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# COMPARISON OF INTRAVENOUS LIGNOCAINE VERSUS INTRAVENOUS DEXMEDETOMIDINE FOR ATTENUATION OF HEMODYNAMIC RESPONSE DURING GENERAL ANESTHESIA

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## Abstract

***BACKGROUND***

*The hemodynamic responses of tachycardia and hypertension, which are associated with Laryngoscopy and endotracheal intubation, are potentially detrimental, especially in high-risk patients. This response has been suppressed with a number of pharmacological agents. The study aimed to compare the effectiveness of intravenous lignocaine and intravenous dexmedetomidine in the attenuation of the hemodynamic response to general anesthesia.*

***METHODOLOGY***

*It is a randomized controlled trial, which was carried out in the Department of Anesthesia, LMDC/Ghurki Trust Hospital, Lahore, from February 2025 to May 2025. Seventy-six patients within the age range of 18-60 years, ASA I-II, undergoing elective laparoscopic and gynecological surgery were selected and randomly split into 2 groups (n= 38 patients assigned to each group). Group A was given intravenous lignocaine (1.5mg/kg), and Group B was given intravenous dexmedetomidine (1 ug/kg). All patients were given general anesthesia. Observance of hemodynamic parameters, such as heart rate and mean arterial pressure, was done at the baseline and 5 minutes following intubation.*

***RESULTS***

*There was equal baseline characteristics in the two groups. At 5 minutes of the intubation, the dexmedetomidine group showed better attenuated hemodynamic response, most patients were in the 60-80 bpm range (65.8%), the others in the lignocaine group were mostly in the 100-120 (52.6%), and 120 or higher range. On the same note, mean arterial pressure was tighter in the dexmedetomidine group, with the majority of the patients being within the range of 70-110 mmHg, whereas in the lignocaine group, 44.7% were within the range of 110-130 mmHg, and the rest 42.1% were above 130 mmHg.*

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***CONCLUSION***

*The intravenous dexmedetomidine is more effective in the attenuation of hemodynamic response to laryngoscopy and endotracheal intubation. It offers better regulation of heart rate and mean arterial pressure and is a more consistent method of maintaining hemodynamic stability in the process of induction of general anesthesia.*

## INTRODUCTION

Laryngeal manipulation is commonly performed in anesthesia [1]. This technique includes stimulation of pharyngeal and laryngeal structures, which are expansively surrounded by parasympathetic and sympathetic-nerves. The discharge of vasoactive mediators practically accompanies direct manipulation of these structures after initiation of anesthesia [2]. Cardiovascular reaction to laryngeal manipulation causes a rise in pulse rate, blood pressure, central nervous system pressures, intraocular tension, and cardiac dysrhythmias. Moreover, it can also lead to cardiac arrest [3].

These clinical findings are observed as a physiological response of laryngeal manipulation, usually enhanced by Light Anesthesia, decreased ventilation, Anxiety and Reflex baroreceptor effect following induction agents like Propofol. Patients with high blood pressure are predisposed to hypertension [4]. Hemodynamic findings are not severe in normal patients but might be dangerous in patients with cardiovascular and central nervous system disorders [5,6].

Dexmedetomidine is a different compound with multiple times more similar receptor affinity than clonidine. Treatment with this drug before anesthesia reduces the cardiovascular reaction to the stress of intubation [7].

In a study done by Prasad et.al. the mean heart rate was 84.76±11.34bpm in dexmedetomidine versus 83.80±10.62bpm in lignocaine group at baseline and at 5 minutes to intubation 84.60±9.60bpm versus 96.50±9.80bpm. Mean blood pressure at baseline was 92.80+10.36mmHg in dexmedetomidine versus 92.60±10.40mmHg in lignocaine group, while at 5 minutes, mean blood pressure was 88.12±9.90mmHg in dexmedetomidine versus 94.60+10.10mmHg in lignocaine group [8].

This study aims to compare the hemodynamic changes of intravenous lignocaine versus intravenous dexmedetomidine on attenuation of hemodynamic response during general anesthesia. No such study has been done earlier on this subject in our local population. Dexmedetomidine is a new drug, so its efficacy must be evaluated in our general population. Therefore, we have planned to associate the effectiveness of Lignocaine and Dexmedetomidine in reduction of cardiovascular reaction to tracheal manipulation during intubation. If results favor Dexmedetomidine, it can be used routinely in our general population.

## METHODOLOGY

This study aim was to compare the hemodynamic responses in intravenous injection of lignocaine and intravenous injection of dexmedetomidine during general anesthesia. Hemodynamic reaction was measured in average heart rate and average arterial pressure. There was a measurement of the mean heart rate in beats per minute, 5 minutes into intubation. Mean arterial pressure was also measured after 5 minutes of intubation, and was determined by the formula MAP = [(2xDP) +sp/3]. The difference between systolic blood pressure (SBP) and diastolic blood pressure (DBP) was considered pulse pressure. The study hypothesis was that the average hemodynamic parameters of intravenous lignocaine and intravenous dexmedetomidine in the attenuation of the hemodynamic response during general anesthesia differ.

