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Original Research Article

A Randomized Controlled Trial on Nebulized Dexmedetomidine vs. Normal Saline for Attenuating Hemodynamic Response to Intubation

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ABSTRACT

Background
Direct laryngoscopy and intubation act as unpleasant stimuli which are known to cause transient, but unpredictable hemodynamic changes. Our hypothesis was that nebulized dexmedetomidine, given as premedication blunts pressor response during laryngoscopy and intubation.

Methods
This was an open label, prospective, randomized controlled study which was carried out in 90 patients, male and female, aged 18 to 60 years, American Society of Anaesthesiologists (ASA) I and II, undergoing planned non-cardiac, non-neurosurgical surgery under general anesthesia with endotracheal intubation. Study was aimed at evaluating the effects of nebulized dexmedetomidine towards bringing down pressor response during laryngoscopy and intubation when compared with normal saline. The study population was split using pre-designed, computer-generated random allocation plan into two groups. Control group (n = 50), where patients were given nebulisation in sitting position, with 5 mL of 0.9% normal saline and study group (n = 50), where patients were administered nebulisation in the dose of 1 µg/kg dexmedetomidine (With 0.9% normal saline added to make a full volume of 5 mL) in sitting position, ten minutes before induction with anesthesia.

Results
The two groups were demographically similar. Following laryngoscopy and intubation in the control group, there were noticeable increase in heart rate (HR), systolic blood pressure (SBP), diastolic systolic blood pressure (DBP) and mean arterial pressure (MAP), but a drop in HR (at 1 min-72.11 ± 5.57; P= 0.001, 3 min - 68.62 ± 5.67; P< 0.001, 5 min-64.867 ± 5.34; P< 0.001, 10 min-60.867 ± 5.20; P< 0.001), SBP (at 1 min-116.53 ± 8.88; P< 0.001, 3 min-110.778 ± 9.2; P< 0.001, 5 min-105.289 ± 8.79; P< 0.001, 10 min-99.133 ± 7.87; P< 0.001), DBP (at 1 min-72.978 ± 6.10; P= 0.001, 3 min-68.73 ± 6.47; P< 0.001, 5 min-64.311 ± 6.07; P< 0.001, 10 min- 60.422 ± 5.79; P< 0.001) and MAP (at 1 min-87.5 ± 6.8; P= 0.001, 3 min-82.76 ±

6.76; *P*< 0.001, 5 min- 78.04 ± 6.26; *P*< 0.001, 10 min- 73.29 ± 5.57; *P*< 0.001) was recorded in the study group.

Conclusion

Nebulized dexmedetomidine when given as a premedication effectually blunts the pressor response during laryngoscopy and intubation.

Key words: Direct laryngoscopy, intubation, hemodynamic, dexmedetomidine, premedication

Key message

a. Tracheal intubation causes unpredictable and variable hemodynamic changes.
b. Nebulized dexmedetomidine helps to blunt the pressor response during laryngoscopy and intubation.

INTRODUCTION

Direct laryngoscopy and intubation are unpleasant stimuli, which are linked to transient, erratic and variable hemodynamic changes in the patient. In susceptible individuals, these changes may lead to development of cardiac arrhythmias, ischemia, increased intracranial pressure, cerebrovascular stroke, and pulmonary edema. Various drugs - β-blockers, calcium channel blockers, vasodilators, opioids, intravenous lidocaine, topical creams, volatile compounds, and α -2 agonists have been tried to date to reduce the effect of this pressor response, but none have been proven to be ideal [1,2]. Dexmedetomidine is a newer selective α-2 adrenergic receptor agonist in action possessing anti-sympathetic, anxiolytic, analgesic, anti-secretory, sedative, and hypnotic properties. It also helps maintain the stability of the nervous, cardiac and respiratory systems. The efficacy of dexmedetomidine in reducing the pressor response during laryngoscopy and intubation has been investigated by intravenous[3], intranasal [4] and nebulized [5,6] routes. Intranasal administration may be associated with transient nasal congestion, coughing, and vocal cord irritation [4], while intravenous administration may cause bradycardia and hypotension [3]. In addition, the bioavailability of nebulized dexmedetomidine in the nasal mucosa is 65% and in the buccal mucosa is 82% [7]. In younger children, nebulized dexmedetomidine has been shown to be an effective premedication at doses of 1 µg/kg and 2 µg/kg [8]. It may replace the intravenous and intranasal administration method due to the accumulation of the drug in the nose, mouth and respiratory mucosa by aerosolization. Our study tested the hypothesis that nebulized dexmedetomidine as a premedication blunts the pressor response during laryngoscopy and intubation in patients undergoing elective surgery.

