https://doi.org/10.48047/AFJBS.6.15.2024.1861-1874

African Journal of Biological Sciences

Journal homepage: http://www.afjbs.com

Research Paper Open Access

Compare the Efficacy between Nebulized Dexmedetomidine with Lignocaine and Plain Lignocaine For Attenuation of Hemodynamic Response to Laryngoscopy and Endotracheal Intubation – Randomized Double-Blind Study

Dr. Arpita Mohanty[1], Dr. B. Anusha[2]*, Dr. Jagan, G[2], Dr. Arun Sampath[3]

[1]Shri SathyaSai Medical College and RI, Sri BalajiVidyapeeth (Deemed to be a University), Ammapettai, KanchipuramDistrict, Nellikuppam, Tamil Nadu 603108, India

[2]Associate Professor, ShriSathyaSai Medical College and RI, Sri BalajiVidyapeeth (Deemed to be a University), Ammapettai, KanchipuramDistrict, Nellikuppam, Tamil Nadu 603108, India

[3]Senior Consultant, Department of NeuroAnaesthesia, Dr. AchantaLakshmipathi Neurosurgical Centre and Voluntary Health services (VHS), Chennai 600113, India

Corresponding author- **Dr. B. Anusha***

Volume 6, Issue 15, Sep 2024

Received: 15 July 2024

Accepted: 25 Aug 2024

Published: 05 Sep 2024

doi: 10.48047/AFJBS.6.15.2024.1861-1874

Abstract: ***Background and Aim*:** Hemodynamic responses [Systolic blood pressure (SBP); Diastolic blood pressure (DBP); mean arterial pressure (MAP); Spo2] during laryngoscopy and intubation are a significant concern for the anesthesiologist. This study was aimed to compare the effects of nebulisation of Dexmedetomidine and Lignocaine and plain lignocaine for control of hemodynamic response to laryngoscopy and intubation when used in combination or alone**.** ***Materials and Methods*:** This was a prospective Randomised double-blind study where in 60 patients undergoing surgeries under general anaesthesia were allocated into two groups.1) Group A- will receive pre-operative nebulisation with Dexmedetomidine + Lignocaine**.** 2) Group B- will receive pre-operative nebulisation with lignocaine aloneHemodynamic response to the laryngoscope and intubation was compared and recorded. ***Results:***The heart rate, SBP, DBP, and spo2, MAP was very well controlled in group A in comparison with group B which is statistically significant (P value: 0.011, 0.031, 0.031& 0.003 respectively). ***Conclusion****:* In comparison to nebulisation lignocaine alone, the combination of nebuliseddexmedetomidine and lignocaine significantly attenuates the pressure response during laryngoscopy and intubation without any hemodynamic side effects.

Keywords: Nebulisation, lignocaine, dexmedetomidine, intubation, laryngoscopy.

INTRODUCTION

The majority of patients who will be administered general anesthesia during their surgical procedure will need a laryngoscopy and intubation. The most effective method for controlling airways is endotracheal intubation. Since it prevents aspiration of gastric contents, also helps in the administration of anesthetic gases, and facilitates positive pressure breathing even at elevated airway pressures.

Laryngoscopy and endotracheal intubation might elicit 10-minute acute hemodynamic responses (HDR) [1, 2]. Stretching the laryngeal and pharyngeal mucosal tissues activates the sympathetic nervous system, which in turn causes catecholamine release, which in turn causes tachycardia, hypertension, and even ischemic changes like ST-segment abnormalities [3, 4].

Those without a poor cardiac reserve are unharmed by these responses, while those with one will suffer serious consequences. It raises intracranial pressure in those with decreased compliance and significantly impacts those with severe

cardiac disease. Heart problems, abnormal heart rhythms, changes in myocardial ischemia, myocardial infarction, cerebral hemorrhage, and hypertensive crisis are among the adverse hemodynamic responses that might result during intubation and laryngoscopy. Hence, it's crucial to control these responses. An ideal medicine would be one that is quickly effective, simple to give, and has few unwanted side effects.

In this endeavor, many medications have been employed in different ways. Many drugs have been studied for their ability to deepen anesthesia [5, 6]. Some examples of these medications are intravenous sodium nitroprusside, beta-blockers, lidocaine, sodium channel blockers, opioids, and vasodilators. Unfortunately, none of these effectively reduce the sympathetic nervous system's reaction.

One way to reduce the intensity of this pressure reaction is to keep the laryngoscopy duration to 15 seconds. Alfentanil (80-100 μg/kg), morphine (0.2 mg/kg), β blockers like lignocaine and esmolol, and low dosage opioids like fentanyl and sufentanil are some other methods. Applying a local anesthetic to the skin before a laryngoscopy or endotracheal intubation procedure might assist reduce heart rate by blocking airway reflexes.

Among the class of local anaesthetics known as amides, lignocaine stands out as an aminoethyl amide [7]. Since 1948, it has been the gold standard for local anesthesia. Bromage found in 1961 that IV hydration lowered intubation pressure [8]. It is recommended that lignocaine be administered intravenously at a dose of 1.5 mg/kg three minutes before to intubation for optimal results.

The α2 adrenergic agonist dexmedetomidine exerts effects that are sedative, anxiolytic, sympatholytic, and analgesic [9]. It provides opioid sparing analgesic effects and dose-dependently decreases sympathetic outflow from the central nervous system. Dexmedetomidine is rapidly replacing all other anesthetics as the gold standard due to its low risk of side effects. When administering dexmedetomidine, it is recommended to suppress the airway and circulatory reflexes during intubation and laryngoscopy [10]. However, it is important to note that there have been cases of bradycardia and hypotension.

Our hypothesis was that by combining the two drugs, we may potentially lower the dose required for each one while also reducing the intubation response more effectively. This research compared lignocaine nebulization with a combination of the two medications in reducing intubation hemodynamic response.

METHODS

The study involved 60 patients from both sexes of the American Society of Anaesthesiologist I or II, age and group of 18 to 60 years, BMI between 18.5 to 24.9 kg/m2 those who underwent any procedure in general surgery, obstetrics and gynaecology and orthopaedics. All included patients were randomly divided into two groups with 30 into each by a computer-generated list of random numbers. Patients and the primary assessor were blinded to the study, anaesthetised, and monitored by the anaesthesiologist who was also blinded to the study, that person was monitoring the parameters during the procedure. The study Nebulized Dexmedetomidine with Lignocaine was prepared by the OT assistant, who was not otherwise involved in the study, as Nebulized dexmedetomidine with lignocaine and plain lignocaine. In the end the recorded date was delivered in a sealed envelope to the primary investigator. Patient coming under American Society of Anaesthesiologist III and above, BMI >30 kg/m2. Emergency procedures, pregnancy, subject on anti-hypertensive medications, seizure disorder, known allergy to Dexmedetomidine and or lignocaine were excluded from the study. The sample was calculated before patient's recruitment.

SAMPLE SIZE CALCULATION

Sample size calculation is based on the previous study [11] the mean and standard deviation of both the groups nebulisation with dexmedetomidine + lignocaine and nebulisation with lignocaine alone for attenuation of Hemodynamic response to Laryngoscopy and Endotracheal intubation. The difference in the mean of heart rate, blood pressure, SPO2 from the baseline and one minute after intubation was used to calculate the effect size of this study; which are 91 +/- 7.8 and 80.5 +/- 7.09 with 5% level of significance and 80% power the total sample size is 30 in each group includes 10% non-response error.

DATA COLLECTION PROCEDURE

After the approval of the institution's ethics committee, patients were randomly allocated into two groups via computer generated group of numbers.

