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Research Article

Comparison Between Nebulised Dexmedetomidine and Nebulised Lignocaine in Attenuation of Haemodynamic Stress Response to Intubation – A Double Blinded Randomised Study

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ABSTRACT

Background: Laryngoscopy and endotracheal intubation are associated with a significant haemodynamic stress response characterized by tachycardia and hypertension, which may lead to adverse cardiovascular events. Various pharmacological agents have been used to attenuate this response, among which dexmedetomidine and lignocaine are commonly studied. Nebulisation offers a non-invasive route of drug administration with improved patient compliance and minimal systemic side effects.

Objectives: To compare the efficacy of nebulised dexmedetomidine and nebulised lignocaine in attenuating the haemodynamic stress response to laryngoscopy and endotracheal intubation, with primary focus on mean arterial pressure at 5 minutes and secondary parameters including heart rate, systolic blood pressure, and diastolic blood pressure at various time intervals.

Methods: This randomized, double-blind, interventional study was conducted in 112 patients undergoing elective surgeries under general anaesthesia. Patients were randomly allocated into two groups of 56 each to receive either nebulised dexmedetomidine (1 µg/kg) or nebulised 4% lignocaine (3 mg/kg) prior to induction. Haemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded at baseline, prior to intubation, and at 0, 1, 3, 5, and 10 minutes following intubation. Data were analysed using unpaired t-test, and $p < 0.05$ was considered statistically significant.

Results: Baseline parameters were comparable between the two groups ($p > 0.05$). Mean arterial pressure at 5 minutes was significantly lower in the dexmedetomidine group (91.0 ± 7.9 mmHg) compared to the lignocaine group (98.0 ± 8.5 mmHg) ($p < 0.001$). Heart rate showed significant reduction in the dexmedetomidine group from 1 minute onwards ($p < 0.05$). Systolic and diastolic blood pressures were significantly lower in the dexmedetomidine group at all post-intubation time points ($p < 0.01$ and $p < 0.001$ respectively). Overall, dexmedetomidine demonstrated superior attenuation of haemodynamic response compared to lignocaine.

Conclusion: Nebulised dexmedetomidine is more effective than nebulised lignocaine in attenuating haemodynamic stress response to laryngoscopy and endotracheal intubation, providing better and sustained haemodynamic stability. It represents a safe and effective non-invasive premedication option in anaesthetic practice.

Keywords: *Dexmedetomidine, Lignocaine, Nebulisation, Laryngoscopy, Endotracheal intubation, Haemodynamic stress response, Mean arterial pressure, Heart rate, General anaesthesia.*

INTRODUCTION

Laryngoscopy and endotracheal intubation are indispensable procedures in the administration of general anaesthesia but are associated with a significant haemodynamic stress response. This response is characterized by transient increases in heart rate, systolic and diastolic blood pressure, and mean arterial pressure due to sympathetic stimulation and catecholamine release. Although short-lived in healthy individuals, this response may lead to serious complications such as myocardial ischemia, arrhythmias, and cerebrovascular events, especially in patients with underlying comorbidities [1]. The magnitude of this haemodynamic response depends on several factors including the depth of anaesthesia, duration of laryngoscopy, and patient-related variables. Studies have shown that the pressor response typically peaks within the first minute after intubation and may persist for up to 10 minutes, necessitating effective attenuation strategies during this critical period [2].

Various pharmacological agents such as opioids, beta-blockers, vasodilators, and local anaesthetics have been used to attenuate this response. However, these agents are often associated with limitations such as respiratory depression, delayed recovery, hypotension, or incomplete suppression of haemodynamic changes. Hence, there is a continuous search for safer and more effective alternatives [3].

Lignocaine is a commonly used local anaesthetic that attenuates airway reflexes by blocking sodium channels and stabilizing neuronal membranes. Nebulised lignocaine provides topical anaesthesia of the airway, reducing the afferent stimuli during laryngoscopy and intubation. It has been shown to moderately reduce the rise in heart rate and blood pressure, although its effect may be short-lasting and less pronounced [4].

Dexmedetomidine, a highly selective alpha-2 adrenergic receptor agonist, has gained significant attention in anaesthetic practice due to its sedative, analgesic, and sympatholytic properties. It reduces sympathetic outflow and plasma catecholamine levels, thereby effectively attenuating haemodynamic responses without causing significant respiratory depression [5].

Recent advancements have explored non-invasive routes of drug administration such as nebulisation, which improves patient compliance and allows gradual systemic absorption through respiratory mucosa. Nebulised dexmedetomidine has been shown to produce stable haemodynamic effects with minimal side effects and better tolerability [6].

