Type of Article: Original Article Research

INTRAVENOUS VERSUS NEBULIZED LIGNOCAINE ON ATTENUATION OF HEMODYNAMIC RESPONSE DURING LARYNGOSCOPY AND INTUBATION UNDER GENERAL ANAESTHESIA - A RANDOMIZED CONTROL TRIAL

Rahul Raj Singh[1], Akash Gupta[2], Tulika Mittal[3], Divya Vijay[4]*

[1]Junior Resident, Department of Anaesthesiology, Rohilkhand Medical College and Hospital, Bareilly, India.

[2]Associate Professor, [3]Assistant Professor, Department of Anaesthesiology, Rohilkhand Medical College and Hospital, Bareilly, India.

[4]Assistant Professor, Shri Ram Murti Smarak Medical College and Hospital, Bareilly

***Corresponding Author: Dr. Divya Vijay**

Assistant Professor, Shri Ram Murti Smarak Medical College and Hospital, Bareilly India.

Abstract

Background: The introduction of deep sedation & tracheal intubation is demanding and causes severe hemodynamic alterations. Laryngoscopy & tracheal manoeuvres were unpleasant stimuli that cause a short-term rise in autonomic response, resulting in dysrhythmias, elevated arterial pressure, and pulse rate.

Objective: To compare the difference in HR, SBP, DBP, and MAP at different intervals (before induction and after induction) in two groups during laryngoscopy and intubation under general anaesthesia.

Methods: A randomized control trial was conducted and a total of 98 patients undergoing elective surgeries under General Anaesthesia were taken and were randomly divided in 2 groups: Group A (I.V Lignocaine) and Group B (Nebulized lignocaine) to 49 patients each. GROUP A: 49 patients were given IV Lignocaine (2%) 1.5mg/kg 3 minutes prior to laryngoscopy. GROUP B: 49 patients were given Nebulized Lignocaine (2%) 5ml 5 minutes prior to laryngoscopy.

Results: Comparing the nebulized lignocaine group to the IV lignocaine group, we observed a statistically significant decrease of hemodynamic responses after intubation in our research. In our study statistically significant results were seen with heart rate, systolic blood pressure and mean arterial pressure with p value less than 0.05 and insignificant results with diastolic blood pressure and all other demographic variables.

Conclusion: Hemodynamic variables were more stable in nebulized lignocaine group and it was seen that nebulized lignocaine attenuates the pressor responses much better than intravenous lignocaine with less hemodynamic instability.

Keywords: Intravenous Lignocaine, Nebulized Lignocaine, Hemodynamic response, General Anaesthesia

INTRODUCTION

Laryngoscopy and tracheal intubation are strong stimuli that raise BP and HR, especially if laryngoscopy lasts up to 45 seconds. The pressor and sympathoadrenal responses produced by laryngoscopy alone and laryngoscopy followed by intubation are virtually same.[1]

Patients with cardiac disorders may experience injury from this sudden, erratic, and transitory rise of HR and BP. According to reports, the average increase in heart rate (HR) is 23 beats higher than baseline, while systolic blood pressure (SBP) rises by 53 to 54 mmHg and the left ventricular ejection fraction falls by about 20%.[7]The incidence of morbidity and mortality among patients with cardiovascular and cerebrovascular illnesses may rise as a result of this set of complications.[2]

Airway manipulation may be associated with an increased risk of airway management difficulties and critical events such as multiple attempts at laryngoscopy, oesophageal intubation, and aspiration. Each can lead to severe hypoxemia, culminating in some with spinal cord-induced bradycardia with subsequent hypotension, decreased cardiac output, and, if uncorrected, cardiovascular collapse. Extreme fatigue, hypoxia, hypercapnia, acidosis, and other stresses can push the patient's physiological system to near maximum sympathetic outflow.[3]

Because of these adverse and detrimental effects of laryngoscopy and intubation, a wide variety of pharmacological agents have been used which includes fentanyl (a synthetic opioid receptor agonist), nalbuphine (opioid agonist-antagonist), in which fentanyl is the best and the potent one. Alpha 2 adrenoceptor agonists like clonidine and dexmedetomidine are used as an adjunctive anaesthetic agent because of their hemodynamic stabilising and anaesthetic sparing effects. Cardio selective beta-adrenergic blocker like esmolol used for attenuating the hemodynamic response and beside this ability it also decreases the dose of anaesthetic agent for maintaining the required depth of anaesthesia. Other agents like nitro-glycerine and magnesium sulphate have also shown the promising effects in attenuating the hemodynamic response. [4,5]

The advantages of lignocaine are that – it decreases the cardiovascular effects to laryngeal procedures, suppresses of cough reflex, its antidysrhythmic action, reduction in intracranial pressure, and ability to reduce minimum alveolar concentrations of inhalational anaesthetics.[6]