- ✓ **Group A**- Received pre-operative nebulisation with Dexmedetomidine (1mcq/kg) + 2% Lignocaine (5ml)-diluted to total volume of 5ml with Normal Saline.
- ✓ **Group B**-Received pre-operative nebulisation with 2% lignocaine (5ml) alone.
- ✓ Same electric nebulizer was used to nebulise the patient of both groups.
- ✓ Both groups receive nebulisation 30 minutes prior to induction.
- ✓ On the day of surgery after securing 18G IV venflon, all patients were connected with multipara monitors and Patients HR; SBP; DBP; SPO2; MAP were recorded. Patient was Pre-medicated with inj Midazolam 1mg IV, injGlycopyrolate .004mg/kg IV, inj Fentanyl 2mcg/kg IV after adequate preoxygenation with 100% oxygen 6-7 litre, patient was induced with injPropofol 1-2 mg/kg IV titrated to loss of verbal response (dose of propofol consumed was noted) .Once adequate bag and mask ventilation was confirmed with good chest rise, patient was paralysed with inj. vecuronium 0.1mg/kg IV after 5 mins patient was intubated with appropriate size ETT followed by intermittent Injvecuronium at its maintaince dose.

- ✓ Systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate, were noted at 1,3,5,10 minutes after intubation, depth of anaesthesia will be maintained by isoflurane in 50% oxygen-nitrous oxide mixture, ventilation was adjusted to maintain etco2 of 35-45mmhg.
- ✓ Reversal with neostigmine (0.05mg/kg) + glycopyrrolate (0.01mg/kg) and patient was completely awake with good muscle tone and airway reflexes fully intact patient was extubated and shifted out of OT.
- ✓ Post operative complications like sore throat, post operative nausea and vomiting, cough was noted.

Statistical Analysis

After completion of the study, data was collected and analysed using proper statistical software using SPSS 23 software. Continuous variables such as blood pressure, heart rate, SPO2 were compared between two groups using unpaired T- test.

RESULTS

In this study involved 60 patients from both gender of the American Society of Anaesthesiologist 1 or 2, age and group of 18 to 60 years, BMI between 18.5 and 24.9 kg/m2 undergoing any procedure in general surgery, obstetrics and gynaecology and orthopaedics. All included patients were randomly divided into two groups with 30 into each by a computer-generated list of random numbers. Patients were blinded to the study, anaesthetised, and monitored by the anaesthesiologist who is also blinded to the study, that person will monitor the parameters during the procedure. The study Nebulized Dexmedetomidine with Lignocaine was prepared by an assistant, who is not otherwise involved in the study, as Nebulized dexmedetomidine with lignocaine and plain lignocaine. In the end the recorded date was delivered in a sealed envelope to the primary investigator.

1 Age distribution of the Patients

Table 1: Age distribution of the patients among the group

AGE	Group A	Percentage	Group B	Percentage
20-30	13	43.33	13	43.33
31-40	6	20	7	23.33
41-50	6	20	5	16.67
51-60	5	16.67	5	16.67
p-value	0.982			

The age distribution of the patients was observed and tabulated in Table 6.1. In both groups, the higher no. of patients was observed in the age group of 20-30 years. The comparison of age distribution of the patients among the group were graphically represented in Figure 7.1. Very lower no. of patients was observed in the age group of 51-60 years. The probability value of the age distribution was calculated, p-value is 0.982 > 0.05 statistically not significant were observed.

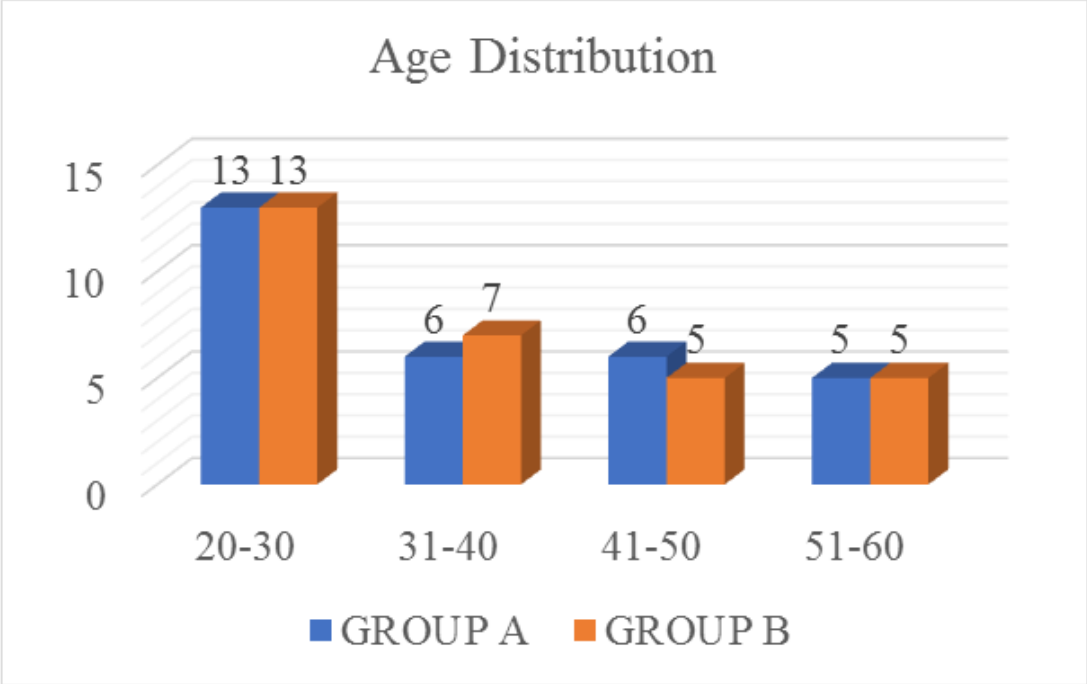

Figure 1: Comparison of age distribution of the patients among the group

Table 2: Mean age distribution of the patients among the group

Age Distribution	Group A	Group B
Mean	36	34.7
SD	11.26	11.52
P Value	0.336	

Mean age distribution of the patients were calculated and tabulated in Table 2 and represented in Figure 2. In group A, the mean age distribution of the patients is 36±11.25 years were observed. In group B, the mean age distribution of the patients is 34.7±11.52 years were observed; the probability value of mean age distribution is 0.336 > 0.05 statistically not significant were observed.