The study was a randomized controlled trial and was carried out in the Department of Anesthesia, LMDC/Ghurki Trust Hospital, Lahore, following approval of the synopsis from Feb 2025 to May 2025. The total sample size of 76 patients, with 38 patients in each group, was determined by using

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the 95% confidence level and 80% power of the test. This was calculated using the anticipated mean blood pressure of 5 minutes in the dexmedetomidine group of 88.12 + 9.90 mmHg and in the lignocaine group of 94.60 + 10.10 mmHg [8].

The study included patients aged 18 to 60 years of either sex, with general elective laparoscopic and gynecological operations, and with ASA I-II. Patients who are known to be allergic to dexmedetomidine or lignocaine, are known to be hypertensive, have ischemic heart disease or arrhythmias, pregnant, undergo oropharyngeal surgeries, or have suspected airway abnormalities or expected difficult airway were excluded.

Patients meeting the inclusion and exclusion criteria were recruited in the study after obtaining the consent of the hospital ethical committee (76 patients in total). The lottery method was employed to assign all the patients to two groups: intravenous lignocaine Group A and intravenous dexmedetomidine Group B. On reaching the operating theatre, initial vital parameters such as pulse rate, blood pressure, respiratory rate as well as oxygen saturation were obtained. Every patient was preloaded with 500 mL of the lactated Ringer solution. Before the procedure, Group A was given 100 mL of 0.9% saline, and intravenous lignocaine at 1.5mg/kg was used three minutes before laryngoscopy, and after ten minutes. Group 2 was injected with dexmedetomidine at a concentration of 1 ug/kg in 100 ml of 0.9% saline intravenously over 10 minutes, followed until all was used, 10 minutes before induction.

Premedication was performed on all patients as follows: intravenous dexamethasone 0.08 mg/kg, nalbuphine 0.1 mg/kg, and midazolam 0.02mg/kg, followed by preoxygenation. Propofol was used to induce. Atracurium 0.5 mg/kg intravenously was used to facilitate mechanical ventilation, three minutes before tracheal manipulation. A Macintosh laryngoscope was used to perform laryngoscopy and endotracheal intubation. Once the tube was properly in place, it was fastened. The first 10 minutes of the procedure did not involve surgical stimulation. An inhalational agent and atracurium were used to maintain anesthesia. All patients were injected with neopyrolate 0.05mg/kg intravenously at the start of the operation. The extubation occurred when the pharyngeal reflexes were recovered. The operational definition recorded the mean arterial pressure and heart rate at 5 minutes. All were recorded in a prewritten proforma.

The entries and analysis were done with SPSS 26.0. Mean age + standard deviation, BMI, blood pressure, and heart rate were quantitative variables. The independent sample t-test helped in comparing the two groups in terms of blood pressure and heart rate. The qualitative variables (gender and ASA status) were in the form of frequencies and percentages. The effect modifiers were determined by stratifying data according to age, gender, ASA status, and BMI. The Student was stratified and T test was applied on the stratified Student and t-values below 0.05 were considered significant values.

## RESULTS

This randomized controlled trial involved 76 patients who were randomized (Group A, intravenous lignocaine, n = 38, and Group B, intravenous dexmedetomidine, n = 38). Every patient was able to complete the research and take part in the analysis. The two groups matched in terms of the demographic or clinical features. Group A showed 65.8 and 34.2 percent males and females respectively, with Group B being 63.2 and 36.8 percent males and females respectively. Concerning ASA status, 60.5% of patients in Group A and 71.1% in Group B were ASA II, with the rest of the patients in the group being ASA I, which means that there is a similarity in the baseline features between the two groups.

The baseline showed similarity in the distributions of heart rate in both groups. The highest percentage of patients with a heart rate of 100-120 bpm was found in Group A (47.4%), and with the range of 80-100 bpm in Group B (50.0%). This was, however, seen at 5 minutes after intubation between the two groups. Group A showed that most of the patients (52.66) were in the range of 100-120 bpm, and a minority proportion (21.1) of the patients had their hearts above 120 bpm, which indicated that the tachycardia had not stopped. Conversely, in Group B, the majority of

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patients (65.8) fell within the 60-80 bpm range of heart rates, with only a smaller percentage in higher heart rate ranges, which is a suggestion of better heart rate response attenuation with dexmedetomidine.

