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Original Research Article

Attenuating Haemodynamic Response to Laryngoscopy and Intubation

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Abstract:

Background: Laryngoscopy and endotracheal intubation for airway management produces intense noxious stress response appearing as an alteration in systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate which could be life threatening in selected group of patients. Dexmedetomidine is a selective alpha 2-adrenoceptor agonist having sedative, hypnotic, anxiolytic, analgesic, sympatholytic properties. Nebulised dexmedetomidine has good bioavailability that is why nebulisation route was chosen.

Aim and Objectives: Purpose of the study is to evaluate the effect of nebulised dexmedetomidine in attenuating haemodynamic response to laryngoscopy and endotracheal intubation.

Materials and Methods: An observational, prospective clinical study was carried out on 130 patients aged between 18-60 years of both sex of ASA grade I and II, undergoing elective surgeries under general anaesthesia. Patients were divided into two groups containing 65 patients in each group to facilitate intubation, Group D (n=65)- received Dexmedetomidine in dose of 1 mcg/kg diluted up to 5 ml with normal saline, Group N (n=65)- received normal saline via nebulisation route.

Result: Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure were comparable in both groups at baseline with no statistically significant difference ($p>0.05$). There was no statistically significant difference found between the groups with respect to heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure after nebulisation and just before intubation ($p>0.05$). After intubation rise in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure were statistically significant in normal saline group when compared to dexmedetomidine group ($p<0.05$). At 1, 5 min post intubation there was a statistically significant difference ($p>0.05$) in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure between the two groups. While 10 mins post intubation difference in heart rate was statistically significant between the two groups but the difference in systolic blood pressure, diastolic blood pressure, mean arterial pressure were not statistically significant between the two groups ($p>0.05$).

Conclusion: Nebulized dexmedetomidine demonstrated its effectiveness in attenuating the intense hemodynamic response induced by laryngoscopy and endotracheal intubation in patients undergoing elective surgeries under general anesthesia. This study highlights its potential as a valuable adjunct in airway management strategies to enhance patient safety and minimize perioperative cardiovascular stress responses.

Keywords: Dexmedetomidine, Nebulization, Hemodynamic Response, Airway Management.

Introduction

Laryngoscopy and endotracheal intubation are indispensable part of general anaesthesia as it provides a definitive airway for the delivery of anaesthetic gases , protects airway from aspiration of gastric contents and useful for positive pressure ventilation with higher airway pressures.

Laryngoscopy and endotracheal intubation are considered as intense noxious stimuli which produces transient stress response appearing as an alteration in systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate in most of the individuals [1].

These responses were first recognized in 1951 by king et al [2] and subsequently in various studies. These occur due to sympathetic responses accompanied by increase in plasma concentration of catecholamines occurring within 30 seconds of intubation which further peaks in 1-2 min and may

last up to 10 min [3]. These responses also depends upon duration and force applied for laryngoscopy and endotracheal intubation.

In normal individuals these sympathetic responses may not bring any serious consequences but in patients having comorbid illness like systemic hypertension, cardiovascular problems, cerebrovascular disease (CVA), intracranial pathologies and hyperactive airways, these hemodynamic changes may lead to life-threatening risk and precipitate hypertensive crisis, heart ischemia, arrhythmia, acute heart failure, myocardial infarction and cerebrovascular events [4]. Hence in such circumstances these reflex circulatory responses to laryngoscopy and endotracheal intubation should be suppressed.

Numerous drugs have been tried till date by various routes to overcome these sympathetic responses caused by laryngoscopy and endotracheal intubation such as opioids , beta blockers ,calcium channel blockers ,intravenous lignocaine ,topical sprays , but none of them are proved to be much effective [5].