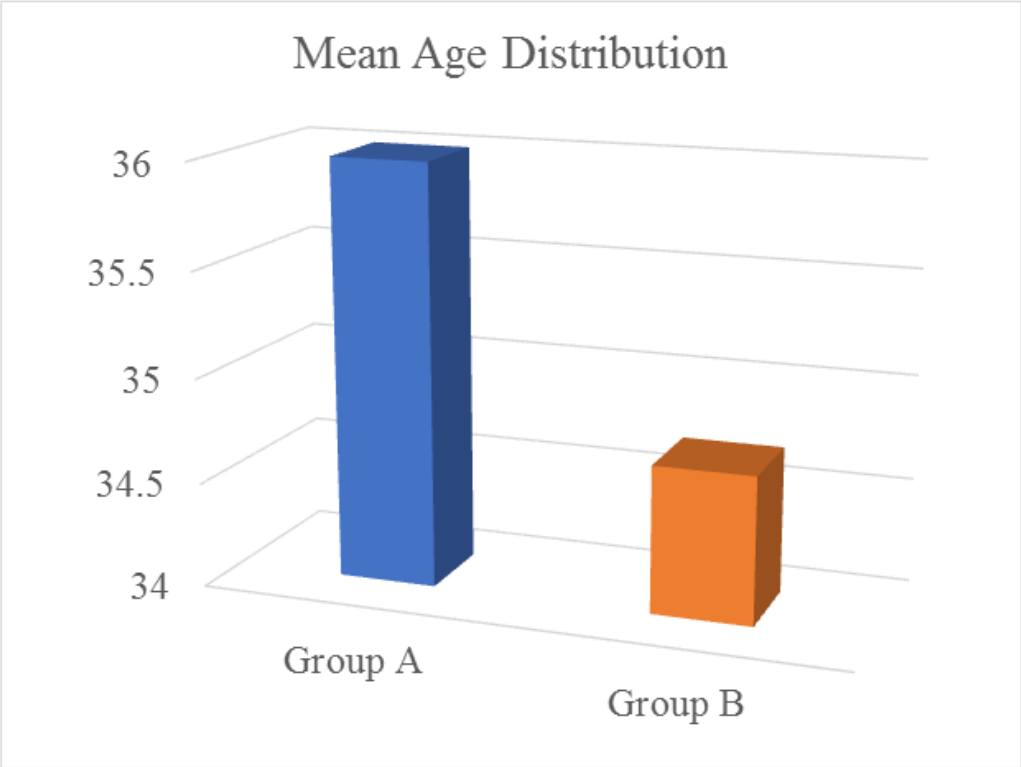

Figure 2: Comparison of mean age distribution among the group

2 Distribution of the gender of patients

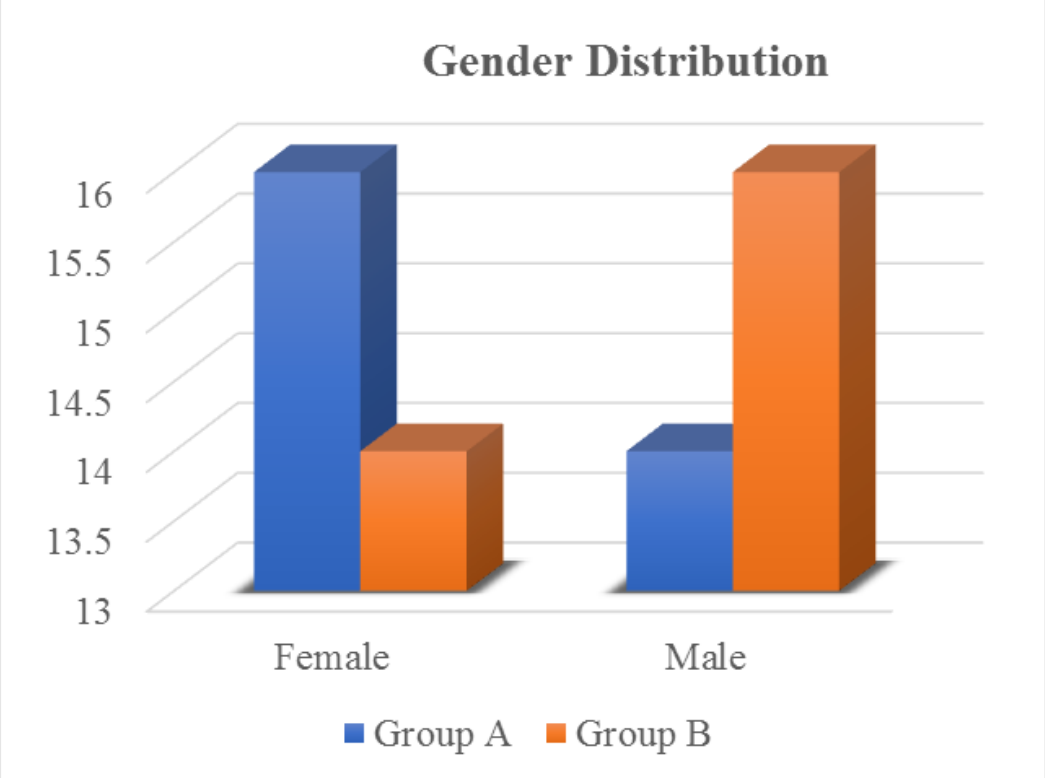

Figure 3: Comparison gender distribution of the patients among the group

The gender distribution of the patients among the group were observed and given in Table 3 & Figure 3. In group A, 16 male patients and 14 female patients were observed. In group B, 14 male patients and 16 female patients were observed.

Table 3: Gender distribution of the patients among the group

Gender Distribution	Group A	Percentage	Group B	Percentage
Female	16	53.33	14	46.67
Male	14	46.67	16	53.33

3 ASA Status of the Patients

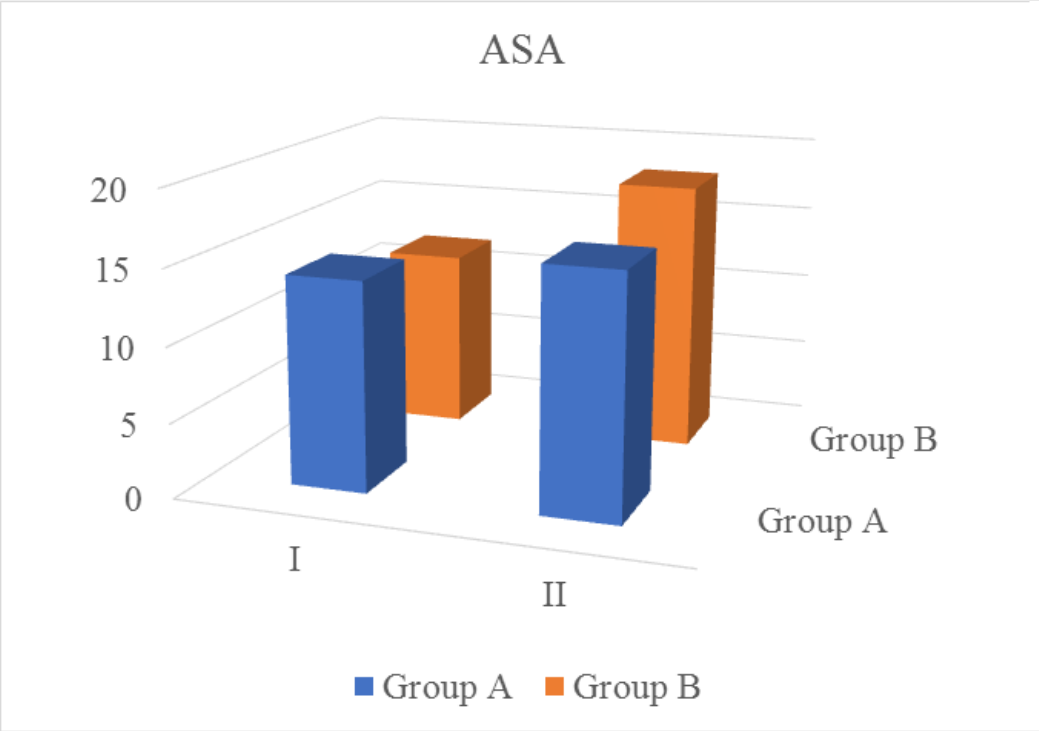

Figure 4: Comparison of ASA status of the patients among the group

ASA status of the patients was observed and given in Table 4 and it is graphically represented in Figure 4. In group A, 46.67 percentages of patients are in ASA I and remaining 53.33 percentages of patients are in ASA II. In group B, 40 percentages of patients are in ASA I and remaining 60 percentages of patients are in ASA II were observed; p-value is 0.602 > 0.05 statistically not significant were observed.

Table 4: ASA status of the patients among the group

ASA	Group A	Percentage	Group B	Percentage
I	14	46.67	12	40
II	16	53.33	18	60
P value	0.602			

4 MallampatiClassification of the Patients

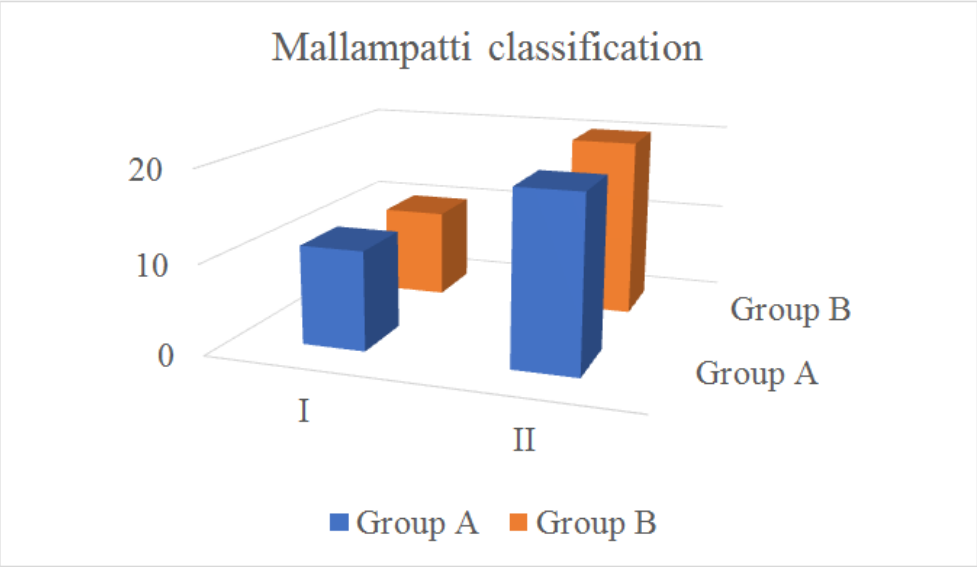

Figure 5: Comparison of Mallampati classification of the patients among the group

Table 5: Mallampati classification of the patients among the group

MPC	Group A	Percentage	Group B	Percentage
I	11	36.67	10	33.33
II	19	63.33	20	66.67
P value	0.786			

In this study, the Mallampati classification of the patients were calculated and mentioned in Table 5 and Figure 5. In group A, 36.67 percentages of patients are in MPC I and remaining 63.33 percentages of patients are in MPC II. In group B, 33.33 percentages of patients are in MPC I and remaining 66.67 percentages of patients are in MPC II were observed; p-value MPC is 0.786 > 0.05 statistically not significant were observed.

