 μg/kg of dexmedetomidine, **Khan et al** [9] found that the higher dose was associated with a higher incidence of hypotension and bradycardia. **Sulaiman et al** [10] also reported similar findings when assessing the effects of a single low dose of dexmedetomidine (0.5 mcg/kg, administered via slow IV infusion over 10 minutes) in patients with coronary artery disease undergoing off-pump coronary artery bypass grafting. In our current study, the administration of smaller doses of dexmedetomidine (0.5 mcg/kg) was well tolerated, with no serious side effects or adverse reactions noted. These results are in line with previous studies indicating significantly lower increases in systolic and diastolic blood pressure, as well as heart rate, following laryngoscopy and intubation with lower doses of dexmedetomidine [11]. There are many studies that have shown that lignocaine alone may be insufficient to suppress the intubation response. Our findings support this, demonstrating that a combination of low-dose lignocaine (1.0 mg/kg) and dexmedetomidine (0.50 mcg/kg) produced minimal cardiovascular effects while effectively preventing the haemodynamic response to laryngoscopy and tracheal intubation.

CONCLUSION

The present study compared the efficacy of a low-dose combination of dexmedetomidine and lidocaine infusion versus standard dose lidocaine infusion alone in adult patients undergoing laryngoscopy and endotracheal intubation under general anaesthesia. While both groups effectively stabilised hemodynamic parameters, a significant difference was noted in favour of the combined low-dose dexmedetomidine and lidocaine group compared to standard dose lidocaine infusion group. Thus, this study concludes that, in comparison to pre-induction administration of 1.5 mg/kg standard dose intravenous lidocaine infusion alone, the combination of 0.5 mcg/kg intravenous dexmedetomidine infusion and 1 mg/kg intravenous lidocaine infusion effectively attenuates the hemodynamic responses during laryngoscopy and intubation, without causing any adverse effects.

SOURCE OF FUNDING

None.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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