In this study, we have used dexmedetomidine which is a selective alpha 2- adrenoceptor agonist and is 8 times more specific for alpha 2 receptors than clonidine. Dexmedetomidine is a short acting drug having sedative, hypnotic, anxiolytic, analgesic, sympatholytic properties [6]. It has been observed to produce bradycardia and hypotension when given as intravenous bolus [9], to evade this problem nebulisation route was chosen. Nebulised dexmedetomidine has a bioavailability of 65% through the nasal mucosa and 82% through the buccal mucosa [7]. Nebulized dexmedetomidine may be considered as better alternative to both intravenous and intranasal routes of administration because after nebulization deposition of drug takes place over nasal, buccal, as well as respiratory mucosa [8]. Moreover, intranasal route may cause transient nasal irritation [8], cough, vocal cord irritation or laryngospasm. To avoid these problems nebulised route was favoured over intranasal route. In this study, we hypothesised that nebulised dexmedetomidine may be effective in blunting the haemodynamic response to laryngoscopy and endotracheal intubation due to its rapid absorption and good bioavailability. Hence, this study was contemplated, in an attempt to investigate, its role in attenuating the stress response to laryngoscopy and intubation.

Materials and methods

The present observational, hospital-based study was conducted after obtaining approval from institutional ethical committee. As per the discretion of anaesthesiologist, the anaesthetic procedure, and the patient profile, 130 patients aged between 18-60 years of either sex of ASA grade I and II, undergoing elective surgeries under general anaesthesia were chosen and divided into two groups containing 65 patients in each group to facilitate laryngoscopy and endotracheal intubation: Group D (n=65)- received Dexmedetomidine in dose of 1 mcg/kg diluted upto 5 ml with normal saline , Group N (n=65)- received normal saline via nebulisation.

Every patient was subjected to complete general physical and systemic examination and detailed history was taken. Basic demographic characteristics such as age, sex, weight and BMI were noted.

Modified mallampati score was evaluated with the patient in the sitting position and the neck held in neutral position and the tongue fully protruded without phonation.

In the operation theatre, intravenous line, pulse oximeter, electrocardiograph and a noninvasive blood pressure monitor were attached and baseline values of hemodynamic parameters such as heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, SpO2 were recorded.

10 mins prior to induction half of the patients were nebulised with 5 ml of normal saline (group N) while other half of the patients were nebulised with dexmedetomidine 1mcg/kg diluted up to 5 ml of volume (Group D).

After nebulization baseline vitals were again recorded and after 10 minutes and Ramsay sedation score was also observed. Patients were premedicated with Inj. ondansetron 0.08 mg/kg, Inj. glycopyrrolate 0.01mg/kg, Inj. midazolam 0.05mg/kg, Inj. Fentanyl 1.5mg/kg.

Preoxygenation with 100% oxygen was done for 3 minutes and general anaesthesia was induced with Inj. Propofol 2mg/kg and Inj. succinylcholine 2mg/kg was used for facilitation of intubation and muscle relaxation. Mask ventilation was done for 1 minute after injection of succinylcholine. Direct laryngoscopy (with appropriate macintosh blade) and endotracheal intubation was done, IPPV was started.

All the vital parameters were again recorded post intubation at 1 ,5 ,10 mins post intubation and our study will end here.

Patients were maintained on 50% nitrous oxide and 50% oxygen and Isoflurane 0.2% - 1%. Atracurium at the dose of 0.1mg/kg body weight was used for maintenance of muscle paralysis. The concentration of isoflurane was increased or decreased during surgery to maintain BP and HR between 80% and 120% of the preoperative values.

Residual neuromuscular block was antagonized with neostigmine 0.05 mg/kg and glycopyrrolate 0.01

mg/kg following which oropharyngeal secretions were aspirated before extubation and the endotracheal tube was removed smoothly.

Ramsay sedation scale

Score	Response
1	Patient anxious and agitated or restless or both
2	Patient cooperative, oriented, and tranquil
3	Patient drowsy but responds to commands
4	Brisk response to light glabellar tap or loud auditory stimulus
5	Sluggish response to light glabellar tap/loud auditory stimulus
6	No response to light glabellar tap or loud auditory stimulus

Statistical analysis

The data was entered in Microsoft excel 2019 (part of Microsoft office professional edition) [computer program]. Microsoft; 2019) and analysed using, MedCalc v18.2.1 (MedCalc statistical software version 18.2.1 (MedCalc software, Ostend, Belgium; http://WWW.medcalc.org;2018).