5 BMI value of the patients

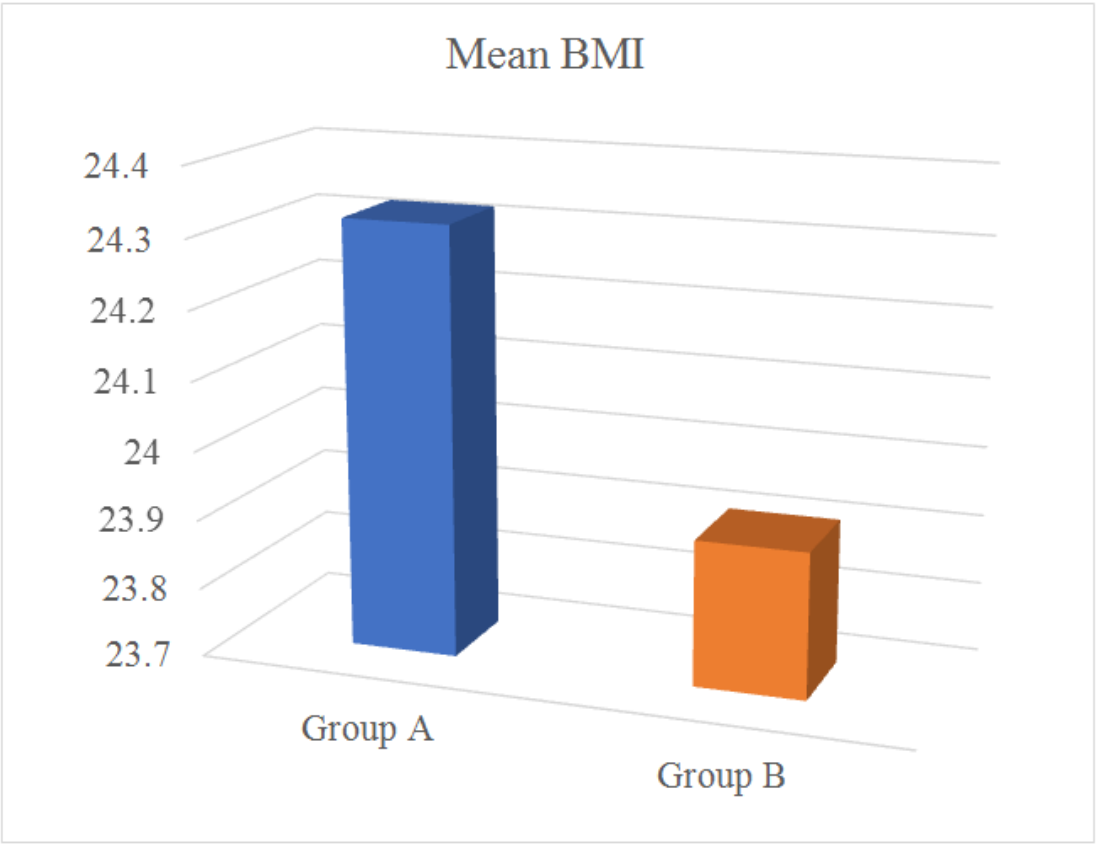

Figure 6: Comparison of mean BMI value of the patients among the group

Table 6: Mean BMI value of the patients among the group

BMI	Group A	Group B
Mean	24.32	23.91
SD	2.42	2.49
P Value	0.264	

Mean BMI value of the patients was calculated and given in Table 6 and it is graphically represented in Figure 6. In group A, the mean BMI value is 24.32±2.42 kg/m2 were observed. In group B, the mean BMI value is 23.91±2.49 kg/m2 were observed, the p-value of mean BMI is 0.264 > 0.05 statistically not significant.

6 Comparison of heart rate of the patients

Table 7: Comparison of heart rate of the patients among the group

Heartrate	Group A		Group B		T value	P value	
	Mean	SD	Mean	SD			
BASELINE	84.36	3.88	87.26	9.78	1.48	0.071	Not Significant
PREMEDICATION	79	5.8	85.96	11.99	-2.82	0.00033	Significant
INDUCTION	79.16	5.13	84.36	9.5	2.59	0.006	Significant
INTUBATION	77.06	5.35	80.1	4.54	2.32	0.011	Significant
1 MINUTES	79.86	4.6	84.93	8.51	-2.81	0.0032	Significant
3 MINUTES	78.1	5.07	83.03	12.6	-1.95	0.027	Significant
5 MINUTES	79.63	4.9	85.16	11.67	-2.35	0.011	Significant
10 MINUTES	83.63	10.41	87.3	2.25	1.85	0.034	Significant

The mean heart rate of the patients was observed with different interval given in Table 7. Comparison between group A and group B, the higher mean heart was observed in group B; the major difference was not observed and almost closer value was observed and it is graphically represented in Figure 7. The p-value of mean heart rate is < 0.05 statistically significant were observed in all interval of time.