MATERIAL & METHODS

This open label, prospective, randomized controlled study has Institutional Ethics Committee approval vide Certificate number – 1562 dated 05/01/2022. This study was conducted at Department of Anaesthesiology at a Tertiary care Hospital in Central India. The study was conducted in accordance with Good Clinical Practice and the Helsinki Declaration of Human Rights (1975, revised in 2013). The most important factors considered were the well-being and safety of patients. The research data is kept confidential, and all concerns and fears of the patients and their families were taken into consideration.

Patient selection

Ninety patients, male and female, aged 18 to 60 years, American Society of Anaesthesiologists (ASA) classes I and II, undergoing planned non-cardiac, non-neurosurgical surgery under general anaesthesia with endotracheal intubation were chosen.

Inclusion criteria

i. American Society of Anaesthesiologists (ASA) Physical status classification system - I and

II
ii. Male or female between ages 18 to 60 years
iii. Undergoing non-cardiac, non-neurosurgical elective surgery under general anaesthesia with endotracheal intubation

Exclusion criteria

i. Patients refusing consent for the study
ii. Patients having predicted difficult airway
iii. Patients requiring more than 2 attempts or 15 seconds for laryngoscopy
iv. Patients with pregnancy
v. Patients with systemic illnesses like uncontrolled hypertension, renal failure or seizure disorders
vi. Patients taking anti-depressants/anti-psychotics/anti-hypertensive medications
vii. Patients having low cardiopulmonary reserve
viii. Patients having body mass index (BMI) above 30 kg/m^2

A visit to the ward was made the day before the planned surgery. After obtaining history, performing general physical and airway examination, and reviewing routine blood reports viz. Complete blood count, liver and renal function tests, serum electrolytes, ECG, and chest X-ray, the patients were selected for this study after applying the strict exclusion criteria. The selected patients were informed about the study policy and consented to participate. They were kept nil by mouth for eight hours before induction with anaesthesia and were given Tab. Pantoprazole 40 mg at night.

Statistical Analysis

With reference to the Kumar NR and others [9] study, sample size was determined considering the pre-post difference in mean SBP as the main outcome measure with Pre-test mean (SBP) of 126.64 (Standard deviation in Pre-test = 26.37), Post-test mean (SBP) of 109.50 (Standard deviation in Post-test = 16.83), effect size of 17.14, alpha error of 1 and power of 99%. Therefore, 90 patients (45 in each group) were selected for this study with no dropouts.

The selected sample of subjects (n=90) were divided randomly using pre-designed, computer-generated random allocation plan into two groups – Group D (Nebulised dexmedetomidine given) and Group C (Nebulised dexmedetomidine not given). Fifteen blocks of size 6 were assigned to the treatment groups (n=45 per group), the use of permutation blocks of size 6 made prediction difficult. The data was examined using the International Business Machine Statistical Package for Social Sciences (IBM SPSS) 21.0 version. Shapiro Wilk test was employed to determine if all variables were adhering to a normal distribution. The data was found to follow a normal distribution, so we conducted bivariate analyses using parametric tests like the independent t-test for comparing two groups, and repeated measures of analysis of variance (ANOVA) to compare the mean values across different time intervals. Chi square test was employed for variables that fell into categorical categories. A p-value less than 0.05 was deemed statistically significant for all the comparisons. The anaesthesiologist giving nebulization to the patients, laryngoscopist and the anaesthesiologist recording observations were non-participating in the study, hence minimising bias.