This study was conducted to see how lignocaine affects patient’s hemodynamic response during intubation. As far as we are aware, there are few studies comparing intravenous lignocaine and nebulized lignocaine. To bridge the paucity of data in Indian scenario we are comparing the effects of intravenous lignocaine (2%) versus Nebulized lignocaine (2%) on attenuation of hemodynamic response during laryngoscopy and intubation under General anaesthesia.[7]

MATERIALS AND METHODS

This randomized control trial was conducted in Department of Anaesthesiology, Rohilkhand Medical College and Hospital, Bareilly after obtaining the approval from board of Institutional Ethics Committee and registration of this study with clinical trial registry of India (CTRI/2023/07/054687); dated 03/07/2023. Duration of study was one Year (1stNovember 2022 – 31stOctober2023)

Study Duration:Inclusion Criteria

- ASA Grade 1 and 2
- Age 18 to 65 years.
- Patients with Mallampati grading 1 and 2

Exclusion Criteria

- Known allergy to the trial drugs
- Emergency surgeries or patients considered as full stomach.
- Predicted difficult airway
- Patients with bronchospastic disease and known hypertensives.

Sample Size: In our study a total of 98 patients were taken, which was statistically calculated by using the software Power and Sample Size Program [8]

GROUP A: 49 patients were given IV Lignocaine (2%) 1.5mg/kg 3 minutes prior to laryngoscopy.

GROUP B: 49 patients were given Nebulized Lignocaine (2%) 5ml 5 minutes prior to laryngoscopy.

Methodology:

Institutional Ethics Committee approval was taken prior to conducting this study.

Patient's consent was taken prior to procedure.

After taking approval from institutional ethics committee a randomized control study was carried out in Rohilkhand Medical College and Hospital Bareilly on total of 98 patients of ASA physical

status class 1 and 2 of either sex, in age group of 18 to 65 years undergoing elective surgeries under General Anaesthesia requiring Endotracheal Intubation with Macintosh laryngoscope. Patients were assigned randomly in 2 groups: Group A (I.V Lignocaine-preservative free) and Group B (Nebulized lignocaine) to 49 patients each.

After taking informed written consent a thorough pre-anaesthetic checkup was done night before and Tablet Alprazolam 0.25mg and Tablet Ranitidine 150mg were given as premedication on night prior and morning of surgery. Patients was shifted to operating theatre and wide calibre (18G) cannula was secured and baseline vitals such as electrocardiogram, non-invasive blood pressure, pulse rate and SpO2 were checked. Group A patients were given IV Lignocaine (2%) 1.5mg/kg 3 minutes prior to laryngoscopy & Group B patients were given Nebulized Lignocaine (2%) 5ml 5 minutes prior to laryngoscopy. Study drug preparation was done by anaesthetist who was not aware of study protocol.

Patients were premedicated with intravenous Butorphanol 0.02 mg/kg and Midazolam 0.02 mg/kg, subsequently preoxygenation done with 100% FiO_2 for 3 minutes. Anaesthesia was introduced with IV Propofol 2 mg/kg, and muscle relaxant IV Vecuronium bromide 0.1mg/kg was given for smooth direct laryngoscopy & endotracheal intubation. Intubation were accomplished by Macintosh curved blade laryngoscope and a proper sized cuffed polyvinylchloride (PVC) Endotracheal tube (7.0 mm for females and 8.0 mm for males). Laryngoscopy and intubation were performed 3 minutes after administration of vecuronium. Anaesthesia was maintained with oxygen 40% plus nitrous oxide 60%, isoflurane 1% and vecuronium 0.02 mg/kg as top up.

Heart rate, Systolic blood pressure,Meanarterial pressure, Diastolic blood pressure were measured (baseline) before induction, after induction and laryngoscopy and 1,3,5 and 10 minutes after endotracheal intubation by an anaesthetist who was blinded for study. Bolus dose of vecuronium 0.02mg/kg was used to maintain neuromuscular blockade (NMB). All patients received injection paracetamol 1gm I.V to provide intraoperative analgesia. I.V Injection Ondansetron 0.1 mg/kg was given 30 minutes prior of completion of surgery to avoid post-operative nausea and vomiting (PONV). When surgery was about to finish, inhalational agents (isoflurane) was stopped, and neuromuscular blockade was overturned by Injection Neostigmine 0.05mg/kg and Injection Glycopyrolate 0.01mg/kg IV. Patients was then extrubated and was shifted to post anaesthesia care unit.

Statistical Analysis:

The data from the present analysis was systematically collected, compiled, and statistically analysed. Descriptive & inferential statistical analysis were derived from results on continuous measurements, conferred as mean ± SD while results on categorical measurements were presented in numbers (%age). The data were entered on a Microsoft Excel spreadsheet and imported into Statistical Package for Social Sciences (SPSS) version 23 for statistical analysis.