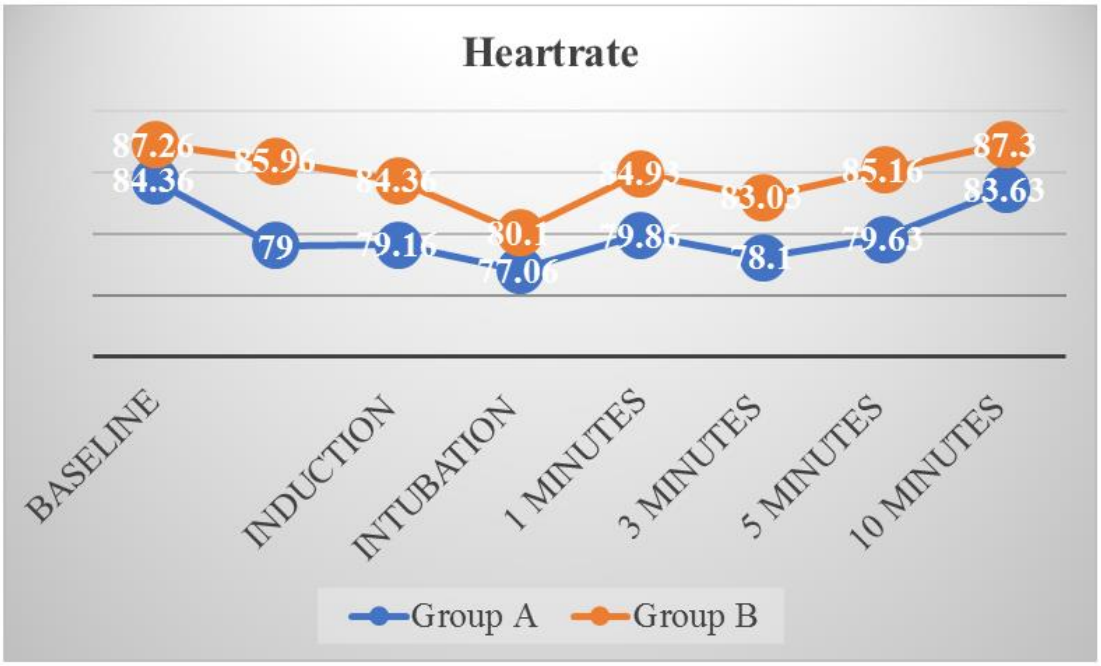

Figure 7: Comparison of heart rate of the patients among the group

7 Comparison of SBP of the patients

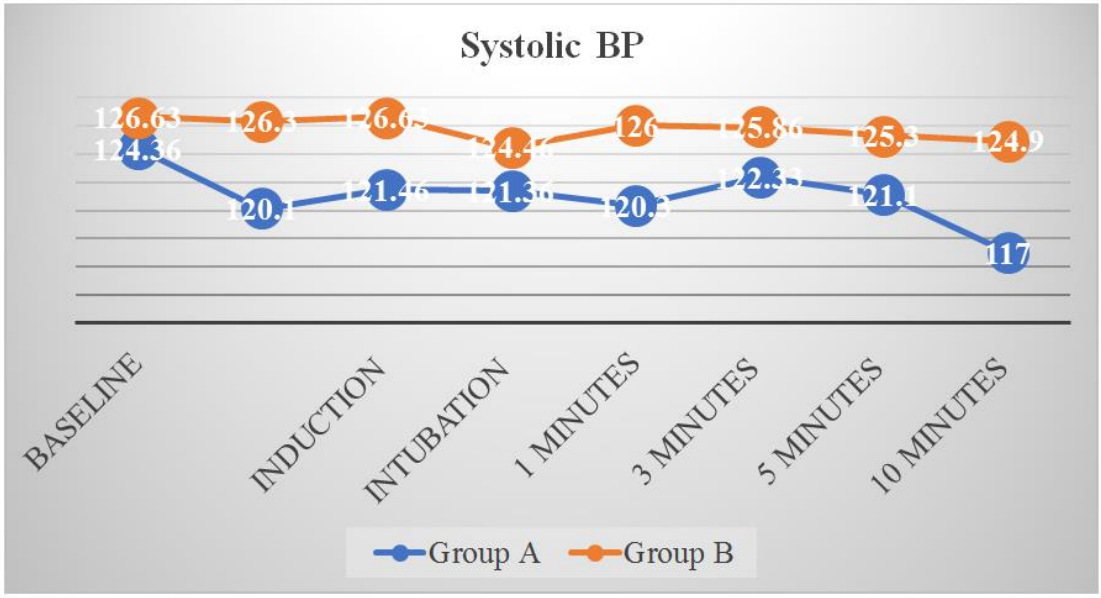

Figure 8: Comparison of SBP of the patients among the group

Table 8: Comparison of SBP of the patients among the group

SBP	Group A		Group B		T value	P value	
	Mean	SD	Mean	SD			
BASELINE	124.36	9.2	126.63	9.76	-0.909	0.183	Not Significant
PREMEDICATION	120.1	7.62	126.3	5.44	-3.56	0.00037	Significant
INDUCTION	121.46	9.01	126.63	8.65	2.22	0.014	Significant
INTUBATION	121.36	9.78	124.46	8.83	1.89	0.031	Significant
1 MINUTES	120.3	8.41	126	9.74	2.38	0.01	Significant
3 MINUTES	122.33	9.91	125.86	6.61	1.91	0.03	Significant
5 MINUTES	121.1	10.4	125.3	7.95	-1.727	0.044	Significant
10 MINUTES	117	7.27	124.9	8.7	-3.75	0.0002	Significant

The mean systolic blood pressure of the patients was calculated with different time and given in Table 8. Comparison between group A and group B, the higher mean SBP were observed in group B and it is graphically represented in Figure 8. The probability value of the mean SBP were calculated; p-value is < 0.05 statistically significant was observed exempt baseline.

8 Comparison of DBP of the patients

The mean diastolic blood pressure of the patients was calculated with different time and given in Table 9. Comparison between group A and group B, the higher mean DBP were observed in group B; the slight variation was observed and it is graphically represented in Figure 9. The probability value of the mean DBP was calculated; p-value is < 0.05 statistically significant was observed exempt baseline.

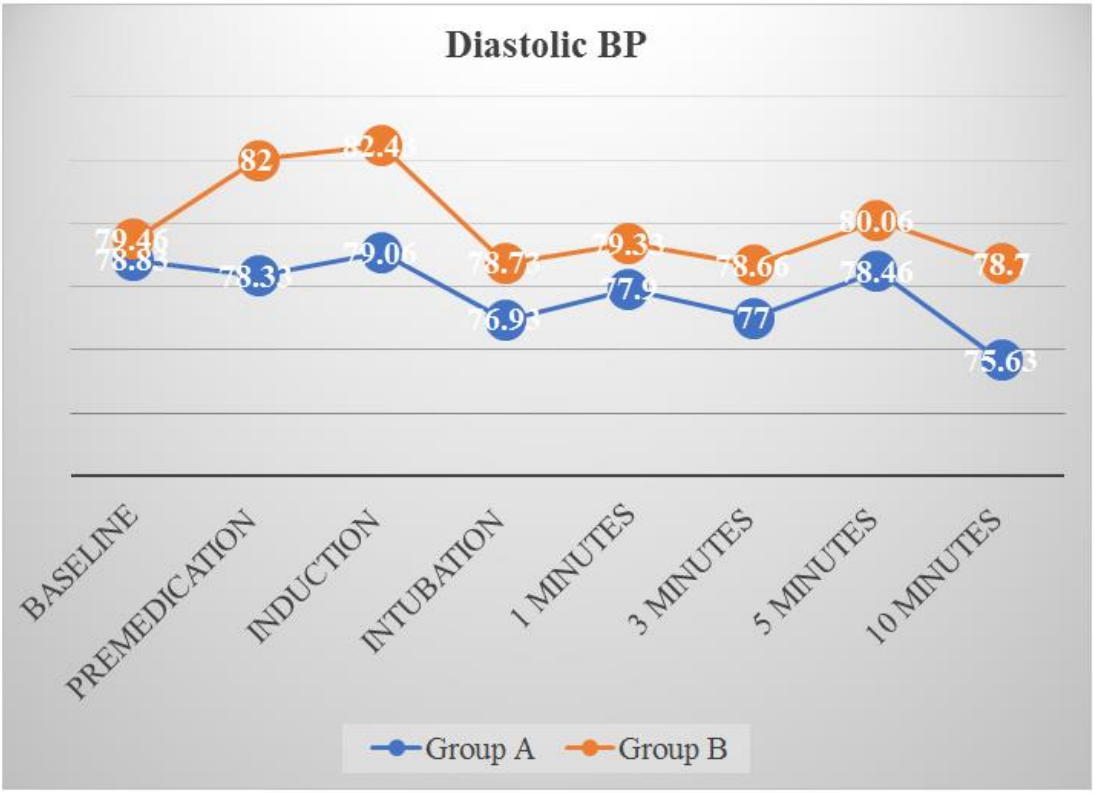

Figure 9: Comparison of DBP of the patients among the group

Table 9: Comparison of DBP of the patients among the group

DBP	Group A		Group B		T value	P value	
	Mean	SD	Mean	SD			
BASELINE	78.83	2.17	79.46	2.18	1.1	0.136	Not Significant
PREMEDICATION	78.33	2.76	82	3.97	-4.07	0.0007	Significant
INDUCTION	79.06	2.11	82.43	4.21	-3.84	0.00015	Significant
INTUBATION	76.93	3.75	78.73	2.01	-2.27	0.031	Significant
1 MINUTES	77.9	2.84	79.33	2.42	2.06	0.021	Significant
3 MINUTES	77	3.16	78.66	2.34	-2.28	0.013	Significant
5 MINUTES	78.46	3.11	80.06	3.29	-2.22	0.014	Significant
10 MINUTES	75.63	3.11	78.7	2.35	4.23	0.00004	Significant