Categorical variables were expressed in terms of frequency and percentages (where applicable), continuous variables expressed as mean and SD. Normal distribution was verified by Shapiro-Francia test. Independent t test / Mann-Whitney test (where applicable) was used to check for significance of observations between two groups. Chi-square or Fisher's exact test (wherever applicable) is done to check for independence of attributes. In all the tests performed, P <0.05 is considered to be significant.

Result:

Table 1: Demographic Parameters

There appeared no difference in age and gender distribution of the two groups

Demographic parameters	Normal saline Group N Mean ± SD	Dexmedetomidine Group D Mean ± SD	P value
Age (yrs)	37.17±12.55	36.06±11.90	0.607(>0.05)
Weight (kg)	63.42±10.78	61.68±9.25	0.326(>0.05)
Sex (M/F)	38/27	34/31	>0.05

Table 2: Comparison of heart rate at various points between two groups

	Normal Saline Group		Dexmedetomidine Group		P Value
HR	Mean	SD	Mean	SD	
Baseline	81.20	14.17	82.08	13.90	0.722
After nebulisation	81.20	13.99	81.77	13.83	0.816
Before laryngoscopy	81.85	14.06	78.85	14.12	0.227
Just after intubation	93.45	16.65	85.86	13.97	0.006
1 min post intubation	90.85	14.95	84.92	13.36	0.019
5 min post intubation	87.85	14.48	82.51	13.21	0.029
10 min post intubation	88.57	13.61	82.40	13.06	0.009

Table 3: comparison of mean systolic blood pressure (in mmHg) at various time points between two groups

	Normal Saline Group		Dexmedetomidine Group		P Value
SBP	Mean	SD	Mean	SD	
Baseline	120.91	11.49	123.31	12.82	0.263
After nebulisation	120.9	11.23	122.15	12.18	0.545
Before laryngoscopy	117.05	11.15	116.51	10.95	0.782
Just after intubation	129.83	12.72	124.89	11.52	0.22
1 min post intubation	127.57	11.98	123.48	11.19	0.046
5 min post intubation	126.45	11.10	122.20	11.30	0.032
10 min post intubation	121.78	10.99	121.74	11.68	0.982