Comparative studies have demonstrated that dexmedetomidine provides superior attenuation of haemodynamic responses compared to traditional agents. It significantly reduces heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure following intubation, particularly in the early post-intubation period [7].

In contrast, while nebulised lignocaine is effective in suppressing airway reflexes, its haemodynamic attenuation is less consistent and may not provide sustained control over the stress response. This difference is attributed to the central sympatholytic action of dexmedetomidine compared to the peripheral action of lignocaine [8].

Several randomized controlled trials have highlighted the efficacy of dexmedetomidine in improving perioperative haemodynamic stability and reducing anaesthetic requirements. Its favourable pharmacological profile makes it a promising agent for attenuating intubation-related stress responses [9].

Furthermore, studies conducted in recent years have emphasized the role of nebulised dexmedetomidine as a safe and effective premedication technique, offering comparable or superior results to intravenous administration with fewer systemic side effects [10].

However, despite growing evidence, there remains limited literature directly comparing nebulised dexmedetomidine and nebulised lignocaine in a double-blinded randomized setting. Therefore, this study was undertaken to evaluate and compare the efficacy of these two agents in attenuating the haemodynamic stress response to laryngoscopy and endotracheal intubation, focusing on changes in mean arterial pressure, heart rate, systolic blood pressure, and diastolic blood pressure at predefined time intervals [11].

METHODOLOGY

This study was conducted as a single-center, randomized, double-blind, active-controlled interventional study with parallel group assignment in the operating theatre of the Department of Anaesthesia at Krishna Institute of Medical Sciences Ltd., Nellore, Andhra Pradesh, after obtaining approval from the Scientific Committee and Institutional Ethics Committee. The study was carried out over a period of 18 months from September 2022 to February 2024. The study population consisted of patients undergoing elective surgical procedures under general anaesthesia. A total of 112 patients were included in the study, with 56 patients in each group, and the sample size was calculated based on previously reported mean arterial pressure values at 5 minutes following intubation, considering 80% power and 5% level of significance.

Patients aged between 18 and 60 years, belonging to ASA physical status I and II and Mallampati class I and II, who provided informed consent were included in the study. Patients with a history of allergy to the study drugs, cerebrovascular disease, bronchial asthma, ischemic heart disease, seizure disorders, uncontrolled hypertension or diabetes mellitus, difficult airway, altered coagulation profile, renal or hepatic dysfunction, and those receiving antidepressants, antipsychotics, anxiolytics, or anticonvulsants were excluded. Eligible patients were recruited after detailed explanation of the study using a patient information sheet in their native language, and written informed consent was obtained.

Randomization was performed using a simple random number generator in Microsoft Excel (2019 version), and patients were allocated in a 1:1 ratio to either the dexmedetomidine group or the lignocaine group. Allocation concealment was ensured using preloaded syringes labeled with number codes, and both the investigator and the patient were blinded to the group allocation. Patients with odd numbers were assigned to the dexmedetomidine group, while those with even numbers were assigned to the lignocaine group.

A preoperative evaluation including detailed history and clinical examination was conducted a day prior to surgery. Patients were then administered the study drug via nebulisation using a face mask with 100% oxygen at a flow rate of 6 L/min for 10 minutes in a sitting position. The dexmedetomidine group received 1 µg/kg dexmedetomidine diluted to a total volume of 4 ml with normal saline, whereas the lignocaine group received 4% lignocaine at a dose of 3 mg/kg. All patients were premedicated with glycopyrrolate (6 µg/kg), midazolam (0.02 mg/kg), and fentanyl (2 µg/kg). Induction of anaesthesia was achieved using propofol (2 mg/kg) titrated to loss of verbal response, followed by rocuronium (0.6 mg/kg) to facilitate endotracheal intubation. Direct laryngoscopy was performed after 3 minutes of ventilation, and intubation was carried out using an appropriately sized endotracheal tube. Anaesthesia was maintained with nitrous oxide, oxygen, and sevoflurane. At the end of surgery, neuromuscular blockade was reversed with neostigmine (0.05 mg/kg) and glycopyrrolate (6 µg/kg), and patients were extubated after fulfilling standard extubation criteria and shifted to the surgical intensive care unit for observation.

Baseline haemodynamic parameters including heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were recorded before nebulisation. These parameters were again recorded prior to intubation, immediately after intubation (0 minute), and at 1 minute, 3 minutes, 5 minutes, and 10 minutes following intubation. The primary outcome measure was mean arterial pressure at 5 minutes following intubation, calculated using the formula MAP = DBP + 1/3 (SBP − DBP). Secondary outcome measures included heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure at other predefined time intervals.