Anaesthesia management

Patients were taken to the operation theatre on the day of the surgery after they had confirmed their nil by mouth status and given written consent. Multipara monitor - non-invasive monitoring of blood pressure, ECG, oxygen saturation was attached, and baseline parameters

were recorded by a non-participating anaesthesiologist. Intravenous access with 18G/20G cannula was secured. In sitting position, ten minutes prior to induction, nebulization was administered by a non-participating anaesthesiologist, using an electrical piston compressor type nebulizer with a nebulizer face mask, with either dexmedetomidine in the dose of 1 µg/kg (With 0.9% normal saline added to make a full volume of 5 mL) or 5 mL of 0.9% normal saline. Nebulization ceased when no mist was seen on gently striking the chamber of nebulizer. The total volume was given in a time of 15-20 minutes.

Then, the patients were made supine and pre-oxygenated for three minutes with a face mask on spontaneous ventilation using oxygen at 100% FiO2. Anaesthesia was standardized for all the patients. Premedication was given with injection Midazolam IV in the dose of 30 µg/kg, injection Glycopyrrolate IV in the dose of 4 µg/kg and injection Fentanyl IV in the dose of 2 µg/kg. Injection Propofol IV titrated to the loss of verbal response was given for induction. Injection Vecuronium IV given in the dose of 100 µg/kg, as a neuromuscular blocking agent to ease the intubation. Direct laryngoscopy using an appropriately sized Macintosh blade and intubation were done with a cuffed endotracheal tube of suitable size by non-participating anaesthesiologist having at least 2 years of experience of administering anaesthesia in the department. Then, the patients were put on volume control mode of ventilator. Till ten minutes following intubation, patients were left untouched. Important parameters like heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP) were noted by a non-participating anaesthesiologist, at the following time points: baseline, instantly following nebulization and at one-, three-, five- and ten-minute intervals after intubation. The study concluded at this point.

For analgesia intra-operatively, all the patients received injection Paracetamol IV in the dose of 20 mg/kg and injection Diclofenac IV in the dose of 1.5 mg/kg. After the completion of surgical procedure, patients were reversed with injection Glycopyrrolate IV in the dose of 8 µg/kg and injection Neostigmine IV in the dose of 50 µg/kg. After gentle oropharyngeal suctioning and once the patients could breathe spontaneously and adequately, open their eyes on command or perform facial grimace, any purposeful movement or attempt to self extubation, extubation was done. Patients were kept in post-anaesthesia care unit after this.

RESULTS:

A study was conducted on the consort chart of 90 patients who underwent surgery under general anaesthesia, as shown [Figure 1]. The data was gathered, organized, examined, and the following observations were recorded. The demographic information was similar between the two groups [Tables 1 and 2].

Change in HR, SBP, DBP, and MAP values following dexmedetomidine nebulisation were measured as an indicator of the pressor response during laryngoscopy and intubation. The main outcome was to measure changes in HR, while measuring changes in SBP, DBP and MAP were secondary outcomes.

The mean HR was comparable in the two groups ($P > 0.05$) at the baseline [Table 3] and immediately after nebulization [Table 4]. However, after intubation, HR was discovered to be substantially higher in the control group than in the study group with P-values of 0.001 at one minute [Table 5], <0.001 at three minutes [Table 6], <0.001 at five minutes [Table 7] and <0.001 at ten minutes [Table 8].

In the two groups, mean SBP was comparable at baseline ($P = 0.337$) and immediately post nebulization ($P = 0.195$) [Tables 3 and 4]. At all time intervals (one-, three-, five-, and ten-minutes), the study group's increase in SBP following intubation was, however, significantly less than that of the control group ($P < 0.001$ at one-, three-, five-, and ten-minutes) [Tables 5, 6, 7 and 8].

The mean DBP at baseline was found to be significantly more (P value of 0.016) in the study group [Table 3]. Post nebulization, the mean DBP in the two groups were comparable (P > 0.05) [Table 4]. Yet, mean DBP following intubation showed a significant difference between the study group and the control group, with P values of less than 0.001 at one minute [Table 5], less than 0.001 at three minutes [Table 6], less than 0.001 at five minutes [Table 7], and less than 0.001 at ten minutes [Table 8].