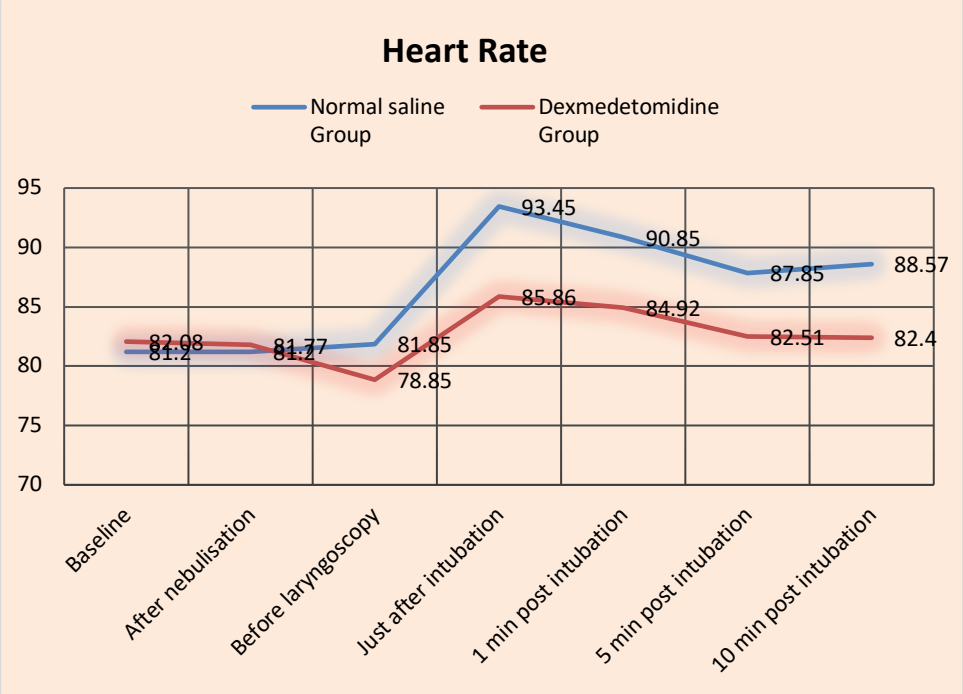

Figure 1: Comparison of heart rate at various points between two groups

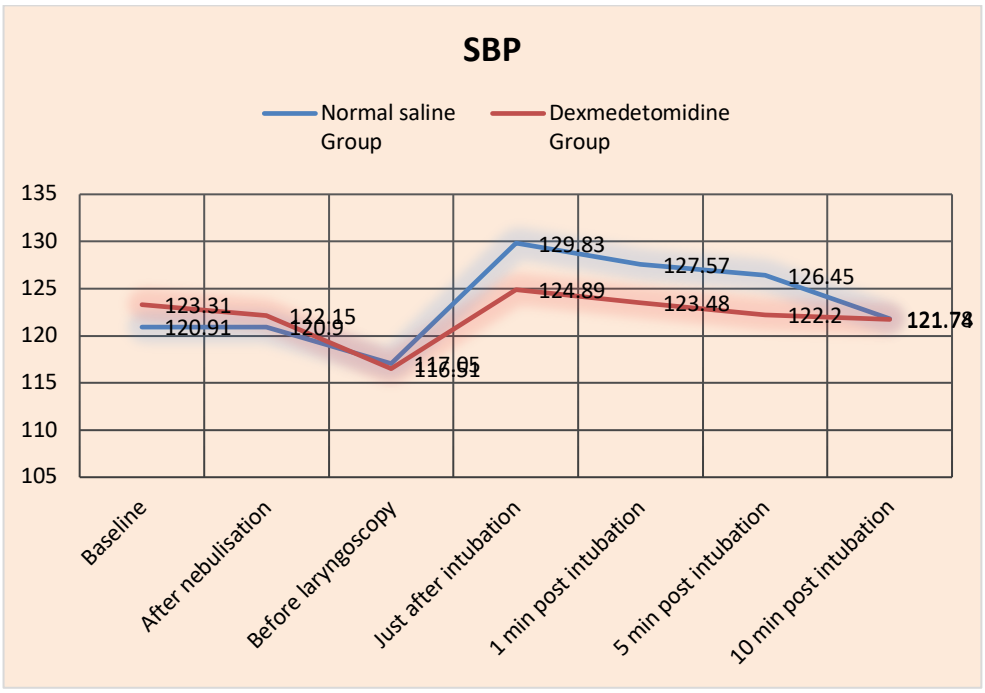

Figure 2: Comparison of mean systolic blood pressure (in mmHg) at various time points between two groups

Table 4: Comparison of mean systolic blood pressure(in mmHg) at various time points between two groups

	Normal Saline Group		Dexmedetomidine Group		P Value
SBP	**Mean**	**SD**	**Mean**	**SD**	
Baseline	120.91	11.49	123.31	12.82	0.263
After nebulisation	120.9	11.23	122.15	12.18	0.545
Before laryngoscopy	117.05	11.15	116.51	10.95	0.782
Just after intubation	129.83	12.72	124.89	11.52	0.22
1 min post intubation	127.57	11.98	123.48	11.19	0.046
5 min post intubation	126.45	11.10	122.20	11.30	0.032
10 min post intubation	121.78	10.99	121.74	11.68	0.982

