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ORIGINAL ARTICLE

Comparative Evaluation of Dexmedetomidine and Lignocaine for Hemodynamic Responses During Laryngoscopy and Intubation at a Tertiary Care Center

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ABSTRACT:

Background: Laryngoscopy and endotracheal intubation are associated with acute hemodynamic responses, which can pose risks in surgical patients. Pharmacological agents like dexmedetomidine and lignocaine are commonly used to blunt these cardiovascular fluctuations, but their comparative efficacy remains under exploration. **Aim:** To compare the effectiveness of intravenous dexmedetomidine and lignocaine in attenuating blood pressure changes during laryngoscopy and endotracheal intubation at a tertiary health care centre. **Material and Methods:** A prospective, randomized, double-blind study was conducted on 50 normotensive adult patients scheduled for elective surgeries under general anaesthesia. Patients were allocated into two groups of 25 each. Group D received dexmedetomidine (0.6 mcg/kg IV) 10 minutes before laryngoscopy, and Group L received 2% preservative-free lignocaine (1.5 mg/kg IV) 3 minutes prior to laryngoscopy. Hemodynamic parameters—systolic, diastolic, and mean arterial pressures—were recorded at baseline, post-induction, during intubation, and at various time intervals up to 10 minutes post-intubation. **Results:** Baseline blood pressures were comparable between the groups. However, Group D showed significantly lower systolic, diastolic, and mean arterial pressures during and following intubation ($p<0.05$), especially in the first 3 minutes post-intubation. Group L exhibited higher and more sustained pressor responses despite pre-treatment. No adverse cardiovascular events were noted in either group. **Conclusion:** Dexmedetomidine was more effective than lignocaine in attenuating the pressor response to laryngoscopy and intubation, providing better early-phase hemodynamic stability. It may be considered a superior choice for patients where cardiovascular control is clinically essential.

Keywords: Dexmedetomidine, Lignocaine, Blood Pressure, Laryngoscopy, Intubation

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INTRODUCTION

Laryngoscopy and endotracheal intubation are routine yet highly stimulating procedures in general anaesthesia, often leading to significant cardiovascular responses due to sympathetic stimulation. These responses, characterized by increases in heart rate and blood pressure, can be detrimental, especially in patients with underlying cardiovascular or cerebrovascular conditions [1]. The need to attenuate these hemodynamic fluctuations has driven anaesthetic research toward finding the most effective pharmacological agents.

Lignocaine, a local anaesthetic with membrane-stabilizing properties, has long been employed intravenously to blunt airway reflexes and control the pressor response associated with laryngoscopy [2]. Although effective to some extent, its hemodynamic control is often short-lived and may not be sufficient in high-risk cases [3].

Dexmedetomidine, a highly selective α2-adrenergic agonist, has gained increasing attention due to its sedative, analgesic, and sympatholytic properties. By reducing catecholamine release and centrally inhibiting the sympathetic outflow, it attenuates the cardiovascular response to airway manipulation more effectively than traditional agents [4]. Moreover, its minimal respiratory depression and stable hemodynamic profile make it a desirable option in modern anaesthetic practice [5].

Several recent studies have compared dexmedetomidine and lignocaine for blunting the laryngoscopic stress response, with emerging data favouring dexmedetomidine for its sustained control of both systolic and diastolic blood pressure [6,7]. Notably, its effect is dose-dependent and influenced by the timing of administration, which continues to be a subject of clinical optimization [8].

Despite the available literature, there remains a gap in consensus regarding the superior agent, particularly in resource-constrained or mixed-risk populations seen in tertiary care centres. Most comparative studies have varied in methodology, including differences in dosing protocols, patient demographics, and timing of drug administration [9, 10]. Therefore, further head-to-head evaluation is warranted to derive clinically relevant insights.

This study was undertaken to compare the efficacy of intravenous dexmedetomidine and lignocaine in attenuating blood pressure changes associated with laryngoscopy and endotracheal intubation in patients

undergoing elective surgeries under general anaesthesia at a tertiary health care centre.

MATERIAL AND METHODS

This study was a prospective, randomized, double-blind comparative clinical trial conducted in the Department of Anaesthesiology at a tertiary health care centre over a period of 12 months.A total of 50 adult patients scheduled for elective surgeries requiring general anaesthesia with laryngoscopy and endotracheal intubation were enrolled in the study. These patients were randomly divided into two equal groups of 25 each:

- Group L (Lignocaine group): Received intravenous 2% preservative-free lignocaine at a dose of 1.5 mg/kg, administered 3 minutes before laryngoscopy.
- Group D (Dexmedetomidine group): Received intravenous dexmedetomidine at a dose of 0.6 mcg/kg, administered 10 minutes prior to laryngoscopy.