Qualitative data was present in frequency and percentage and quantitative data was presented in mean and standard deviation. A chi-square test was performed to find associations in different variables between the 2 groups, and student independent t-test was performed to find significant differences in mean in different variables among the two groups. The **p-value** was taken significant when less than 0.05 **($p<0.05$)** andConfidence interval of 95% was taken.

RESULTS

The randomized control trial was executed in patients posted for elective surgeries undergoing general anaesthesia to compare and evaluate efficacy of I.V Lignocaine versus Nebulised Lignocaine in attrition of circulatory response to endotracheal instrumentation in patients of ASA 1 and ASA 2 grade. A total of 98 patients were randomly cleaved in two groups in 1:1 allocation ratio and each group comprising of 49 patients.

The two groups were comparable regarding Age, Gender, ASA Grade, MPG Grade and hemodynamic variables (HR, SBP, DBP, MAP). All demographic variables were insignificant between both groups.

TABLE-1 Comparison of mean Heart Rate (bpm) at different time interval in Group A & Group B.

	Group A (IV)	**Group B (NEBS)**	
Heart Rate(bpm)	**Mean ± SD**	**Mean ± SD**	**P-Value**
Baseline	85.27 ± 10.0	86.0 ± 8.43	0.695#
After Induction	80.65 ± 8.55	80.24 ± 7.37	0.801#

After Laryngoscopy & intubation	84.59 ± 9.55	81.33 ± 5.84	0.044*
1 min after	76.0 ± 9.84	72.23 ± 6.91	0.030*
3 min after	74.9 ± 9.5	71.1 ± 8.22	0.036*
5 Min after	73.02 ± 10.25	71.55 ± 8.73	0.447#
10 min after	69.67 ± 7.89	71.02 ± 7.96	0.402#

#statistically insignificant; * statistically significant

The mean Heart rate of individuals at baseline in our study in Group A (IV) was85.27 ± 10.0 bpm and in Group B (NEBS) was 86.0 ± 8.43 bpm, Mean Heart rate of patients After Induction in our study in Group A(IV) was80.65 ± 8.55 bpm and in Group B (NEBS) was 80.24 ± 7.37 bpm, Mean Heart rate of subjects After Laryngoscopy & Intubation in our study in Group A(IV) was84.59 ± 9.55bpm and in Group B (NEBS) was 81.33 ± 5.84 bpm, Mean Heart rate of patients 1 min after Laryngoscopy and Induction in our study in Group A (IV) was76.0 ± 9.84 bpm and in Group B (NEBS) was 72.23 ± 6.91 bpm ,Mean Heart rate of patients 3min after Laryngoscopy and Induction in our study in Group A (IV) was74.9 ± 9.5 bpm and in Group B (NEBS) was 71.1 ± 8.22 bpm, Mean Heart rate of patients 5 min following Laryngoscopy and Induction in our study in Group A (IV) was73.02 ± 10.25 bpm and in Group B (NEBS) was 71.55 ± 8.73 bpm , Mean Heart rate of patients 10 min later Laryngoscopy and Induction in our study in Group A (IV) was69.67 ± 7.89 bpm and in Group B (NEBS) was 71.02 ± 7.96bpm. The Mean Heart Rate of individuals in Group B (NEBS) was smaller and statistically significant as collated to Group A (IV) After Laryngoscopy & intubation and at 1 min, 3 min and then difference in Heart Rate becomes insignificant.