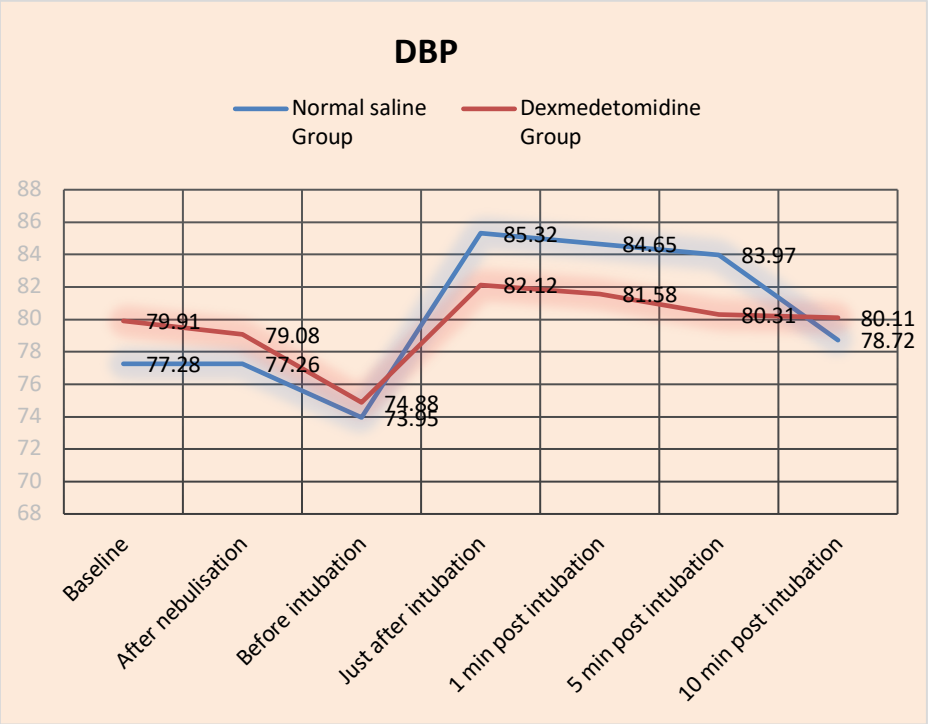

Figure 3: Comparison of mean systolic blood pressure (in mmHg) at various time points between two groups

Table 5: Comparison of mean arterial blood pressure (in mmHg) at various points between two groups

	Normal Saline Group		Dexmedetomidine Group		P Value
MAP	**Mean**	**SD**	**Mean**	**SD**	
Baseline	91.45	9.53	94.12	9.58	0.113
After nebulisation	91.49	9.39	93.12	9.04	0.315
Before intubation	87.94	9.51	88.66	8.66	0.651
Just after intubation	99.88	10.07	96.14	8.96	0.027
1 min post intubation	99.21	9.64	95.28	8.67	0.015
5 min post intubation	98.15	8.83	94.23	8.81	0.012
10 min post intubation	93.05	8.99	94.68	9.57	0.319

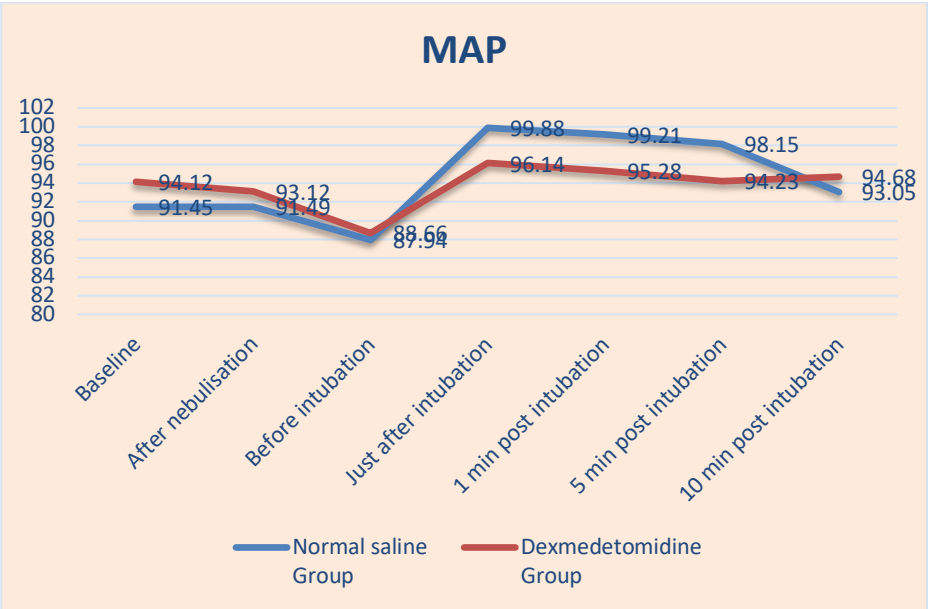

Figure 4: Comparison of mean arterial blood pressure (in mmHg) at various points between two groups

