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ORIGINAL RESEARCH

Efficacy of dexmedetomidine in attenuation of haemodynamic response to laryngoscopy and endotracheal intubation

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ABSTRACT

Background and Aims: Laryngoscopy and endotracheal intubation are associated with strong sympathetic responsein the form of tachycardia and hypertension. The aim of this study was to evaluate the efficacy of intravenous Dexmedetomidine in attenuation of haemodynamic response to laryngoscopy and endotracheal intubation.

Materials and methods: In this prospective, randomized, double blinded study,A total of hundred patients of ASA grade I and II between 18 to 50 years of age scheduled for various elective surgical procedures under general anesthesiawere selected and randomized into two groups of fifty patients each. Group C received 10 ml of normal saline intravenously over 10 min, 10 minutes prior to induction. Group D received injection Dexmedetomidine 0.5µg/kg body weight diluted to 10 ml normal saline intravenously over 10 min, 10 minutes prior to induction. Baseline parameters like Heart rate [HR], Systolic blood pressure [SBP], Diastolic blood pressure [DBP] and Mean arterial pressure [MAP] were recorded in all patients before giving studydrug, 2 ,5 and 8 minutes after studydrug, just beforeinduction, immediately after induction, 1,3,4,10 minute after laryngoscopy andintubation.

Results: There was no significant difference in the Age, Gender, body weight of patients between Group C and Group D. After induction, In group D, there was no statistically significant increase in the mean HR, SBP, DBP and MAP compared to basal value whereas in group C, there was a statistically significant increase in mean HR, SBP, DBP and MAP compared to basal value in group C.

Conclusion: In the present study, Dexmedetomidine at a dose of 0.5µ/kg body weight given 10 minutes before induction significantly attenuated the haemodynamic responses to laryngoscopy and tracheal intubation without significant side effects.

Keywords: laryngoscopy, endotracheal intubation, dexmeditomidine, haemodynamic response

INTRODUCTION

Laryngoscopy and endotracheal intubation are essential tools in hands of an anaesthesiologist in maintaining airway. Laryngoscopy and endotracheal intubation in adults are commonly accompanied by increase in arterial blood pressure and heart rate.[1]The magnitude of haemodynamic changes observed may be dependent on various factors such as depth of

anaesthesia, whether any measures are taken prior to airway manipulation, the anaesthetic agent used, the duration of laryngoscopy and intubation. To date, the exact mechanism of haemodynamic responses to laryngoscopy and intubation has not been clarified. The principle mechanism in hypertension and tachycardia is the sympathetic response[2,3]which may be the result of increase in catecholamineactivity.Transient hypertension and tachycardia are probably of no consequence in healthy individuals but either or both may be hazardous to those with hypertension, myocardial insufficiency or cerebrovascular diseases. This laryngoscopic reaction in such individuals may predispose to development of pulmonary edema, myocardial insufficiency and cerebrovascular accident.[2]

Intravenous anaesthetic induction agents do not adequately or predictably suppress the circulatory responses evolved by endotracheal intubation.So prior to initiating laryngoscopy, additional pharmacological measures like use of volatile anaesthetics,topical and intravenous Lidocaine,Opioids,vasodilators – Sodium nitroprusside,Nitroglycerine,Calcium channel blockersand β-blockershave been tried by variousauthors.Besides minimizing the cardiovascular response, anaesthesia induction for patients at risk mustalso satisfy the requirements. It must be applicable regardless of patient group, prevent impairment of cerebral blood flow and avoid awareness of the patient, it should neither be time consuming nor affect the duration or modality of the ensuing anaesthesia and also should not have any effect on the recovery characteristics.None of the drugshave been found to be effective to attenuate the sympathetic response to intubation and also not able to meet all the required criteria. Hence there is a need of finding out the drugs which can meet both the requirements.Various studies have also found that Dexmedetomidine can decrease the haemodynamic response to laryngoscopy and intubation.

The present study is aimed at attenuation ofhaemodynamic response to laryngoscopy and endotrachealintubationwith Dexmedetomidine at 0.5µg/kg body weight.

MATERIALS AND METHODS

This Prospective Randomized Double Blind Controlled Clinical study was conducted from October 2021 to January 2022 in hundred patients, scheduled for various elective surgical procedures under general anaesthesia in atertiary care teaching institute, after obtaining ethical committee clearance as well as informed consent from allpatients.

INCLUSION CRITERIA

Adult patients aged between 18 and 50 years of bothsex, belonging to ASA class I andII, Mallampatti grade I andIIundergoingelective surgeries under generalanaesthesia were included in this study.

EXCLUSION CRITERIA

Patients with cardiac, coronary, renal, hepatic, cerebral diseases and peripheral vasculardiseases, hypertension , heart rate less than 60 bpm, systolic blood pressure less than 100 mm of Hg and presence of 1^{st}, 2^{nd} or 3^{rd} degree heartblock, difficult airway and obese patients(BMI>30) , endocrinal diseases like hyperthyroidism, hypothyroidism and diabetesmellitus.

SAMPLE SIZE

A sample size was determined by power analysis performing a pilot study. A sample size of 50 subjects per group was required to detect a 20% change in heart rate and blood pressure between baseline and intubation, with a power of 80 % at 5% significancelevel.

MODE OF SELECTION

The study population was randomly divided into two groups with 50 patients in each group using sealed envelopes containing the name of the group and patient is asked to pick up the envelope. The envelope was opened by senior anaesthesiologist who was assigned to prepare the solutions and not involved with the study.**Group C - Control group (n=50)** received 10 ml of normal saline intravenously over 10 min, 10 minutes prior to induction using syringe pump.**Group D – Dexmedetomidine (n=50)**received injection Dexmedetomidine 0.5µg/kg body weight diluted to 10 ml normal saline intravenously over 10 min, 10 minutes prior to induction using syringepump.