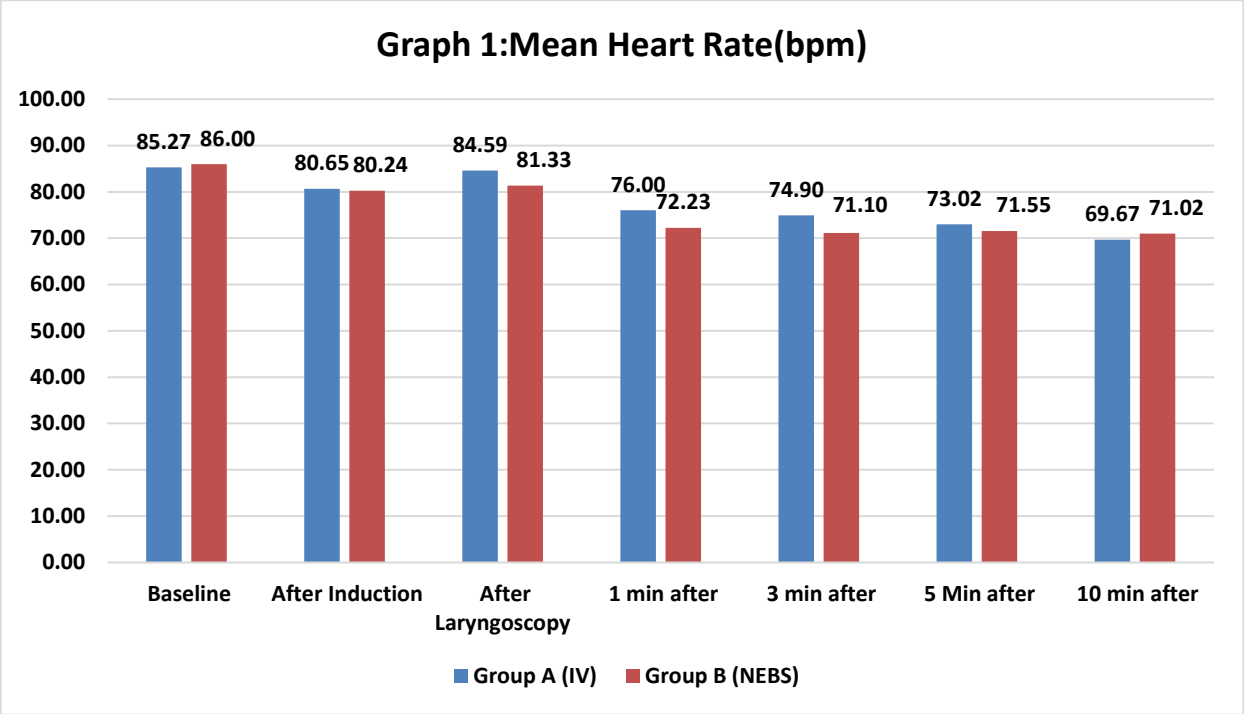


TABLE-2 Comparison of mean SBP (mmHg) at different time interval in Group A; Group B.

	Group A (IV)	Group B (NEBS)	
SBP (mmHg)	**Mean ± SD**	**Mean ± SD**	**P-Value**
Baseline	129.0 ± 7.92	130.43 ± 7.86	0.373#
After Induction	109.73 ± 8.71	110.2 ± 7.83	0.78#
After Laryngoscopy and intubation	125.76 ± 9.69	121.96 ± 8.17	0.038*
1 min after	116.37 ± 8.61	113.19 ± 5.97	0.036*
3 min after	110.84 ± 7.84	108.17 ± 5.20	0.049*
5 Min after	107.73 ± 8.15	109.02 ± 7.98	0.432#
10 min after	109.76 ± 7.62	109.27 ± 6.06	0.726#

#statistically insignificant; * statistically significant

The mean SBP(mmHg) of patients at baseline in our study in Group A (IV) was129.0 ± 7.92 mmHg and in Group B (NEBS) was 130.43 ± 7.86 mmHg, Mean SBP of patients After

Induction in our study in Group A (IV) was109.73 ± 8.71 mmHg and in Group B (NEBS) was 110.2 ± 7.83 mmHg, Mean SBP of patients after Laryngoscopy and Intubation in our study in Group A (IV) was125.76 ± 9.69 mmHg and in Group B (NEBS) was 121.96 ± 8.17 mmHg, Mean SBP of patients 1 min after Laryngoscopy and Intubation in our study in Group A (IV) was116.37 ± 8.61 mmHg and in Group B (NEBS) was 113.19 ± 5.97 mmHg , Mean SBP of patients 3 min after Laryngoscopy and Induction in our study in Group A (IV) was110.84 ± 7.84 mmHg and in Group B (NEBS) was 108.17 ± 5.20 mmHg, Mean SBP of patients 5 min after Laryngoscopy and Induction in our study in Group A (IV) was107.73 ± 8.15 mmHg and in Group B (NEBS) was 109.02 ± 7.98 mmHg, Mean SBP of patients 10 min after Laryngoscopy and Induction in our study in Group A (IV) was109.76 ± 7.62 mmHg and in Group B (NEBS) was 109.27 ± 6.06 mmHg. The Mean SBP of patients in Group B (NEBS) was not so much and statistically notable in contrast to Group A (IV) After Laryngoscopy, at 1 min & 3 min and then the difference in Mean SBP becomes insignificant.