In the same vein, the baseline mean arterial pressure between the two groups was similar. At the 5-minute post-intubation, Group A exhibited an inclination to have increased mean arterial pressure values, and 44.7 percent of patients had higher mean arterial pressure values in the range of 110-130 mmHg; 42.1 percent had higher mean arterial pressure values above the range of 130 mmHg, a sign of increased blood pressure. Conversely, Group B showed improved management of mean arterial pressure, whereby most patients were within the 70-110 mmHg range, and only a few patients had high values.

Comprehensively, patients treated with intravenous dexmedetomidine had reduced heart rates and improved mean arterial pressure control 5 minutes following intubation, which showed reduced hemodynamic response to laryngoscopy and intubation. Comparatively, the patients in the lignocaine group also experienced a relative rise in heart rate and mean arterial pressure, indicating ineffective attenuation of the hemodynamic response.

## DISCUSSION

The current randomized controlled trial compared the effect of intravenous lignocaine and dexmedetomidine in reducing the hemodynamic response to laryngoscopy and endotracheal intubation. This study has shown that dexmedetomidine is superior to lignocaine in regulating the heart rate and mean arterial pressure, thus offering better damping of stress response.

Patients receiving dexmedetomidine in this study showed a significant decrease in heart rate at 5 minutes, with most of the patients showing a range of values of 60-80 bpm, unlike the patients in the lignocaine group, who still reported higher values. Equally, mean arterial pressure was more controlled in the dexmedetomidine group, and a high percentage of patients in the lignocaine group recorded high MAP values (>130 mmHg). These results indicate that dexmedetomidine has a superior sympatholytic control, which results in a reduction of tachycardia and hypertension that occurs with laryngoscopy and intubation.

The findings of the current study are in agreement with several trials that have been published before. Dexmedetomidine (1 ug/kg) was found to have better attenuation of the pressor response, in comparison with lignocaine (1.5 mg/kg), in a randomized study by Gulabani et al.. It resulted in significant heart rate and blood pressure control after intubation [9]. Equally, a recent clinical study performed in Lahore revealed that patients undergoing dexmedetomidine were found to have lower heart rate and blood pressure than patients undergoing lignocaine after 5 minutes ( $p = 0.001$), further supporting that dexmedetomidine is better at maintaining hemodynamic stability [10]. A second comparison of several agents has shown that dexmedetomidine had superior control of the heart rate and blood pressure than lignocaine, and more stable hemodynamics during laparoscopy [11].

Moreover, controlled trial evidence shows that despite the possible attenuating effect of lignocaine on the hemodynamic response, the response is rarely completely suppressed and increases in blood pressure and heart rate may persist following intubation [12]. These findings collectively support the results of the present study, confirming that dexmedetomidine offers superior attenuation compared to lignocaine.

The excellence of dexmedetomidine could be attributed to its pharmacological characteristics. Dexmedetomidine is a selective A2-adrenergic agonist and a sympathetic outflow reducer, catecholamine release decreaser, sedative, and analgesic, but without respiratory depression [13]. Lignocaine, by contrast, is more of a sodium channel blocker and offers airway reflex blunting but less sympatholytic activity, perhaps explaining its comparatively less robust control of hemodynamic responses.

The clinical implications of the findings of this study are significant. Dexmedetomidine may be viewed as an agent of choice in hemodynamic response attenuation during intubation and is especially beneficial in patients where tachycardia

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and hypertension are potentially detrimental, such as cardiac or neurosurgical patients. Lignocaine is a useful but not adequate agent in high-risk situations.

There are a number of limitations to this study. The hemodynamic data were documented in categorical values, and not in mean +- SD values which restricted a definite statistical comparison. Statistical significance could not be confirmed as P-values were not possible to compute. It was a unicenter study with a relatively small sample size (n= 76), and only the short-term 5-minute hemodynamic response was evaluated.

Continuous variables (mean + SD) should be added to future studies in order to provide stronger statistical analysis. Smaller randomized trials should be abandoned in favor of larger ones that are multicentric. Adverse effects like bradycardia and hypotension should also be evaluated and further follow-up is also required to check sustained hemodynamic stability.

According to the results of this trial, intravenous dexmedetomidine demonstrates a superior ability to reduce the hemodynamic response to laryngoscopy and endotracheal intubation compared to intravenous lignocaine based on the control of heart rate and mean arterial pressure.

## CONCLUSION

I.V. dexmedetomidine was considered superior to I.V. lignocaine in the prevention of hemodynamic response to laryngoscopy and endotracheal intubation. Dexmedetomidine patients had superior control of heart rate as well as mean arterial pressure at 5 minutes after intubation, signifying their better suppression of the stress response. Lignocaine, in comparison, demonstrated relatively poorer attenuation, and there was a continued high heart rate and blood pressure.

Thus, dexmedetomidine can be viewed as a more consistent method of ensuring hemodynamic stability in the process of induction of general anesthesia, especially in cases when over-reactive cardiovascular reactions are unwanted.

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