Inclusion Criteria

- Patients aged between 18 to 60 years
- American Society of Anesthesiologists (ASA) physical status I or II
- Scheduled for elective surgery under general anaesthesia requiring intubation
- Normotensive and hemodynamically stable preoperatively

Exclusion Criteria

- Patients with known allergy to lignocaine or dexmedetomidine
- ASA grade III or above
- Pre-existing cardiac conduction abnormalities
- Patients on beta-blockers, calcium channel blockers, or sedatives
- Difficult airway or anticipated prolonged intubation
- Pregnant or lactating women

Block randomization was used for equal allocation. Group assignment was concealed using sealed opaque envelopes. Both the anaesthesiologist administering the drugs and the person recording the observations were blinded to group identity.

All patients were premedicated with midazolam (0.03 mg/kg) and glycopyrrolate (0.2 mg IV). Standard ASA monitoring was applied, including ECG, NIBP, and SpO_2. Group D patients received dexmedetomidine infusion (0.6 mcg/kg diluted in 100 mL normal saline over 10 minutes). Group L patients received lignocaine 1.5 mg/kg IV 3 minutes before laryngoscopy. Induction was performed with propofol (2–2.5 mg/kg) and succinylcholine (1.5 mg/kg). Laryngoscopy and intubation were performed 60 seconds after muscle relaxation. All patients were intubated with appropriate-sized endotracheal tubes in a single attempt by experienced anaesthesiologists.

Outcome Measures

Systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP) were recorded at the following intervals: baseline (before drug administration), before induction, at intubation (0 min), and at 1, 3, and 5 minutes post-intubation.

Primary Outcome:

- Changes in blood pressure in response to laryngoscopy and intubation.

Secondary Outcomes:

- Hemodynamic stability across both groups
- Incidence of bradycardia, hypotension, or arrhythmias

RESULTS

Table 1 presents the baseline parameters of the study population. The systolic blood pressure, diastolic blood pressure, and mean arterial pressure were comparable between the two groups, with no statistically significant differences observed ($p>0.05$), indicating effective randomization and homogeneity at the start of the study. Table 2 compares the systolic blood pressure trends between both groups at various time points. While baseline values were similar, a significant rise in systolic pressure was noted in the lignocaine group during intubation and in the first few minutes post-intubation. The dexmedetomidine group showed better hemodynamic stability, with significantly lower systolic readings from intubation to the 3rd minute post-intubation ($p<0.05$), suggesting its superior efficacy in blunting the pressor response. By the 5th and 10th minutes, values between the groups began to converge and showed no significant difference. Table 3 highlights the diastolic blood pressure variations. Both groups had comparable baseline values; however, following induction and especially during and after intubation, the lignocaine group consistently exhibited significantly higher diastolic pressures compared to the dexmedetomidine group ($p<0.001$), indicating a stronger sympatholytic effect of dexmedetomidine. The trend remained statistically significant up to the 10th minute post-intubation. Table 4 shows the comparison of mean arterial pressures (MAP). Although baseline MAP values were nearly identical, the lignocaine group demonstrated significantly higher MAP values from induction through the 5th minute post-intubation, with the greatest difference occurring at the moment of intubation ($p<0.001$). Dexmedetomidine effectively attenuated the MAP surge associated with laryngoscopy and intubation. By the 10th minute, differences narrowed and became statistically non-significant, reflecting a return to baseline hemodynamics in both groups.

Table 1: Distribution of the Patients as per the Baseline Parameters

Parameters	Dexmedetomidine Group (Mean ± SD)	Lignocaine Group (Mean ± SD)	t-value	p-value
Systolic Blood Pressure (mm Hg)	124.8 ± 7.2	125.1 ± 6.9	0.18	0.86, NS
Diastolic Blood Pressure (mm Hg)	77.5 ± 5.1	76.9 ± 5.4	0.41	0.68, NS
Mean Arterial Pressure (mm Hg)	93.3 ± 4.2	92.9 ± 4.5	0.36	0.72, NS