Table 6: Showing Ramsay Sedation Scale Score After Nebulisation

Group		Sedation Score After Nebulisation						Total
		1	2	3	4	5	6	
Normal Saline	Count	27	38	0	0	0	0	65
	% within grp	41.50%	58.50%	0.00%	0.00%	0.00%	0.00%	100.00%
Dexmedetomidine	Count	18	44	3	0	0	0	65
	% within grp	27.70%	67.70%	4.60%	0.00%	0.00%	0.00%	100.00%
Total	Count	45	82	3	0	0	0	130
	% within grp	34.60%	63.10%	2.30%	0.00%	0.00%	0.00%	100.00%

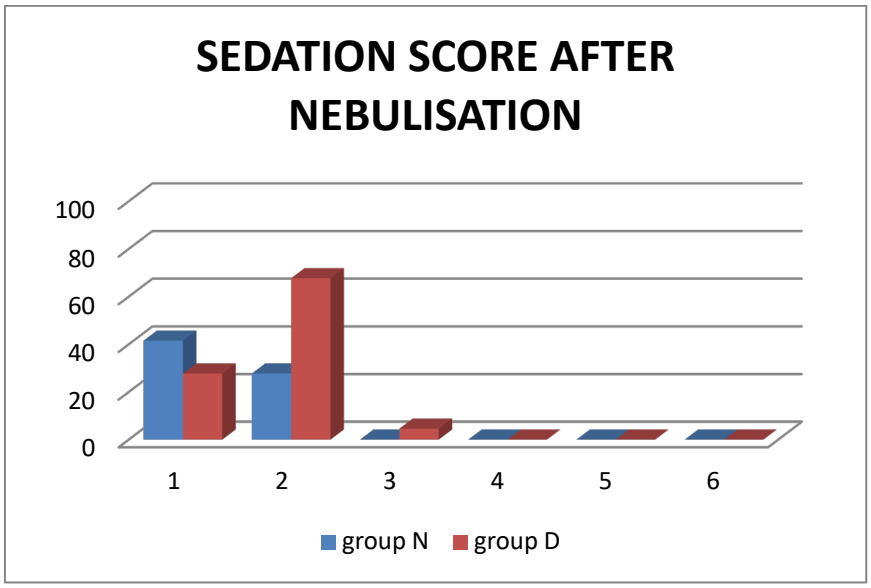

Figure 5: Ramsay Sedation Scale Score After Nebulisation

Two groups were comparable and there was no statistically significant difference between mean age, sex, weight.

The baseline mean HR±SD in the present study group N and group D were 81±14.17 and 82.08±13.90 respectively with no clinical statistical difference ($p>0.05$). After intubation in group N mean HR increased to 93.45±16.58 from basal mean HR, whereas in group D after intubation mean HR increased to 85.86±13.97 which was statistical highly significant when compared to group N ($p<0.01$). At 1, 5, 10 minutes after intubation in group N mean HR was 90.85±14.95, 87.85±14.48, 88.57±13.61 respectively whereas in group D mean HR was 84.92±13.36, 82.51±13.21, 82.40±13.06 respectively, which was statistical significant when compared to group N ($p<0.05$).