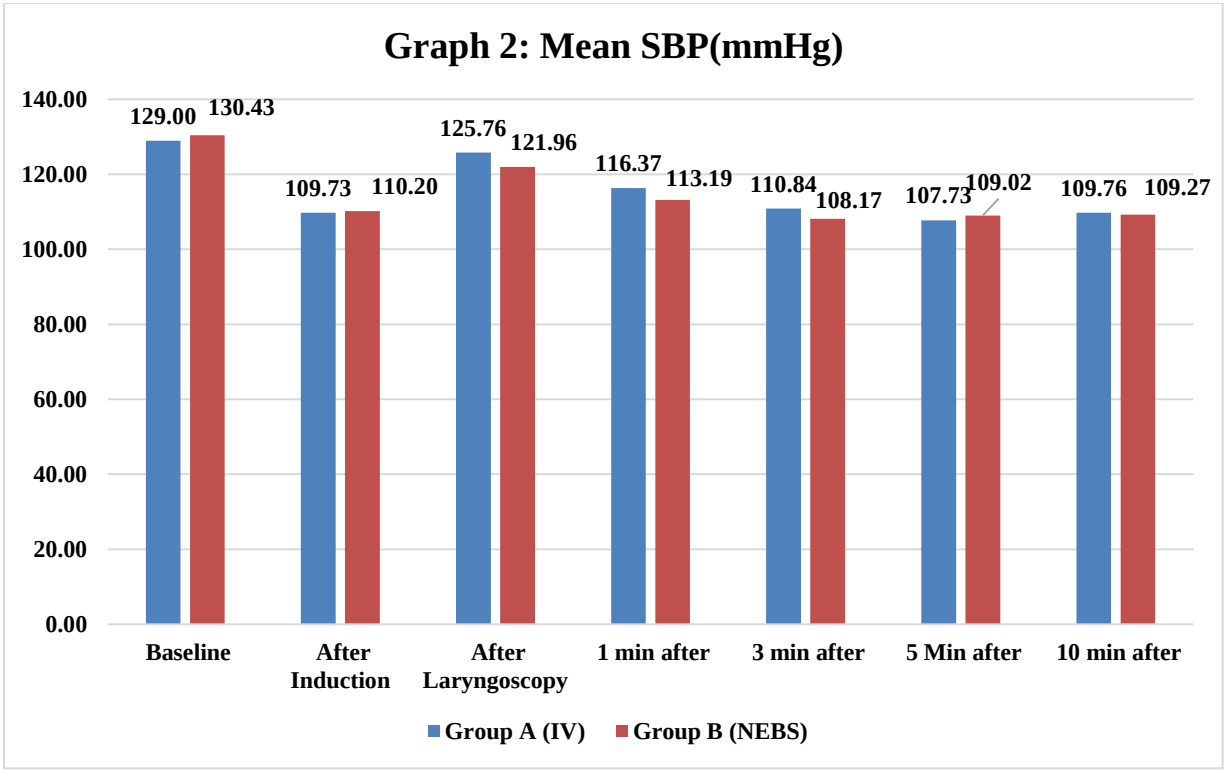


TABLE-3 Comparison of mean DBP (mmHg) at different time interval in Group A vs Group B.

	Group A (IV)	Group B (NEBS)	
DBP (mmHg)	**Mean ± SD**	**Mean ± SD**	**P-Value**
Baseline	79.57 ± 8.66	80.98 ± 8.63	0.422#
After Induction	65.82 ± 7.65	65.76 ± 7.71	0.969#
After Laryngoscopy and intubation	72.76 ± 9.03	71.51 ± 7.31	0.455#
1 min after	67.2 ± 8.7	66.53 ± 7.28	0.679#
3 min after	62.73 ± 6.75	63.98 ± 6.76	0.364#
5 Min after	62.16 ± 5.32	62.84 ± 6.99	0.593#
10 min after	65.49 ± 7.15	65.96 ± 5.89	0.724#

statistically insignificant.

The mean DBP(mmHg) of patients at baseline in our study in Group A(IV) was79.57 ± 8.66 mmHg and in Group B(NEBS) was 80.98 ± 8.63 mmHg, Mean DBP of patients After Induction in our study in Group A(IV) was65.82 ± 7.65 mmHg and in Group B(NEBS) was 65.76 ± 7.71 mmHg, Mean DBP of patients after Laryngoscopy and Intubation in our study in Group A (IV) was72.76 ± 9.03mmHg and in Group B (NEBS) was 71.51 ± 7.31 mmHg, Mean DBP of patients 1 min after Laryngoscopy and Intubation in our study in Group A (IV) was67.2 ± 8.7 mmHg and in Group B (NEBS) was 66.53 ± 7.28 mmHg , Mean DBP of patients 3min after Laryngoscopy and Induction in our study in Group A (IV) was62.73 ± 6.75 mmHg and in Group B (NEBS) was 63.98 ± 6.76 mmHg, Mean DBP of patients 5 min after Laryngoscopy and Induction in our study in Group A (IV) was62.16 ± 5.32 mmHg and in Group B (NEBS) was 62.84 ± 6.99 mmHg, Mean DBP of patients 10 min after Laryngoscopy and Induction in our study in Group A (IV) was65.49 ± 7.15 mmHg and in Group B (NEBS) was 65.96 ± 5.89 mmHg. There have been no notable differences in mean DBP of patients in between Group A (IV) and Group B (NEBS) at different time periods.