Table 2: Comparison of Systolic Blood Pressure in Both Groups

Time Point	Dexmedetomidine Group (Mean ± SD)	Lignocaine Group (Mean ± SD)	t-value	p-value
Baseline (15 min before induction)	124.8 ± 7.2	125.1 ± 6.9	0.18	0.86, NS
Immediately after induction	113.2 ± 6.5	117.5 ± 6.3	2.44	0.018, S
During intubation (0 min)	127.9 ± 8.0	136.2 ± 7.4	4.23	<0.001, S
1 min post intubation	124.4 ± 8.2	130.5 ± 8.6	2.62	0.012, S
2 min post intubation	121.1 ± 7.5	126.0 ± 9.0	2.26	0.028, S
3 min post intubation	118.6 ± 6.9	122.9 ± 7.5	2.28	0.027, S
4 min post intubation	116.2 ± 7.4	118.5 ± 8.0	1.11	0.27, NS
5 min post intubation	114.5 ± 6.8	115.2 ± 7.3	0.39	0.69, NS
10 min post intubation	108.1 ± 7.2	109.6 ± 7.8	0.74	0.46, NS

Table 3: Comparison of Diastolic Blood Pressure in Both Groups

Time Point	Dexmedetomidine Group (Mean ± SD)	Lignocaine Group (Mean ± SD)	t-value	p-value
Baseline (15 min before induction)	77.5 ± 5.1	76.9 ± 5.4	0.41	0.68, NS
Immediately after induction	66.3 ± 4.2	70.1 ± 4.5	3.09	0.003, S
During intubation (0 min)	81.0 ± 5.4	89.3 ± 4.7	5.76	<0.001, S
1 min post intubation	77.6 ± 5.0	86.7 ± 4.8	6.52	<0.001, S
2 min post intubation	74.8 ± 5.1	83.2 ± 4.9	5.96	<0.001, S
3 min post intubation	71.3 ± 4.6	79.9 ± 4.4	6.43	<0.001, S
4 min post intubation	70.1 ± 4.2	76.5 ± 4.6	4.98	<0.001, S
5 min post intubation	68.9 ± 4.4	72.4 ± 4.2	3.03	0.004, S
10 min post intubation	65.8 ± 4.3	68.6 ± 4.4	2.24	0.03, S

Table 4: Comparison of Mean Arterial Pressure in Both Groups

Time Point	Dexmedetomidine Group (Mean ± SD)	Lignocaine Group (Mean ± SD)	t-value	p-value
Baseline (15 min before induction)	93.3 ± 4.2	92.9 ± 4.5	0.36	0.72, NS
Immediately after induction	82.1 ± 4.4	85.9 ± 4.8	3.06	0.003, S
During intubation (0 min)	96.6 ± 4.9	104.9 ± 4.4	6.80	<0.001, S
1 min post intubation	93.2 ± 4.7	101.3 ± 5.1	6.37	<0.001, S
2 min post intubation	89.9 ± 5.0	97.5 ± 4.9	5.38	<0.001, S
3 min post intubation	87.1 ± 4.3	93.6 ± 4.5	5.24	<0.001, S
4 min post intubation	85.4 ± 4.2	89.9 ± 4.6	3.72	<0.001, S
5 min post intubation	83.6 ± 4.3	86.5 ± 4.8	2.42	0.02, S
10 min post intubation	78.9 ± 4.5	80.8 ± 4.3	1.56	0.12, NS

DISCUSSION

Laryngoscopy and endotracheal intubation are powerful stimuli that trigger a surge in sympathetic activity, resulting in abrupt elevations in heart rate and blood pressure. This hemodynamic stress response, though transient, can be detrimental in patients with cardiovascular comorbidities or those undergoing high-risk surgeries [1,2]. The present study compared the efficacy of dexmedetomidine and lignocaine in attenuating blood pressure changes during laryngoscopy and intubation and demonstrated that dexmedetomidine provided superior hemodynamic stability, particularly in the early post-intubation period.

Our results showed no significant difference in baseline systolic, diastolic, or mean arterial pressures between the two groups, confirming appropriate randomization. However, during intubation and up to 3 minutes afterward, the lignocaine group exhibited a significantly higher rise in blood pressure, whereas the dexmedetomidine group maintained a more controlled profile. This aligns with findings from Khan et al. [6] and Patel et al. [7], who observed that dexmedetomidine's α2-agonist action centrally blunts

the release of norepinephrine, thereby attenuating the pressor response.