9 Comparison of MAP of the patients

Table 10: Comparison of MAP of the patients among the group

MAP	Group A		Group B		T value	P value	
	Mean	SD	Mean	SD			
BASELINE	93.9	3.26	95.03	3.48	-1.27	0.103	Not Significant
PREMEDICATION	92.3	3.46	96.63	3.07	5.03	<0.00001	Significant
INDUCTION	93.16	3.09	97.06	4.68	3.73	0.0002	Significant
INTUBATION	91.73	3.85	94.3	3.04	2.81	0.003	Significant
1 MINUTES	91.96	3.09	94.73	2.99	3.45	0.0005	Significant
3 MINUTES	91.93	4.07	94.56	2.3	3.02	0.0018	Significant
5 MINUTES	92.46	4.01	80.06	3.29	-2.68	0.004	Significant
10 MINUTES	89.4	3.36	94	3.21	-5.32	<0.00001	Significant

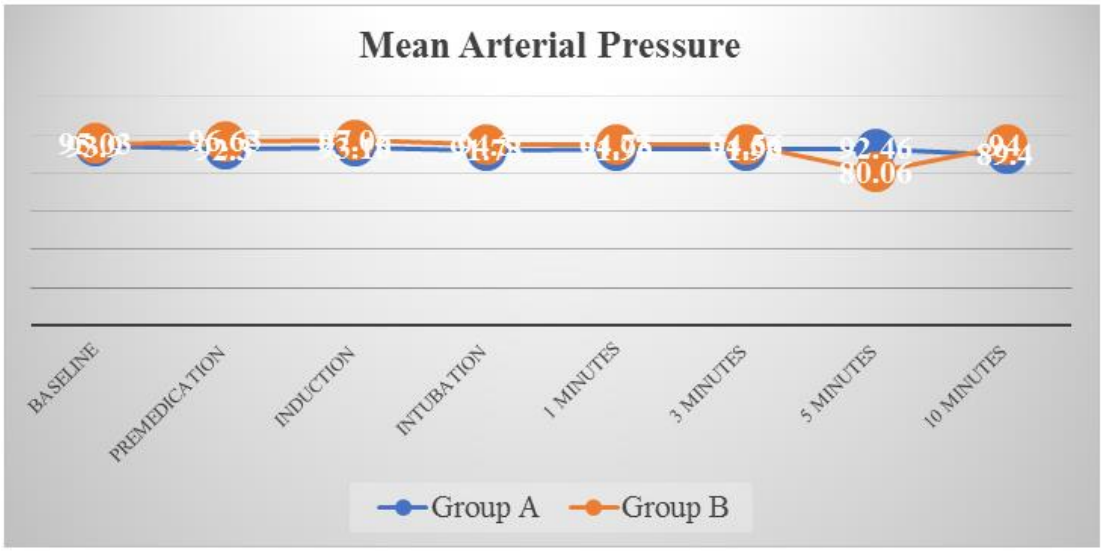

Figure 10: Comparison of MAP of the patients among the group

The mean arterial pressure of the patients was calculated with different time and given in Table 10. Comparison between group A and group B, the higher mean MAP was observed in group B; the slight variation was observed and it is graphically represented in Figure 10. The probability value of the mean MAP was calculated; p-value is < 0.05 statistically significant was observed exempt baseline.

10 Comparison of SPO2 of the patients

The mean SPO2 of the patients were calculated with different time and given in Table 11. Comparison between group A and group B, both groups having similar effects was observed in this study, slight variation was observed and it is not significant concern and it is represented in Figure 11. The probability value of the mean SPO2 was calculated; p-value mean SPO2 is > 0.05 statistically not significant was observed in all time.

Table 11: Comparison of SPO2 of the patients among the group

SPO2	Group A		Group B		T value	P value	
	Mean	SD	Mean	SD			
BASELINE	98.96	0.707	99.03	0.835	-0.328	0.372	Not Significant
PREMEDICATION	99.1	0.718	99.1	0.83	0.163	0.435	Not Significant
INDUCTION	99.03	0.706	98.9	0.65	0.747	0.228	Not Significant
INTUBATION	98.96	0.75	98.76	0.61	-1.108	0.136	Not Significant
1 MINUTES	99.06	0.727	98.93	0.512	-1.12	0.133	Not Significant
3 MINUTES	99.4	0.711	99	0.632	1.42	0.079	Not Significant
5 MINUTES	99.3	0.66	99.1	0.39	-1.31	0.096	Not Significant
10 MINUTES	99.16	0.73	99.1	0.47	-0.41	0.341	Not Significant

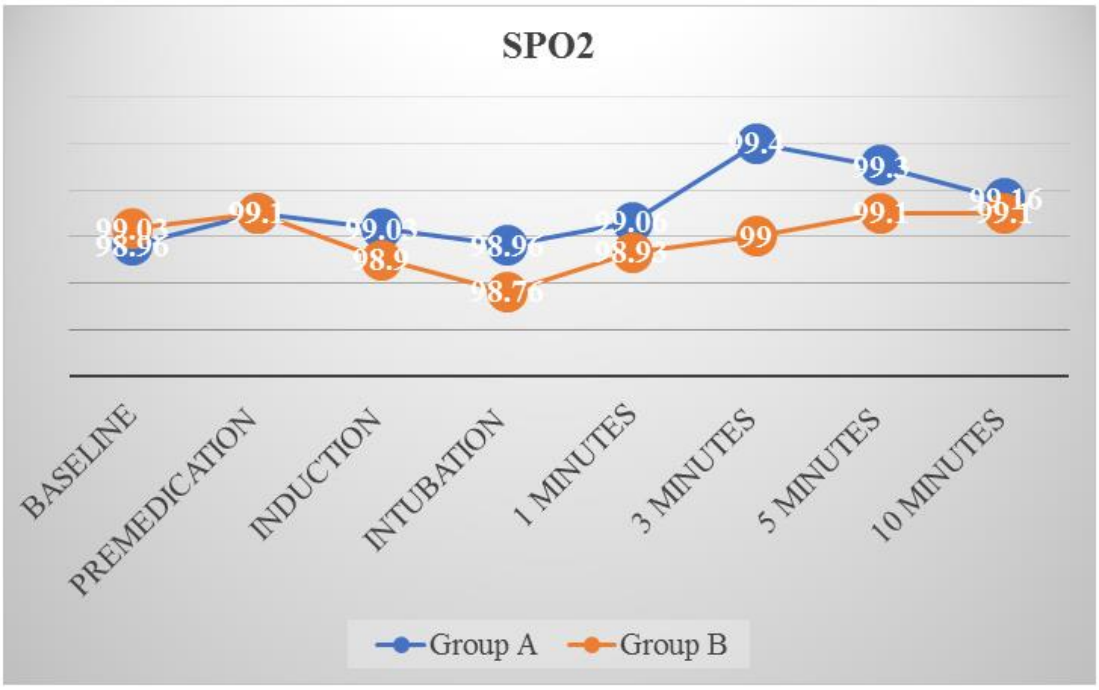

Figure 11: Comparison of SPO2 of the patients among the group

11 Comparison of propofol dose

Comparison of propofol dose used in groups compared and represented in Table 12 and Figure 12. Mean propofol dose in group A was 85±6.16mg and in group B was 99.67±11.89mg.