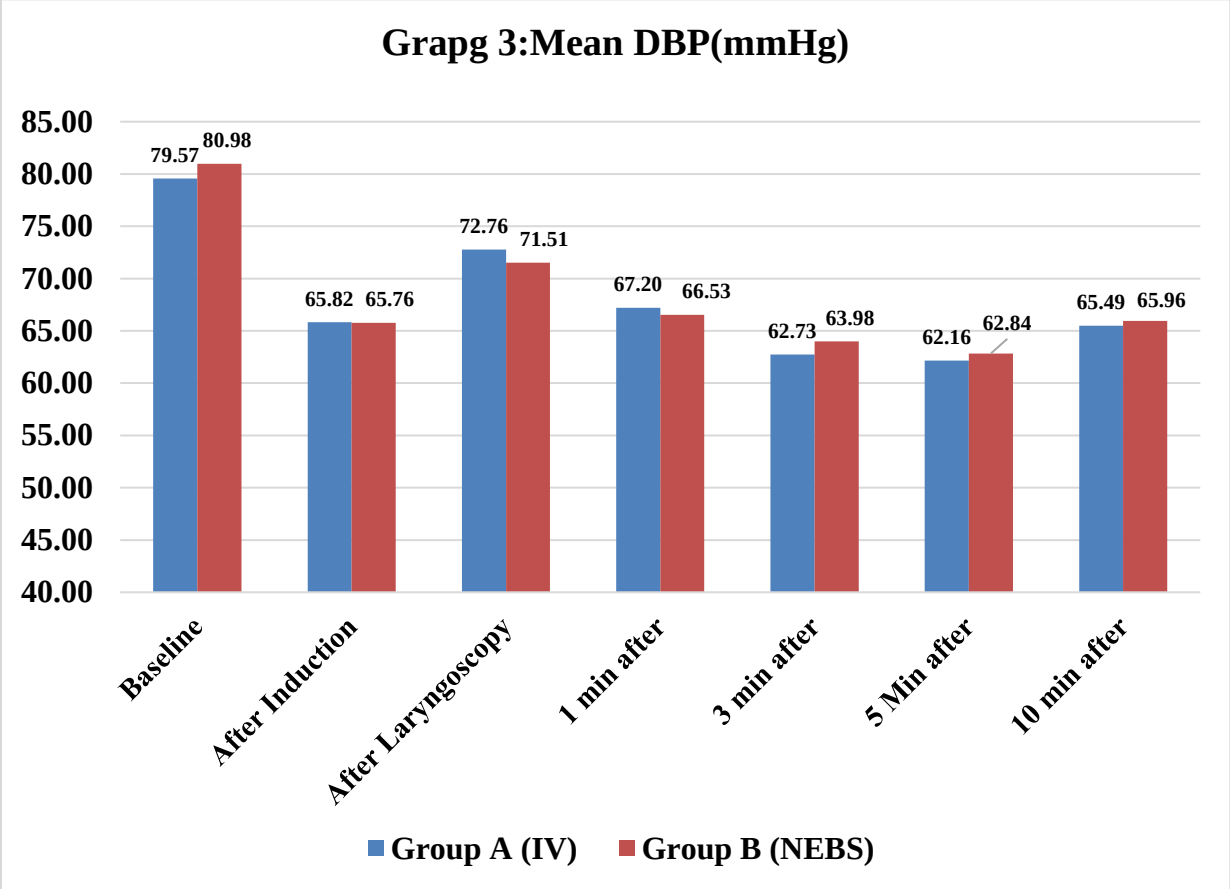


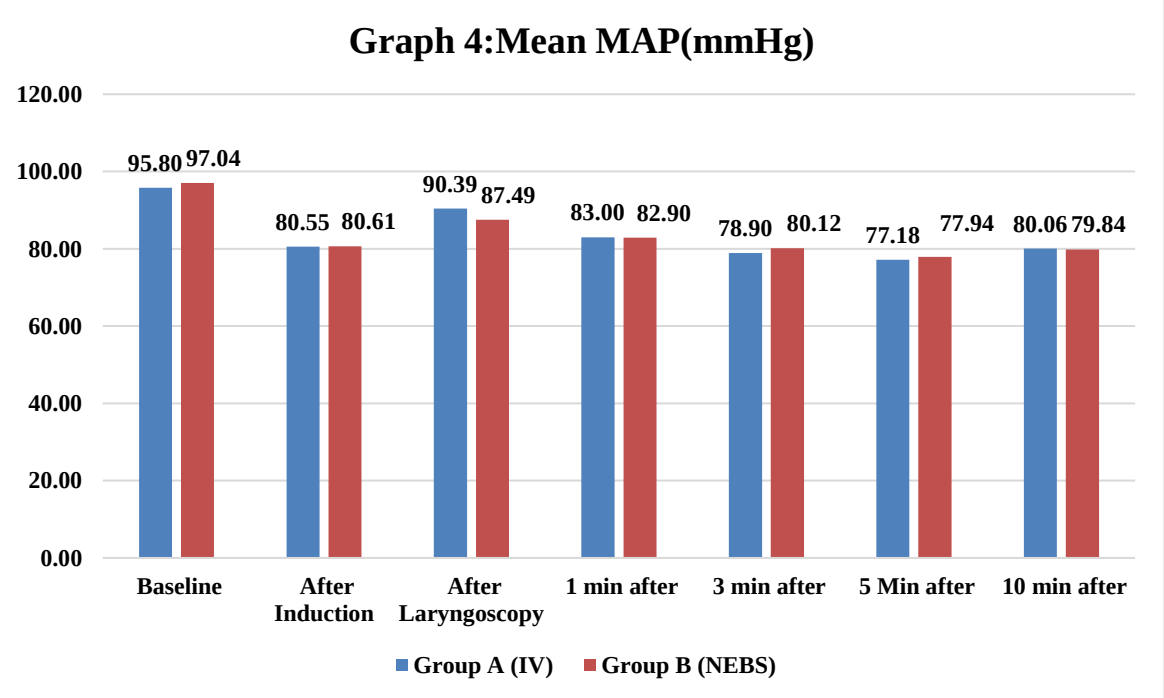
TABLE-4 Comparison of mean MAP (mmHg) at different time interval in Group A with Group B.

	Group A (IV)	Group B (NEBS)	
MAP (mmHg)	**Mean ± SD**	**Mean ± SD**	**P-Value**
Baseline	95.8 ± 7.5	97.04 ± 8.12	0.432#
After Induction	80.55 ± 7.47	80.61 ± 7.17	0.967#
After Laryngoscopy and intubation	90.39 ± 8.29	87.49 ± 4.41	0.043*
1 min after	83.0 ± 8.25	82.9 ± 6.32	0.945#
3 min after	78.9 ± 6.32	80.12 ± 6.33	0.34#
5 Min after	77.18 ± 5.37	77.94 ± 6.65	0.538#

10 min after	80.06 ± 6.47	79.84 ± 5.58	0.854#

#statistically insignificant; * statistically significant

The mean MAP of patients at baseline in our study in Group A (IV) was95.8 ± 7.5 mmHg and in Group B (NEBS) was 97.04 ± 8.12 mmHg, Mean MAP of patients After Induction in our study in Group A (IV) was80.55 ± 7.47 mmHg and in Group B (NEBS) was 80.61 ± 7.17 mmHg, Mean MAP of patients after Laryngoscopy and Induction in our study in Group A (IV) was90.39 ± 8.29 mmHg and in Group B (NEBS) was 87.49 ± 4.41mmHg, Mean MAP of patients 1 min after Laryngoscopy and Induction in our study in Group A (IV) was83.0 ± 8.25 mmHg and in Group B (NEBS) was 82.9 ± 6.32 mmHg , Mean MAP of