The MAP measured in the two groups was found to be comparable at the baseline [Table 3] and immediately after nebulization (P>0.05) [Table 4]. Following intubation, the control group's MAP values were consistently higher than those of the study group in this investigation, with P values of 0.001 at one minute [Table 5], <0.001 at three minutes [Table 6], <0.001 at five minutes [Table 7], and <0.001 at ten minutes [Table 8].

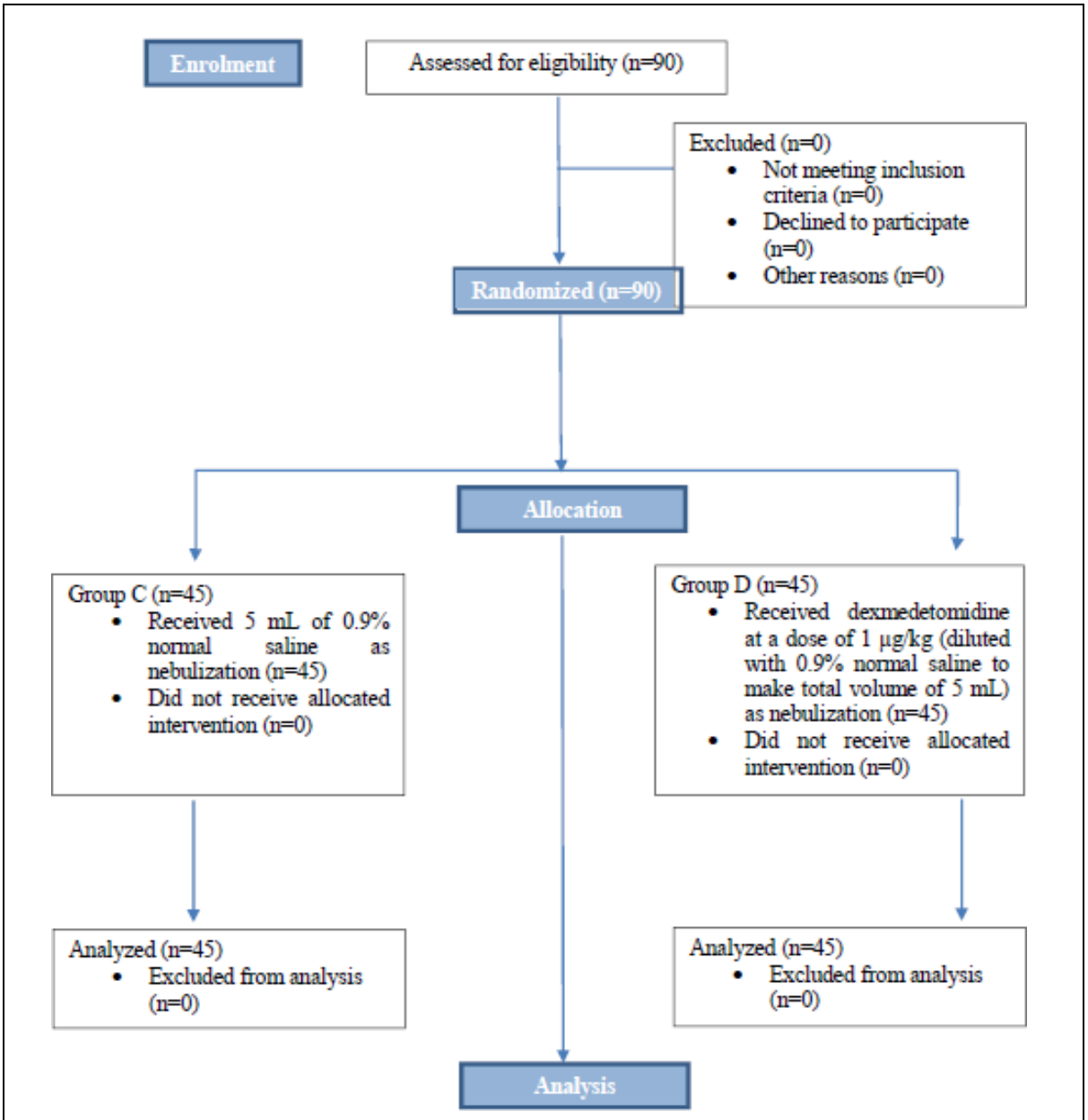

Figure 1: Consort flow chart for selection, randomization, allocation, and analysis of study subjects