All collected data were entered into Microsoft Excel and analyzed using SPSS and R software. Continuous variables were expressed as mean ± standard deviation. Baseline characteristics between the two groups were compared to ensure adequacy of randomization. Unpaired t-test was used to compare mean arterial pressure at 5 minutes and other haemodynamic parameters at different time points between the two groups. A p-value of less than 0.05 was considered statistically significant. Graphical representations of the data were generated using ggplot.

A total of 112 patients were included in the study, with 56 patients each in the dexmedetomidine and lignocaine groups. Baseline characteristics including age, gender distribution, ASA physical status, BMI, and baseline haemodynamic parameters were comparable between the two groups, with no statistically significant difference ($p > 0.05$), indicating effective randomization. The primary outcome, mean arterial pressure (MAP) at 5 minutes following intubation, was significantly lower in the dexmedetomidine group (91.0 ± 7.9 mmHg) compared to the lignocaine group (98.0 ± 8.5 mmHg), with a statistically significant difference ($p < 0.001$).

RESULTS

Heart rate variations showed that there was no significant difference between the groups at baseline, after nebulisation, prior to intubation, and immediately after intubation ($p > 0.05$). However, from 1 minute onwards, the dexmedetomidine group demonstrated significantly lower heart rate compared to the lignocaine group, with values of 94 ± 11 vs 100 ± 13 at 1 minute ($p = 0.01$), 89 ± 11 vs 97 ± 13 at 3 minutes ($p < 0.001$), 85 ± 11 vs 94 ± 13 at 5 minutes ($p < 0.001$), and 86 ± 11 vs 95 ± 13 at 10 minutes ($p < 0.001$).

Similarly, systolic blood pressure (SBP) was comparable between the two groups at baseline and before intubation ($p > 0.05$), but showed a significantly higher rise in the lignocaine group following intubation. At 0 minute, SBP was 130.9 ± 11.3 mmHg in the dexmedetomidine group compared to 141.9 ± 10.5 mmHg in the lignocaine group ($p < 0.01$). This difference remained statistically significant at 1, 3, 5, and 10 minutes post-intubation ($p < 0.01$ for all time points).

Diastolic blood pressure (DBP) also demonstrated a similar trend, with no significant difference at baseline and prior to intubation. However, following intubation, DBP was significantly lower in the dexmedetomidine group at all time points, with values of 79.2 ± 7.4 vs 85.7 ± 8.1 at 0 minute, 79.1 ± 7.5 vs 85.8 ± 8.9 at 1 minute, 77.5 ± 8.0 vs 84.8 ± 9.3 at 3 minutes, 73.4 ± 8.1 vs 79.4 ± 9.2 at 5 minutes, and 71.7 ± 8.3 vs 78.2 ± 8.6 at 10 minutes, all showing highly significant differences ($p < 0.001$).

Overall, the results demonstrated that nebulised dexmedetomidine provided significantly better attenuation of haemodynamic stress response compared to nebulised lignocaine, with lower heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure at most post-intubation time points, particularly from 1 minute onwards.

TABLE 1: Baseline Characteristics and Primary Outcome Comparison

Variable	Dexmedetomidine (n=56)	Lignocaine (n=56)	Test Used	p-value	Significance
Age (years)	45.9 ± 10.24	43.2 ± 11.3	t-test	0.18	Not Significant
Male	25 (44.6%)	22 (39.3%)	Chi-square	0.56	Not Significant
ASA I	30 (53.6%)	33 (58.9%)	Chi-square	0.58	Not Significant
ASA II	26 (46.4%)	23 (41.1%)	—	—	—
BMI	25.2 ± 2.4	25.9 ± 2.4	t-test	0.21	Not Significant
HR (baseline)	83 ± 11	81 ± 10	t-test	0.43	Not Significant
SBP (baseline)	127 ± 11	124 ± 10	t-test	0.15	Not Significant
DBP (baseline)	76 ± 8	74 ± 8	t-test	0.27	Not Significant
MAP (baseline)	93 ± 8	91 ± 8	t-test	0.15	Not Significant
MAP at 5 min (Primary Outcome)	**91.0 ± 7.9**	**98.0 ± 8.5**	t-test	**<0.001**	**Significant**