The baseline mean SBP±SD in group N and group D were 117.05±11.15 and 116.51±10.95 respectively with no clinical statistical difference ($p>0.05$). Just after intubation in group N mean SBP increased to 129.83±12.72 from basal mean SBP whereas in group D after intubation mean SBP increased to 124.89±11.52 mmHg, which was statistical significant when compared to group N ($p<0.05$). At 1, 5 minutes after intubation in group N mean SBP was observed 127.5±11.98, 126.45±11.10 respectively whereas in group D mean SBP was 123.48±11.19, 122.20±11.30 respectively, which was statistical significant when compared to group N ($p<0.05$). At 10 minutes mean SBP±SD in group N and group D were 121.78±10.99 and 121.74±11.68 respectively, with no clinical statistical difference ($p>0.05$)

The baseline mean DBP±SD in group N and group D were 77.28±9.32 and 79.91±8.79 mmHg respectively, with no clinical statistical difference ($p>0.05$). Just after intubation in group N mean DBP increased to 85.32±9.58 whereas in group D after intubation mean DBP increased to 82.12±8.40 mmHg from basal mean DBP which was statistical significant when compared to group N ($p<0.05$) At 1, 5 minutes after intubation in group N mean DBP was 84.65±9.25, 83.97±8.42 mmHg respectively whereas in group D mean DBP was 81.58±8.16, 80.31±8.29 mmHg, which was statistical significant compared to group N ($p<0.05$). At 10 minutes mean DBP±SD in group N and group D was 93.0±8.99 and 94.68±9.57 respectively, with no clinical statistical difference ($p>0.05$)

The baseline mean MAP±SD in group N and group D were 91.45±9.53 and 94.12±9.58 respectively, with no clinical statistical difference ($p>0.05$). Just after intubation in group N mean MAP increased to 99.88±10.07 mmHg whereas in group D after intubation mean MAP increased to 96.14±8.96

mmHg from basal mean MAP, which was statistical significant when compared to group N ($p<0.05$). At 1, 5 minutes after intubation in group N mean MAP were 99.21±9.64 , 98.15±8.83 whereas in group D mean MAP were 95.28±8.67, 94.23±8.81 respectively which was statistical significant when compared to group N ($p<0.05$). At 10 minutes mean MAP±SD group N and group D were 93.05±8.99 and 94.68±9.57 respectively, with no clinical statistical difference ($p>0.05$)

None of the patients developed sedation requiring any intervention in both groups. None of the patients had bradycardia while only one patient had episode of hypotension post-nebulisation with dexmedetomidine.

Discussion

Dexmedetomidine is a highly selective alpha 2 agonist which can be used via intravenous, intranasal, nebulisation route to attenuate hemodynamic response to laryngoscopy and intubation.

In our study, we compared 130 patients by dividing them in two groups containing 65 patients in each group to facilitate laryngoscopy intubation, Group D (n=30) - received Dexmedetomidine and Propofol, Group F (n=30) - received Fentanyl and Propofol, Group M (n=30)- received Fentanyl and Midazolam.

Hemodynamic response was evaluated using parameters such as heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP).

There was no significant difference between the two groups with respect to demographic profiles. Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure were comparable in both groups at baseline with no statistically significant difference($p>0.05$).

There was no statistically significant difference found between the groups with respect to heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure after nebulisation and just before intubation($p>0.05$).

After intubation rise in heart rate , systolic blood pressure, diastolic blood pressure, mean arterial pressure were statistically significant in group N when compared to group D ($p<0.05$). At 1, 5 min post-intubation there was a statistically significant difference($p>0.05$) in heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure between the two groups. While 10 mins post intubation difference in heart was statistically significant between two groups, the difference in systolic blood pressure, diastolic blood pressure, mean arterial pressure were not statistically significant between the two groups ($p>0.05$).

In a study by Satyajeet et al (2020) [10] also found a significantly lower trend of increase in HR in the dexmedetomidine group versus the saline group ($P = 0.012$) similar to our study.

Similar to our study Shrivastava P, et al (2022) [11] also observed that heart rate, SBP, DBP, MAP were attenuated in the dexmedetomidine group at 1, 5 min in a statistically significant manner.

Conclusion

Based on our experience in the present study, we conclude that dexmedetomidine via nebulisation route in dose 1mcg/kg can efficiently be used to attenuate haemodynamic response to laryngoscopy and endotracheal intubation.

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