	Group	n	Mean	Standard Deviation	Standard Error Mean
Age	Study	45	42.733	11.3726	1.6953
	Control	45	44.778	11.7045	1.7448
P value	0.403				

Table 1: Age wise (in years) distribution of subjects in study and control groups

			Sex		Total
			F	M	
Group	Control	n	22	23	45
		%	48.9%	51.1%	100.0%
	Study	n	23	22	45
		%	51.1%	48.9%	100.0%
Total		n	45	45	90
		%	50.0%	50.0%	100.0%
P value		0.833			

Table 2: Gender wise (male or female) distribution of subjects in study and control groups

Baseline	Group	n	Mean	Standard Deviation	Standard Error Mean	P Value
HR	Study	45	77.133	5.4087	.8063	0.271
	Control	45	75.911	5.0624	.7547	
SBP	Study	45	122.733	9.0539	1.3497	0.337
	Control	45	120.733	10.5300	1.5697	
DBP	Study	45	78.089	6.3812	.9512	0.016*
	Control	45	74.644	6.8661	1.0235	
MAP	Study	45	92.689	7.1249	1.0621	0.109
	Control	45	90.111	7.9722	1.1884	

Table 3: Comparison of mean Heart rate (HR in beats per minute), mean Systolic Blood Pressure (SBP in millimetre of mercury), mean Diastolic Blood Pressure (DBP in millimetre of mercury) and Mean Arterial Pressure (MAP in millimetre of mercury) at baseline in study and control groups, where n is sample size

	Group	n	Mean	Standard Deviation	Standard Error Mean	P Value
HR	Study	45	75.267	5.8286	.8689	0.132
	Control	45	77.000	4.9360	.7358	
SBP	Study	45	121.222	9.0927	1.3555	0.195
	Control	45	123.933	10.5667	1.5752	
DBP	Study	45	76.556	6.3552	.9474	0.479
	Control	45	77.444	5.4712	.8156	
MAP	Study	45	91.356	6.6952	.9981	0.224
	Control	45	93.133	7.0794	1.0553	

Table 4: Comparison of mean Heart rate (HR in beats per minute), mean Systolic Blood Pressure (SBP in millimetre of mercury), mean Diastolic Blood Pressure (DBP in millimetre of mercury) and Mean Arterial Pressure (MAP in millimetre of mercury) post- nebulization in study and control groups, where n is sample size.

	Group	n	Mean	Standard Deviation	Standard Error Mean	P Value
HR	Study	45	72.111	5.5768	.8313	0.001
	Control	45	87.533	7.8265	1.1667	
SBP	Study	45	116.533	8.8769	1.3233	<0.001
	Control	45	134.511	9.2874	1.3845	
DBP	Study	45	72.978	6.1032	.9098	0.001
	Control	45	85.222	5.9882	.8927	
MAP	Study	45	87.556	6.6999	.9988	0.001
	Control	45	101.556	6.6897	.9972	

Table 5: Comparison of mean Heart rate (HR in beats per minute), mean Systolic Blood Pressure (SBP in millimetre of mercury), mean Diastolic Blood Pressure (DBP in millimetre of mercury) and Mean Arterial Pressure (MAP in millimetre of mercury) post-intubation at one minute interval, in study and control groups, where n is sample size

	Group	n	Mean	Standard Deviation	Standard Error Mean	P Value
HR	Study	45	68.622	5.6741	.8458	<0.001*
	Control	45	83.444	7.5062	1.1190	
SBP	Study	45	110.778	9.1995	1.3714	<0.001*
	Control	45	130.444	9.0643	1.3512	
DBP	Study	45	68.733	6.4716	.9647	<0.001*
	Control	45	81.356	5.5027	.8203	
MAP	Study	45	82.756	6.7560	1.0071	<0.001*
	Control	45	97.689	6.2444	.9309	