Table 12: Comparison of propofol dose between groups

PROPOFOL DOSE	GROUP A	GROUP B
MEAN	85	99.67
SD	6.16	11.89
P VALUE	<0.00001	

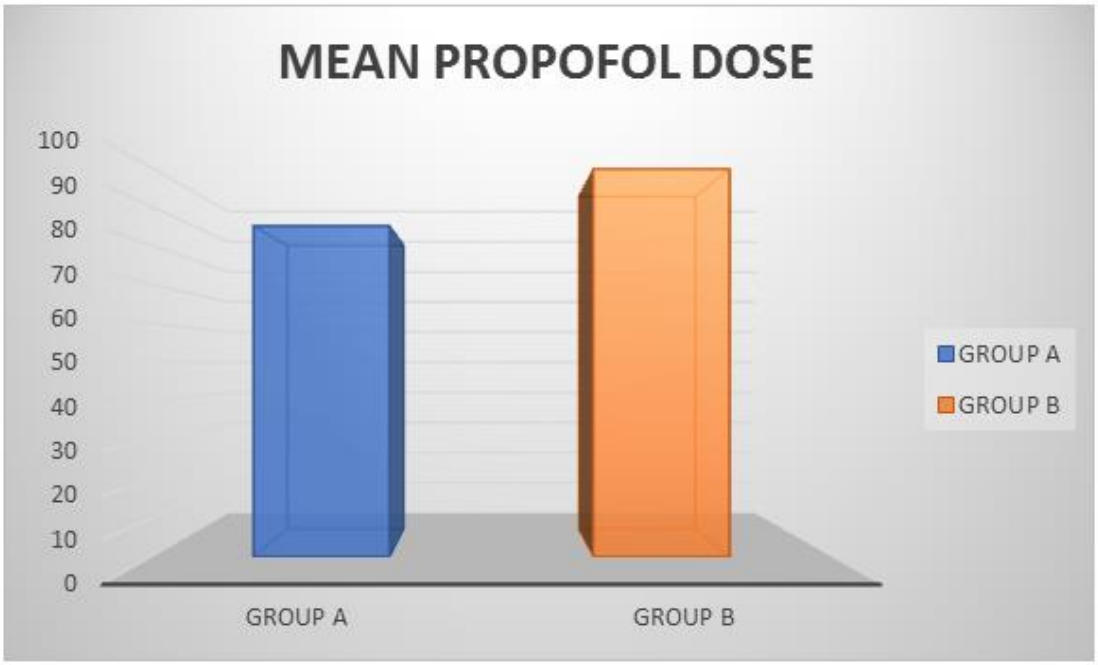

Figure 12: Comparison of propofol dose between groups

12 Complications

Complications like sore throat, post-operative nausea and vomiting and cough was recorded and represented in Table 13 and Figure 13.

Table 13: Complications

COMPLICATIONS	GROUP A	%	GROUP B	%
SORETHROAT	1	3.33	10	33.33

PONV	2	6.67	5	16.67
COUGH	0	0	1	3.33
P VALUE	0.279			

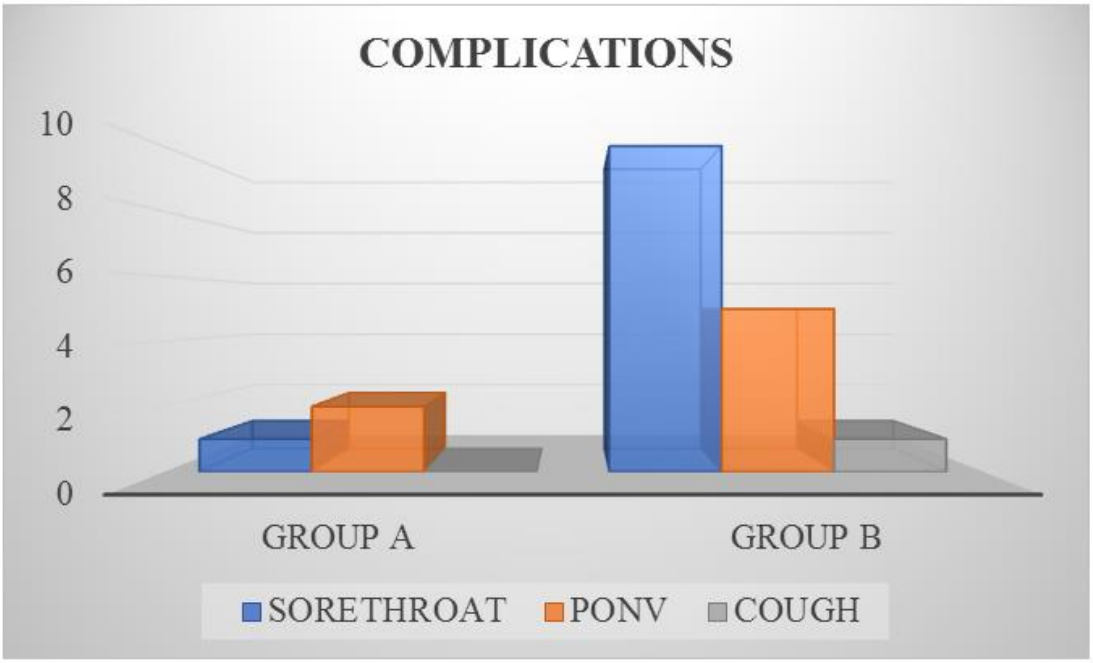

Figure 13: Complications

DISCUSSION

It is thought that general anaesthesia causes a regulated state of unconsciousness with analgesia and renders patients ignorant of what is happening during any surgical operation. It becomes necessary to give an appropriate airway with airway maintenance equipment, such as an endotracheal tube, because it is impossible for them to maintain it on their own in such situation. Endotracheal intubation and laryngoscopy have been shown to be the cornerstones for maintaining and defending the airway.

Intubation results in unwanted adverse outcomes even if it has been shown to be beneficial in maintaining a secured airway, avoiding aspiration, and delivering anaesthetic gases as intended. It is well recognised that laryngoscopy and endotracheal intubation can result in unwanted unpleasant sensations that generate unwarranted pressor reactions, such as elevated mean blood pressure and pulse rate, bronchospasm, laryngospasm, increased intracranial pressure, and intraocular pressure.

This unwanted hemodynamic response is believed to be caused by stimulating the mechanoreceptors found in the voice cords, epiglottis, and pharyngeal wall.

Reid and Brace were the first to describe the undesirable haemodynamic alterations brought on by laryngoscopy and intubation. It is observed that the hemodynamic reaction is triggered by direct laryngoscopy in a matter of seconds, and that this is followed by an ongoing stress response that occurs with the insertion of the endotracheal tube after it crosses the glottic aperture. After 5 seconds of direct laryngoscopy, the stress reaction starts, peaks in 1 to 2 minutes, and reduces to normal levels in another 5 minutes. Patients without cardiovascular or cerebrovascular disease are less able to withstand these stress-induced hemodynamic changes, which can result in life-threatening conditions like myocardial ischaemia, ventricular arrhythmias, cardiac failure, pulmonary oedema, and cerebrovascular accidents.

Myocardial ischemia can occur during direct laryngoscopy and during endotracheal intubation in individuals with coronary artery disease. There is also a larger likelihood that intraoperative ischemia will proceed to perioperative myocardial infarction. Attenuation of the direct laryngoscopy reaction is necessary to prevent tachycardia and the hypertensive response that results in strong sympathetic stimulation.

Many medication regimens and methods have been explored to obstruct the stress response, including lignocaine, opioids, nitroglycerine, calcium channel blockers like diltiazem, and β-blockers like esmolol. To lessen the pressor reaction to laryngoscopy and intubation, lignocaine preparations have been utilised as IV lignocaine as well as for topical anaesthesia in the form of lignocaine spray and nebulization.

In this study we compared the efficacy of nebulisedDexmeditomidine at a dose of 1mcg/kg and lignocaine 4mg/kg with the efficacy of nebulised lignocaine at a dose of 4mg/kg in attenuating the hemodynamic responses to laryngoscopy and intubation.

Most patients were observed in the age group of 18-60 years. In group A, the mean age distribution of the patients is 36±11.25 years were observed. In group B, the mean age distribution of the patients is 34.7±11.52 years were observed. In group A, 16 male patients and 14 female patients were observed. In group B, 14 male patients and 16 female patients were observed. In group A, the mean BMI value is 24.32±2.42 kg/m2 were observed. In group B, the mean BMI value is 18-60 kg/m2 were observed.

We discovered that there were no significant differences between any of the two groups' baseline and post-nebulization values for HR, SBP, DBP, and MAP. After intubation, the combination of nebulized Dexmedetomidine and nebulized Lidocaine resulted in superior HR control (P <.05) and this effect persisted for ten minutes. None of the patients, meanwhile, had bradycardia. Since it could be risky to conduct the preliminary study on ASA grade 3 and 4 patients before confirming its safety in healthy people, we have chosen ASA grade 1 and 2 patients. The greatest rise in heart rate following intubation was 19.6% lower in the Dexmedetomidine 0.5 mcg/kg group, according to K. Kumari and Colleagues' (2015) findings. Additionally, the drug was unable to totally block the hemodynamic response to laryngoscopy and intubation. Conversely, Zhan Guan *et al*., discovered that 1 mcg/kg dexmedetomidine markedly reduced the cardiovascular reactions associated with tracheal intubation; nevertheless, it also resulted in a notable drop in arterial pressure five minutes after intubation.