patients 3min after Laryngoscopy and Induction in our study in Group A (IV) was78.9 ± 6.32 mmHg and in Group B (NEBS) was 80.12 ± 6.33 mmHg, Mean MAP of patients 5 min after Laryngoscopy and Induction in our study in Group A (IV) was77.18 ± 5.37 mmHg and in Group B (NEBS) was 77.94 ± 6.65 mmHg, Mean MAP of patients 10 min after Laryngoscopy and Induction in our study in Group A (IV) was80.06 ± 6.47 mmHg and in Group B (NEBS) was 79.84 ± 5.58 mmHg. The Mean MAP of patients in Group B (NEBS) was reduced and statistically noteworthy in resemblance to Group A (IV) after Laryngoscopy & intubation and then difference in Mean MAP becomes insignificant.



DISCUSSION

In the current analysis 98 patients were taken and split into 2 groups, which consisted of 49 patients each. With Group A (I.V Lignocaine) and Group B (Nebulised Lignocaine). All the demographic variables which were compared in this study came out to be insignificant**.**

Laryngoscopy & endotracheal man oeuvres significantly increase HR and BP, especially if the procedure takes more than 45 seconds.[4] As a result of this set of problems, the incidence of morbidity and death among patients with cardiovascular and cerebrovascular diseases may grow.[8]