Table 6: Comparison of mean Heart rate (HR in beats per minute), mean Systolic Blood Pressure (SBP in millimetre of mercury), mean Diastolic Blood Pressure (DBP in millimetre of mercury) and Mean Arterial Pressure (MAP in millimetre of mercury) post-intubation at three minutes interval, in study and control groups, where n is sample size

	Group	n	Mean	Standard Deviation	Standard Error Mean	P value
HR	Study	45	64.867	5.3453	.7968	<0.001*
	Control	45	79.622	7.3246	1.0919	
SBP	Study	45	105.289	8.7895	1.3103	<0.001*
	Control	45	126.133	9.2309	1.3761	
DBP	Study	45	64.311	6.0784	.9061	<0.001*
	Control	45	77.622	5.7733	.8606	
MAP	Study	45	78.044	6.2594	.9331	<0.001*
	Control	45	93.733	6.6243	.9875	

Table 7: Comparison of mean Heart rate (HR in beats per minute), mean Systolic Blood Pressure (SBP in millimetre of mercury), mean Diastolic Blood Pressure (DBP in millimetre of mercury) and Mean Arterial Pressure (MAP in millimetre of mercury) post-intubation at five minutes interval, in study and control groups, where n is sample size

	Group	n	Mean	Standard Deviation	Standard Error Mean	P value
HR	Study	45	60.867	5.2075	.7763	<0.001*
	Control	45	74.956	7.3669	1.0982	
SBP	Study	45	99.133	7.8700	1.1732	<0.001*
	Control	45	119.978	8.9987	1.3414	
DBP	Study	45	60.422	5.7898	.8631	<0.001*
	Control	45	72.889	5.7770	.8612	
MAP	Study	45	73.289	5.5744	.8310	<0.001*
	Control	45	88.533	6.5387	.9747	

Table 8: Comparison of mean Heart rate (HR in beats per minute), mean Systolic Blood Pressure (SBP in millimetre of mercury), mean Diastolic Blood Pressure (DBP in millimetre of mercury) and Mean Arterial Pressure (MAP in millimetre of mercury) post-intubation at ten minutes interval, in study and control groups, where n is sample size

DISCUSSION

Laryngoscopy and endotracheal intubation form the key components of anaesthesia management in general anaesthesia and critical care. During laryngoscopy, the insertion of an endotracheal tube into the trachea stimulates sympathoadrenal receptors, releasing catecholamines into the bloodstream that momentarily cause a pressor reaction, which is seen as a surge in heart rate, systolic and diastolic blood pressures. The reaction to tracheal intubation and laryngoscopy is a somatovisceral kind of reflex[10]. These reflex responses may be blunted or modified locally or centrally. Several attempts have been made to accomplish this using different techniques and agents, but with limited success[1,2,10]. So far, no drug has been found to completely blunt this response.

In this study, the nebulization route was chosen for drug delivery as this route is more convenient, painless, odourless, tasteless and does not need any intravenous infusion. The bioavailability of nebulized Dexmedetomidine in the nasal mucosa is 65% and in the buccal mucosa is 82%[7]. Due to this, it rapidly comes into the systemic circulation and bypasses the liver's first-pass metabolism. When given as IV bolus, dexmedetomidine may precipitate sudden bradycardia and hypotension, which are avoided when the drug is given by nebulization route.

Various authors have studied the role of blunting of pressor response during laryngoscopy and intubation using dexmedetomidine via several routes of administration and with varying drug dosages[8,9,10-19]. It has been given in the dose of 0.75 µg/kg[14], 0.6 µg/kg[15] and 0.5 µg/kg[16]. However, to the best of our knowledge, no recommendation regarding dosage via nebulization route was found. But, Kumar NR and others[9], Misra S and others[11], and Shrivastava P and others[12], all administered dexmedetomidine via nebulization route in the dose of 1 µg/kg. Therefore, in this study, nebulization of dexmedetomidine was given in the dose of 1 µg/kg.