In sixty hypertensive patients, Moustafa A *et al*., evaluated the effectiveness dexmedetomidine (0.25 mg/kg)–lignocaine (1.0 mg/kg) in combination with each medication alone in reducing the hemodynamic and catecholamine responses during tracheal extubation. Following tracheal extubation, they discovered that patients receiving the combination of dexmedetomidine and lidocaine had significantly minimal changes in heart rates, mean arterial pressures, and rate-pressure products than patients getting either drug alone. In our study also efficacy of combined dexmedetomidine and lignocaine was higher than lignocaine alone.

According to Bruder*et al*.,'s previous proposal, lidocaine was helpful in preventing the pressor response to tracheal intubation, regardless of the delivery method (IV or intratracheal). This is supported by our study, which found that nebulized lidocaine was less effective than dexmedetomidine in suppressing HR.

When Patil*et al*., tested two dosages of nebulized lidocaine, they found that the intubation response was more effectively attenuated at 4% than at 2%. We have discovered that nebulized lidocaine, at a concentration of 4%, works well when administered on its own. However, when combined with dexmedetomidine, the impact was more coordinated and improved [16].

Nebulized lidocaine (120 mg) was observed by Sklar and colleagues to be more efficacious than IV (1 mg kg-1) in attenuating HDR to intubation for a duration of 10 minutes [18]. However, nebulized lidocaine (3 mg kg-1, 4%), as demonstrated by Kumar *et al*., was less successful in attenuating HDR to intubation than IV fentanyl (2 µg kg-1). The combo group's decreased MBP at 10 minutes, as reported by the authors, was not statistically significant [19]. The authors concur with Barton *et al*., that topical anaesthesia may not block sub mucosal deep proprioceptors, which are necessary for HDR attenuation for laryngoscopy. However, nebulized lidocaine does anaesthetize the trachea and lessen the reactions to intubation.

CONCLUSION

In comparison to lignocaine alone, the combination of nebulized dexmedetomidine and lignocaine effectively attenuates the pressor response during laryngoscopy and intubation without any hemodynamic fluctuations. This conclusion is based on the results of the current study and a correlation between the results of previous studies conducted by different authors.

Ethical Consideration

Institutional Ethical Committee approval, from ShriSathyaSai Medical College and Research Institute, Ammapettai was obtained before the start of the study (IEC NO 767 Informed written consent was obtained.

Confidentiality of the patient was maintained throughout the study.

Source of Funding: None Declared

Conflict of Interest: NIL

REFERENCE

1. Bruder, N., Ortega, D., &Granthil, C. (1992). Consequences and means of prevention hemodynamic changes in the laryngoscopy and endotracheal intubation haemodynamic reactions to laryngoscopy and intubation and their prevention. *Ann FrAnesthReanim*, 11, 57–71.
2. Sener, E. B., Ustun, E., Ustun, B., &Sarihasan, B. (2012). Hemodynamic responses and upper airway morbidity following tracheal intubation in patients with hypertension: Conventional laryngoscopy versus an intubating laryngeal mask airway. *Clinics*, 67, 49-54.
3. Fox, E. J., Sklar, G. S., Hill, C. H., Villanueva, R., & King, B. D. (1977). Complications related to the pressor response to endotracheal intubation. *Anesthesiology*, 47, 524–525.
4. Matot, I., Sichel, J. Y., Yofe, V., &Gozal, Y. (2000). The effect of clonidine premedication on hemodynamic responses to microlaryngoscopy and rigid bronchoscopy. *AnesthAnalg*, 91, 828–833.

5. Waikar, C., Singh, J., Gupta, D., &Agrawal, A. (2017). Comparative study of oral gabapentin, pregabalin, and clonidine as premedication for anxiolysis, sedation, and attenuation of pressor response to endotracheal intubation. *Anesth Essays Res*, 11, 558.
6. Srinivas, V. Y., Priya, K., &Kokila, N. (2019). Intravenous dexmedetomidine versus fentanyl to attenuate haemodynamic stress response to laryngoscopy and endotracheal intubation. *Indian J ClinAnaesth*, 6, 513-518.
7. Catterall, W. A., & Mackie, K. (2011.). Local Anesthetics. In: Brunton, L., Chabner, B., Knollman, B., editors. Goodman and Gilman's the Pharmacological Basis of Therapeutics. 12th ed. New York: McGraw Hill Publishers; pp. 564–582.
8. Bromage, P. R., & Robson, J. G. (1961). Concentrations of lignocaine in the blood after intravenous, intramuscular epidural and endotracheal administration. *Anaesthesia*, 16, 461-478.
9. Panzer, O., Moitra, V., & Robert, N. (2011). Pharmacology of sedative analgesics agents. Dexmedetomidine, remifentanil, ketamine: Volatile anesthetics and the role of peripheral Mu antagonists. *AnesthesiolClin*, 2, 587–605.
10. Gertler, R., Brown, H. C., Mitchell, D. H., &Silvius, E. N. (2001). Dexmedetomidine: A novel sedative-analgesic agent. *Proc (BaylUniv Med Cent)*, 14, 13-21.
11. Sriramka, B., Warsi, Z. H., &Sahoo, J. (2023). Effects of adding dexmedetomidine to nebulized lidocaine on control of hemodynamic responses to laryngoscopy and intubation: A randomized clinical trial. *J AnaesthesiolClinPharmacol*, *39*(1), 11-17.
12. Seangrung, R., Pasutharnchat, K., Injampa, S., Kumdang, S., &Komonhirun, R. (2021). Comparison of the hemodynamic response of dexmedetomidine versus additional intravenous lidocaine with propofol during tracheal intubation: a randomized controlled study. *BMC anesthesiology*, *21*, 1-11.
13. Selvaraj, V., &Manoharan, K. R. (2016). Prospective randomized study to compare between intravenous dexmedetomidine and esmolol for attenuation of hemodynamic response to endotracheal intubation. *Anesth Essays Res*, *10*(2), 343-348.
14. Mahjoubifard, M., Heidari, M., Dahmardeh, M., Mirtajani, S. B., &Jahangirifard, A. (2020). Comparison of Dexmedetomidine, Lidocaine, and Fentanyl in Attenuation Hemodynamic Response of Laryngoscopy and Intubation in Patients Undergoing Cardiac Surgery. *Anesthesiol Res Pract*, 2020, 4814037.
15. Kamali, A., Taghizadeh, M., Esfandiar, M., &Akhtari, A. S. (2018). A Comparison of the Effects of Dexmedetomidine and Propofol in Controlling the Hemodynamic Responses after Intubation: A Double-Blind, Randomized, Clinical Trial Study. *Open Access Maced J Med Sci*, *6*(11), 2045-2050.
16. Patil, V., Kulkarni, A., Rathod, C. V., &Sanjivani, C. R. (2013).Comparitive study between 2% and 4% lignocaine nebulisation on pressor response to laryngoscopy and intubation. *Executive Editor*, *4*(4), 277.
17. Mohsin, S., Ahmad Ganaie, Z., Kundi, H., Ahmed, M. B., Riaz, B., Khurshid Ahmed, N., AnirudhChunchu, V., &Haq, A. (2022). Comparison of Fentanyl and Dexmedetomidine in Preventing an Increase in Heart Rate during Intubation among Patients Undergoing General Anesthesia: A Meta-Analysis. *Cureus*, *14*(6), e26194.
18. Sklar, B. Z., Lurie, S., Ezri, T., Krichelli, D., Savir, I., &Soroker, D. (1992). Lidocaine inhalation attenuates the circulatory response to laryngoscopy and endotracheal intubation. *J ClinAnesth*, *4*(5), 382-385.
19. Kumar, A., Seth, A., Prakash, S., Deganwa, M., &Gogia, A. R. (2016), Attenuation of the hemodynamic response to laryngoscopy and tracheal intubation with fentanyl, lignocaine nebulization, and a combination of both: A randomized controlled trial. *Anesth Essays Res*, *10*(3), 661-666.