TABLE 2: Comparison of Hemodynamic Parameters at Different Time Intervals

Time Point	HR (Dex) Mean ± SD	HR (Ligno) Mean ± SD	SBP (Dex) Mean ± SD	SBP (Ligno) Mean ± SD	DBP (Dex) Mean ± SD	DBP (Ligno) Mean ± SD	p-value (HR)	p-value (SBP)	p-value (DBP)
Before Nebulisation	83 ± 11	81 ± 10	126.8 ± 11.2	123.8 ± 10.3	75.6 ± 7.5	73.9 ± 8.4	0.43	0.15	0.27
After Nebulisation	83 ± 11	86 ± 11	125.7 ± 11.0	123.2 ± 12.8	72.3 ± 7.4	74.8 ± 7.7	0.13	0.27	0.08
Prior to Intubation	89 ± 12	92 ± 11	113.4 ± 10.4	117.1 ± 12.8	71.2 ± 7.3	69.8 ± 8.4	0.09	0.10	0.35
PI-0	99 ± 11	103 ± 13	130.9 ± 11.3	141.9 ± 10.5	79.2 ± 7.4	85.7 ± 8.1	0.08	<0.01*	<0.001*
PI-1	94 ± 11	100 ± 13	129.3 ± 11.4	140.5 ± 10.6	79.1 ± 7.5	85.8 ± 8.9	0.01*	<0.01*	<0.001*
PI-3	89 ± 11	97 ± 13	128.8 ± 11.2	139.4 ± 10.9	77.5 ± 8.0	84.8 ± 9.3	<0.001*	<0.01*	<0.001*
PI-5	85 ± 11	94 ± 13	126.0 ± 11.1	135.4 ± 11.0	73.4 ± 8.1	79.4 ± 9.2	<0.001*	<0.01*	<0.001*
PI-10	86 ± 11	95 ± 13	125.7 ± 10.4	133.1 ± 10.4	71.7 ± 8.3	78.2 ± 8.6	<0.001*	<0.01*	<0.001*

FIGURE 1: Heart Rate Comparison

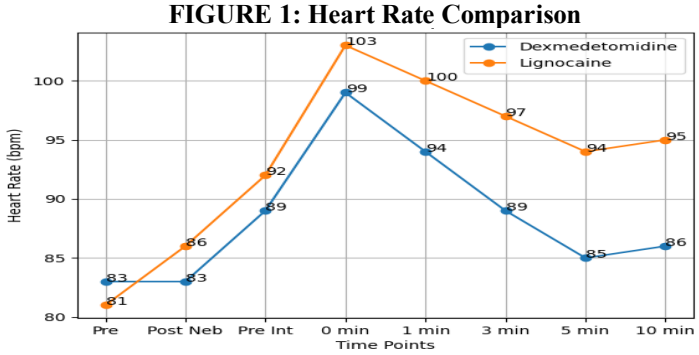
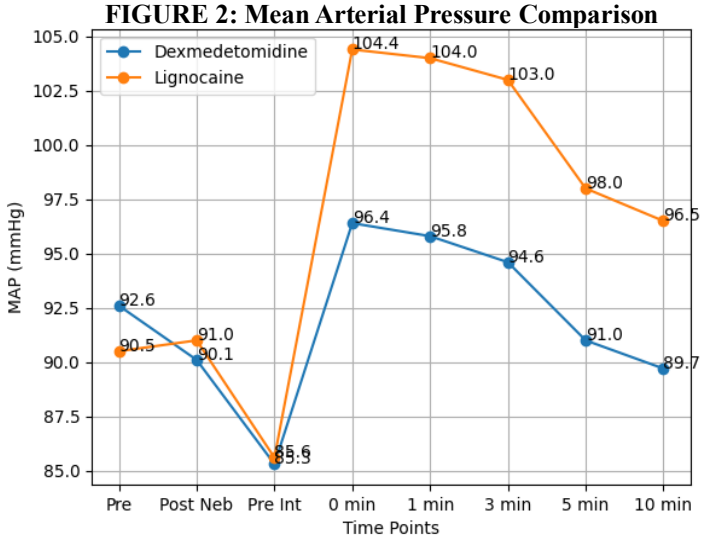


FIGURE 2: Mean Arterial Pressure Comparison



DISCUSSION

In the present study, baseline characteristics including age, gender distribution, ASA physical status, BMI, and baseline haemodynamic parameters were comparable between the dexmedetomidine and lignocaine groups, with no statistically significant difference. This indicates that the observed differences in haemodynamic responses were attributable to the study drugs rather than baseline variation. Similar baseline comparability has been reported in previous randomized studies evaluating nebulised dexmedetomidine and lignocaine. [12]