The effects of intravenous dexmedetomidine start to show in 15 minutes. In healthy adults, its distribution half-life is six minutes, and its elimination half-life is roughly two to three hours[7]. The pressor response during laryngoscopy and intubation begins within 5 sec of laryngoscopy, attains peak in 1–2 min and comes back to normal levels by 5 min. Taking both of these into consideration, the timing for administering nebulized dexmedetomidine in our

study was chosen as 10 min prior to induction with anaesthesia. This was similar to the timing as chosen in the study by Kumar NR and others[9]. Whereas, Misra S and others[11] and Shrivastava P and others[12] administered nebulized dexmedetomidine 30 min prior to induction.

The study found no significant difference (P = 0.403) in the mean age of the control group, which was 44.78 ± 11.70 years, and the study group, which had a mean age of 42.73 ± 11.37 years. The mean age of the included subjects in a related study by Shrivastava P and others [12] was 38 years for the study group and 39 years for the control group. The mean age of the study group was 37 years, while the control group's age was 40 years in two studies (Kumar NR and others [9] and Misra S and others [11]). [Table 1]

Also, 48.9% control population was female, while 51.1% were males. Females formed 51.1% of the study population, while males formed 48.9%. Therefore, P = 0.83 indicates that there was no significant difference in the subjects' distribution in either group. In a related study conducted by Kumar NR and others[9], there were 52% men and 48% women in the control group. In contrast, 58% of the study group's participants were female, and 42% were male. [Table 2]

In this study, mean HR for the two groups at the baseline and immediately after nebulization (P>0.05) were comparable. However, after intubation, the control group's HR was found to be significantly higher than the study group's mean HR of 87.5 ±7.83 bpm vs. 72.11 ± 5.57 bpm at 1 min (P=0.01), 83.4 ± 7.50 bpm vs. 68.62 ± 5.67 bpm at 3 min (P<0.001), 79.6 ± 7.32 bpm vs. 64.867 ± 5.34 bpm at 5 min (P<0.001) and 74.96 ± 7.37 bpm vs. 60.867 ± 5.20 bpm at 10 min (P<0.001) respectively. When HR was compared within the control group, it was found that there were significant differences from baseline to, post nebulization to post intubation at, three-, five- and ten-minutes, but the difference in mean HR at one minute after intubation was not significant. However, intragroup comparison of HR in study group showed overall significant fall in mean HR from baseline to post intubation at all intervals. In a related study by Misra S and others[11], where dexmedetomidine and saline were compared, post-laryngoscopy, and intubation, It was discovered that the dexmedetomidine group's HR rise was significantly lower than the saline group's (P=0.012). An identical study by Shrivastava P and others[12] found a significant reduction in HR at 1 minute, 5 minutes, and 10 minutes after intubation in the group of patients who received dexmedetomidine nebulisation (P-values of 0.001, 0.003, and 0.013, respectively). Our study's conclusions align with the findings of these two studies. [Tables 3-8]

In the current study, there was no significant difference in the mean SBP between the study and control groups at baseline (P = 0.337) or right after nebulisation (P = 0.195). However, with regards to increase in SBP after intubation, the values in study group were much lower than control group with mean SBP values at 116.53 ± 8.88 mm Hg vs. 134.51 ± 9.29 mm Hg at 1 min after intubation (P < 0.001), 110.778 ± 9.2 mm Hg vs. 130.44 ± 9.06 mm Hg at 3 min after intubation (P < 0.001), 105.289 ± 8.79 mm Hg vs. 126.133 ± 9.23 mm Hg at 5 min after intubation (P < 0.001) and at 99.133 ± 7.87 mm Hg vs. 119.978 ± 8.99 mm Hg at 10 min after intubation (P < 0.001). Kumar NR and others[9], discovered that both groups' mean SBPs were comparable right after intubation and after nebulisation. However, the study group's SBP readings at one-, five-, and ten-minutes following intubation were significantly lower, with P values of 0.01, 0.02, and 0.03 respectively. P-values obtained before laryngoscopy, after intubation, one minute after intubation, five minutes after intubation, and ten minutes after intubation were 0.019, 0.007, 0.001, 0.001, and 0.010, respectively, according to a related study by Shrivastava P and others [12]. There was also a significant statistical difference between the mean SBP of the study and control groups. Present study has shown results which are like these two studies.