In our study we found mean baseline HR were proportionate in both groups with p value 0.695. We documented our result after laryngoscopy and intubation, 1, 3 minutes after in Group B which was found statistically significant with p value is less than 0.05 as 0.004, 0.030, 0.036 respectively.Similar results were concluded by **Jokar *et al***[8] as he concluded that the average reduction of HR was quick in Group 1 (inhalation) than Group 2 (IV) and Control group. **Sklar *et al***[9] had found in his study that lidocaine 120mg inhalation was most effectual in preventing the HR response to laryngoscopy then lidocaine 1mg/kg iv which was statistically significant. **Ganesan *et al***[10]compared intravenous versus nebulized lignocaine and found that the increase in HR during endotracheal intubation was greater with IVL as compared to NL which came out to be statistically significant ($p < 0.05$).**Nabil *et al***[11] compared two group one which receives nebulised lignocaine 2% at a dose of 4.5mg/kg and second which receives 0.9 % NaCl and found out that the HR was significantly decreased in 1 and 3 min after tracheal intubation in lignocaine group rather that saline group with p values 0.041 and 0.042 respectively. **Agrawal *et al***[12] found in their study that group L (nebulized lignocaine) attenuate heart rate more effectively as measured to group C immediately after intubation, 2, 5 & 10 minutes after endotracheal intubation and the results were statistically significant with p value 0.044, 0.042, 0.046, 0.039 respectively.

Mean SBP was compared in both the groups and was found that Group B nebulised lignocaine attenuates SBP better than Group A intravenous lignocaine which was given 5 minutes prior to laryngoscopy and intubation with significant p values 0.038, 0.036, 0.049after laryngoscopy and intubation, 1, 3 minutes. **Gavale*et al***[13] found similar trend with significant reduction in systolic blood pressureoccurred in group A as collated with B group at 3 and 5 minutes after endotracheal intubation with p value < 0.05 as 0.0375, 0.0476 respectively. **Ganesan *et al***[10] concluded that upon intubation, there was a notable variation in the SBP levels among the groups.Compared to group NL, which reached baseline levels by approximately the third minute postintubation, the SBP in group IVL climbed considerably from baseline. The mean SBP was lower with NL than with IVL during the fourth and fifth minute. **Bhaskar *et al***[14] evaluated the consequences of intravenous and nebulized lignocaine on the inhibition of hemodynamic responses after tracheal intubation. The study included 40 patients who were randomly consigned to one of two groups: LI (intravenous lignocaine) or LN (nebulized lignocaine). They implemented that IV lignocaine

group had significantly higher SBP after intubation compared to the nebulized lignocaine group and that the nebulized lignocaine group significantly attenuate SBP with p value 0.003. **Ahmed Abdulmaged Ahmed, Hasan Sarhan Haider**[15] assess the effectiveness of sprayed and inhaled nebulized lidocaine and the observations were made that hemodynamic instability was less with nebulized lidocaine with statistically significant results in the 1st, and 3rd minute post intubation with p values 0.0163 and 0.0259 respectively.

In our study we found that both groups Group A and Group B were successfully attenuated the DBP but nebulised lignocaine attenuates DBP better than intravenous lignocaine however the results were statistically insignificant. **Sklar *et al***[9] established that the DBP of the control group climbed by 25.9 mmHg from the starting value, whereas the IV lidocaine group experienced a rise of 23.1 mmHg, the 40 mg nebulised group experienced a 22.1 mmHg increase, and the 120 mg inhalation group experienced a 13.6 mmHg increase and concluded that there was no statistically significant attenuation of the blood pressure response in the IV lidocaine and lidocaine 40 mg inhalation groups. **Gupta *et al***[16] and **Ganesan *et al***[10] also concluded that the results were not assuring and diastolic blood pressure increases when intubation was done in patients with nebulized lignocaine group.

In our study baseline MAP was comparable between both groups which was 95.8 ± 7.5 mmHg in group A and 97.04 ± 8.12 mmHg in group B with p value 0.432. Both the route of administration of lignocaine reduced MAP in the trial but in patients with nebulised lignocaine reduction in MAP was statistically significant after laryngoscopy and intubation with p value less than 0.05 which was 0.043. **Gavale*et al***[13] found equivalent results as baseline MAP between both groups was comparable (p value 0.369) and suggested that at 3 and 5 minutes following endotracheal intubation, patients in group A (nebulized lignocaine) show a statistically significant reduction when evaluated with group B (intravenous lignocaine). **Jokar *et al***[2] implied that compared to the other groups, Group 1's average MAP drop occurred more quickly. Group 1's (inhalation) MAP was clinically smaller than Group 2's (IV lignocaine); nevertheless, there was no statistically significant difference among the two groups (P = 0.116). In contrast to the control group, there was a noticeable difference observed between these two groups. **Sklar *et al***[9] proposed that increment in MAP was significant in the groups receiving lidocaine 40mg by inhalation, saline control, and iv lidocaine (22.9 mmHg, 29.2 mmHg, 21.1mmHg, respectively). When compared to the other groups, the rise in the lidocaine 120 mg inhaled group was significantly smaller ($p < 0.05$).

CONCLUSION

We conclude from our report that the hemodynamic variables were more stable in nebulized lignocaine group and it was seen that nebulized lignocaine attenuates the pressor responses much better than intravenous lignocaine with less hemodynamic instability.

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