The primary outcome of this study, mean arterial pressure (MAP) at 5 minutes following intubation, was significantly lower in the dexmedetomidine group (91.0 ± 7.9 mmHg) compared to the lignocaine group (98.0 ± 8.5 mmHg), indicating superior attenuation of haemodynamic stress response. A comparable study reported significantly lower MAP values in the dexmedetomidine group at 5 minutes post-intubation, demonstrating effective suppression of the pressor response. [13] Another recent study also showed that dexmedetomidine was more effective than lignocaine in reducing MAP following intubation, supporting the findings of the present study. [14]

With respect to heart rate, this study showed no significant difference immediately after intubation; however, from 1 minute onwards, heart rate was significantly lower in the dexmedetomidine group and remained controlled up to 10 minutes. A similar trend was observed in another randomized trial, where nebulised dexmedetomidine significantly attenuated heart rate response compared to control. [12] Another comparative study demonstrated that dexmedetomidine maintained better heart rate stability than lignocaine during the post-intubation period. [14]

In terms of systolic blood pressure, the present study demonstrated a significantly lower SBP in the dexmedetomidine group at all post-intubation time points, starting from immediately after intubation. Comparable findings were reported in previous studies where dexmedetomidine significantly attenuated systolic blood pressure rise compared to lignocaine and other agents. [13] Additionally, studies comparing different routes of dexmedetomidine administration have shown effective SBP control with nebulised forms, similar to intravenous administration. [15]

Diastolic blood pressure in the present study also remained significantly lower in the dexmedetomidine group compared to the lignocaine group at all post-intubation intervals. This finding is consistent with previous studies where dexmedetomidine showed superior control over diastolic blood pressure due to its central sympatholytic action. [14] Another study reported that lignocaine, although effective in attenuating airway reflexes, provided less consistent control of DBP compared to dexmedetomidine. [16]

The overall haemodynamic profile in the present study clearly demonstrates that dexmedetomidine provided better attenuation of stress response across all parameters—heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure. Similar conclusions have been drawn in comparative studies where dexmedetomidine was found superior to lignocaine in controlling haemodynamic responses to laryngoscopy and intubation. [17] Furthermore, studies evaluating combination therapies have shown that the addition of dexmedetomidine enhances haemodynamic stability compared to lignocaine alone. [18]

Thus, the findings of this study are consistent with existing literature and reinforce that nebulised dexmedetomidine is more effective than nebulised lignocaine in attenuating haemodynamic stress response to laryngoscopy and intubation, particularly in the early and sustained post-intubation period.

CONCLUSION

The present study demonstrates that nebulised dexmedetomidine is more effective than nebulised lignocaine in attenuating the haemodynamic stress response associated with laryngoscopy and endotracheal intubation. While both agents were comparable at baseline, dexmedetomidine showed significantly better control of mean arterial pressure at 5 minutes, along with sustained reduction in heart rate, systolic blood pressure, and diastolic blood pressure at multiple post-intubation time points. The haemodynamic stability achieved with dexmedetomidine was more consistent and prolonged compared to lignocaine, likely due to its central sympatholytic action. These findings suggest that nebulised dexmedetomidine is a safe, effective, and non-invasive alternative for mitigating intubation-induced haemodynamic stress response in patients undergoing elective surgery under general anaesthesia.

LIMITATIONS

This study had certain limitations. Being a single-center study, the findings may have limited generalizability to other populations and clinical settings. The study included only ASA I and II patients, and therefore, the results cannot be extrapolated to high-risk patients with significant cardiovascular comorbidities. The sample size, although adequate for detecting primary outcome differences, may not be sufficient to evaluate rare adverse effects. Long-term haemodynamic effects and postoperative outcomes were not assessed. Additionally, plasma drug levels were not measured, which could have provided better insight into pharmacokinetic variability of nebulised drug delivery.

RECOMMENDATIONS

Based on the findings of this study, nebulised dexmedetomidine can be considered as an effective premedication for attenuating haemodynamic responses to laryngoscopy and intubation. It may be particularly useful in patients where haemodynamic stability is crucial. Further multicentric studies with larger sample sizes are recommended to validate these findings across diverse populations. Future research should include high-risk patients such as those with cardiovascular diseases to assess safety and efficacy in such groups. Comparative studies involving different doses and routes of administration of dexmedetomidine may help optimize its clinical use. Additionally, evaluation of postoperative outcomes, sedation levels, and recovery profiles would provide a more comprehensive understanding of its benefits.

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