In contrast to this study, Misra S and others[11] who compared the SBP response between saline and nebulised dexmedetomidine found no significant difference between the two groups. This might be due to the 30 minutes time difference between administration of nebulized dexmedetomidine and laryngoscopy and intubation as opposed to ten minutes in our study.

The mean DBP was found to be significantly higher (P=0.016) in the study group, even though there was no significant difference in the mean SBP, MAP, or HR between the two groups at baseline in this study. The two groups' mean DBPs were similar after nebulisation. At one minute, three minutes, five minutes, and ten minutes after intubation, the study group's mean blood pressure was 72.978 ± 6.10 mm Hg compared to 85.22 ± 5.98 mm Hg with P=0.001, 68.73 ± 6.47 mm Hg compared to 81.356 ± 5.50 mm Hg with P<0.001, 64.311 ± 6.07 mm Hg compared to 77.62 ± 5.77 mm Hg with P<0.001, and 60.422 ± 5.79 mm Hg compared to 72.889 ± 5.77 mm Hg with P<0.001, according to those values.

In a related study by Kumar NR and others[9], DBP readings amongst the two groups were comparable after nebulization and right away upon intubation. With P values of 0.001, 0.001, and 0.01 respectively, the study group's DBP values were lower 1, 5, and 10 min after laryngoscopy and intubation. In a similar study, Shrivastava P and others[12] compared the mean DBP of the two research groups at various intervals. The two groups' baseline DBPs were not significantly different, but the mean DBP changed significantly (P=0.011 at one minute after intubation, P=0.005 at three minutes, and P=0.009 at ten minutes).

There was no discernible difference in the MAP between the study and control groups at baseline in the current study (P>0.05). Likewise, following nebulisation, there was no discernible variation between the study and control groups' mean MAP (P=0.132). Following intubation, the control group's MAP values were found to be significantly higher than those of the study group in this study. In the study group, the average mean arterial pressure (MAP) values varied from 87.5 ± 6.8 mm Hg to 101.556 ± 6.69 mm Hg at one minute after intubation (P=0.001); 82.76 ± 6.76 mm Hg to 97.689 ± 6.24 mm Hg at three minutes after intubation (P<0.001); 78.04 ± 6.26 mm Hg to 93.733 ± 6.62 mm Hg at five minutes after intubation (P<0.001); and 73.29 ± 5.57 mm Hg to 88.533 ± 6.54 mm Hg at ten minutes after intubation (P<0.001). Kumar NR and others[9] in a related study, found that the two groups' mean MAP values following nebulisation and right after intubation were similar. With P values of 0.001 at 1 minute, 0.003 at 5 minutes, and 0.008 at 10 minutes, it was discovered that the study group's post-intubation MAP values were significantly lower. A comparison of MAP between the two research groups at different times was conducted by Shrivastava P and others[12]. The two groups were not significantly different at baseline, but the MAP varied greatly before laryngoscopy, after intubation, after the first minute, after five minutes, and after ten minutes, with P values of less than 0.047, 0.042, 0.001, 0.006, and 0.018, respectively. Present study has shown results which are similar to these two studies.

CONCLUSION

Numerous stress reactions, including tachycardia, hypertension, laryngospasm, bronchospasm, elevated intracranial pressure, and intraocular pressure, can be brought on by laryngoscopy and endotracheal intubation. The hemodynamic response is initiated 5 sec of laryngoscopy, attains peak in 1–2 min and comes back to normal levels by 5 min. There is a need for appropriate techniques and interventions to minimize this. According to the results of our study, nebulised dexmedetomidine administered as a premedication to patients undergoing elective surgery appears to blunt the pressor response during laryngoscopy and intubation.

LIMITATIONS

It would have been better to confirm the effects of nebulised dexmedetomidine in this patient population with a larger patient group, as the study was single centred and had a small sample size. Small sample size constraints might be addressed by combining data from several centres. Since the study only includes a portion of the referred patients—as befits a tertiary referral centre—there may be some referral bias, and the findings may not apply to other settings. However, we think that this study's noteworthy findings contribute to the body of expanding knowledge for upcoming research and may have clinical implications and warrant additional study to address hemodynamic changes during intubation.

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