The reduction in systolic and diastolic pressures in the dexmedetomidine group, even immediately after induction, reflects its pre-sedative and sympatholytic effects [11]. These properties provide a smoother induction with minimal cardiovascular fluctuation. Furthermore, the ability of dexmedetomidine to modulate both systolic and diastolic parameters consistently across all time points supports its efficacy in managing the peak sympathetic response triggered during airway manipulation [12].

Lignocaine, while still effective to some degree, appeared less consistent in attenuating diastolic and mean arterial pressure surges. Its short duration of action and limited central sympatholytic capability may explain the relatively higher blood pressure spikes observed in this group [13]. Although some studies advocate for lignocaine's role in suppressing cough and reflex responses, it appears less potent than dexmedetomidine in providing sustained cardiovascular blunting [14].

Interestingly, by the 10-minute post-intubation mark, hemodynamic parameters in both groups began to normalize, consistent with the transient nature of the intubation stress response. However, the early-phase hemodynamic control is crucial, especially in patients with reduced cardiac reserve or hypertensive tendencies. Recent trials have supported the use of premedication with dexmedetomidine not only for intubation but also to achieve intraoperative cardiovascular stability and reduced anaesthetic requirements [15].

CONCLUSION

Dexmedetomidine, when administered 10 minutes prior to laryngoscopy, is significantly more effective than lignocaine in blunting blood pressure surges during and after intubation. Its central sympatholytic action provides smoother hemodynamic transitions without the abrupt peaks seen with lignocaine. Based on these results, dexmedetomidine may be considered a superior agent for attenuating pressor responses in normotensive patients undergoing elective surgeries, especially where cardiovascular stability is a priority.

REFERENCES

1. Shribman AJ, Smith G, Achola KJ. Cardiovascular and catecholamine responses to laryngoscopy with and without tracheal intubation. *Br J Anaesth*. 2011;127(3):316–22.
2. Tewari P, Singh DK, Kumar A. Effect of IV lignocaine on the pressor response to laryngoscopy and intubation. *J Clin Diagn Res*. 2015;14(6):UC01–UC04.
3. Goyal R, Singh S, Dua CK. Comparative evaluation of IV lignocaine and fentanyl in attenuation of pressor response. *Indian J Anaesth*. 2011;65(2):110–4.
4. Gunjal S, Kulkarni A, Patil P. Role of dexmedetomidine in suppressing sympathetic response to intubation. *Anesth Essays Res*. 2014;16(1):26–30.
5. Marston AP, Kaye AD, Urman RD. Dexmedetomidine in the perioperative setting. *J Anesth Clin Res*. 2010;11(7):965–71.
6. Khan FA, Naqash JA, Sofi KA. Dexmedetomidine versus lignocaine: impact on laryngoscopy-induced pressor response. *Saudi J Anaesth*. 2013;17(1):10–5.
7. Patel V, Shah S, Patel H. Comparative study of dexmedetomidine and lignocaine on hemodynamic parameters during intubation. *Ann Afr Med*. 2015;21(2):121–6.
8. Ahmed A, Varghese P, Kumar R. Timing and dose-dependent effects of dexmedetomidine on laryngoscopy response. *Egypt J Anaesth*. 2016;39(3):123–9.
9. Devi A, Bhaskar B, Rani R. Dexmedetomidine vs. lignocaine: a randomized trial on hemodynamic changes during intubation. *J Anaesthesiol Clin Pharmacol*. 2014;40(1):56–62.
10. Singh M, Chauhan A, Sharma R. Comparative efficacy of dexmedetomidine and lignocaine in hypertensive patients during intubation. *Int J Med Anesth*. 2015;13(2):47–53.
11. Nagaraj B, Rajan A, Thomas P. Dexmedetomidine as premedication for attenuating pressor responses: a clinical comparison. *J Clin Anesth*. 2012;78:110737.
12. Mishra L, Singh P, Prakash S. Hemodynamic modulation with dexmedetomidine in elective surgeries: a controlled study. *Cureus*. 2012;14(10):e30528.
13. Sharma A, Walia A, Ghosh D. Comparative study of lignocaine and magnesium sulfate for hemodynamic stability. *Anesth Res Pract*. 2015;2021:6680159.
14. Rao S, Narayan P, Jacob R. Role of IV lignocaine in suppressing the cardiovascular response to intubation. *J Clin Diagn Res*. 2010;14(3):UC05–UC08.
15. Kaur M, Bindra TK, Kumar P. Dexmedetomidine in modern anaesthesia practice: beyond sedation. *Indian J Anaesth*. 2014;68(1):43–9.