Pre-anaesthetic evaluation was done on the evening before surgery with complete history, clinical, airway and systemic examination of cardiovascular and respiratory was done.All patients underwent the basic investigations. All patients included in the study were premedicated with tablet Alprazolam 0.5 mg and tablet Ranitidine 150 mg orally at bed time the previous night before surgery. On arrival of the patient in the operating room, an 18-gauge intravenous cannula was secured and an infusion of ringer lactate was started. The patients were connected to multiparameter monitor that records heart rate, non-invasive measurements of SBP, DBP, MAP, EtCO2 and continuous ECG monitoring and oxygen saturation. The baseline systolic, diastolic blood pressure, mean arterial pressure and heart rate were recorded.

The premedication, induction agent and muscle relaxant were standardised for both the groups.

After recording the baseline reading, study group – group D patients were given Dexmedetomidine 0.5µg/kg body weight diluted in 10 ml normal saline intravenously over 10 min, 10 min before induction using syringe pump and in control group-group C normal saline 10 ml given intravenously over 10 min, 10 min before induction using syringe pump. The study drug was prepared by the senior anaesthesiologist who was not involved with the study and observer as well as patient was blinded for the study. 50 µg of Dexmedetomidine (0.5 ml) was added to 9.5 ml of normal saline and made to 10 ml with each ml containing 5 µg of Dexmedetomidine. Based on the body weight, volume of the diluted drug in saline or normal saline is infused through a syringe pump.

All the patients were premedicated with injection Glycopyrrolate 0.008mg/kg body weight, injection Midazolam 0.02mg/kg body weight and injection Fentanyl 1µ/kg body weight IV after test drug administration. Then patients were preoxygenated with 100% oxygen for 3 minutes. Patient were induced with injection Propofol 2 mg/ kg body weight, as a 1% solution till loss of response to verbal commands occurred. Endotracheal intubation was facilitated with injection Vecuronium 0.1mg/kg, intubation was performed with appropriate size oral cuffed portex endotracheal tube using Macintosh no.3 blade lasting for not more than 15 seconds and after confirmation of bilateral equal air entry,theendotracheal tube was fixed. If time for laryngoscopy and intubation exceeds 15 seconds, such patients were excluded from the study.

Anaesthesia was maintained with intermittent positive pressure ventilation with 66% nitrous oxide and 33% of oxygen and sevoflurane 1 %, vecuronium using circle absorber system connected to Boyle's machine. After the patients recovered from loading dose of vecuronium further neuromuscular blockade was maintained with vecuronium 0.02 mg/ kg body weight. No surgical or any other stimulus was applied during the first 10 minutes of study period. At the end of the procedure, patients were reversed with injection Neostigmine 0.05 mg/kg body weight and injection glycopyrrolate 0.01 mg/ kg body weight. Sedation at the end of the surgery was assessed using Ramsay sedation score.

The following cardiovascular parameters were recorded in all patients.Heart rate [HR] in beats perminute, Systolic blood pressure [SBP] in mm ofHg, Diastolic blood pressure [DBP]

in mm ofHg, Mean arterial pressure [MAP] in mm ofHg.The cardiovascular parameters were monitored at time interval are Basal, before giving studydrug, 2, 5,8 minutes after studydrug, beforeinduction, afterinduction, 1,3,4,10 minutes after laryngoscopy andintubation. Incidences of all these parameters were recorded in both the groups.The side effects of the study drug like hypotension, bradycardia and sedation were noted.Sedation scoring was done as per Ramsay sedation scale.

STATISTICAL ANALYSIS

All the data from the patients in two groups were collected and subjected to statistical analysis in a systematicway.Data were collected, tabulated, coded then analysed using Statistical Programme in Social Sciences (SPSS) for windows (version27.0).Numerical values were presented as mean value and standarddeviation. As regard to numerical variables unpaired t test was used whenever appropriate for between groupcomparisions.Paired t test was used to compare the variable before and afterintervention.Chi square test was used to analyse the categorical data and for testing the association between the variables.ANOVA was used to measure the related dependent variables that represent different measurements of the sameattribute.$p<0.05$ was considered as significant and $p<0.01$ was considered as highly significant.

RESULTS

Table 1: Showing the demographic distribution

Age (years)	Group C (Control)		Group D (Dexmedetomidine)	
	No. of patients	Percentage (%)	No. of patients	Percentage (%)
18-28	12	24	13	26
29-38	13	26	12	24
39-48	15	30	20	40
49-55	10	20	05	10
Total	50		50	
Age (Mean±SD)	36.8 ±9.7		36.42 ± 9.36	
t-value	0.209			
p-value	0.835 (NS)			
Gender				
Male	27	54	22	49
Female	23	56	28	56
Weight in kgs				
40-44	0	0	3	6
45-49	10	20	8	16
50-54	10	20	14	28
55-59	9	18	13	26
60-64	18	36	6	12
65-69	2	4	4	8
70+	1	2	2	4
Total	50	100	50	100
Weight (Mean± SD)	56.12±6.15		55.34±7.56	
Minimum body weight in kg	45		43	
Maximum body weight in kg	70		73	
t-value	1.322			
p-value	0.189 (NS)			

Demographic variables are insignificant when compared in both groups.

Table-2: Showing the type and mean duration of surgicalprocedures

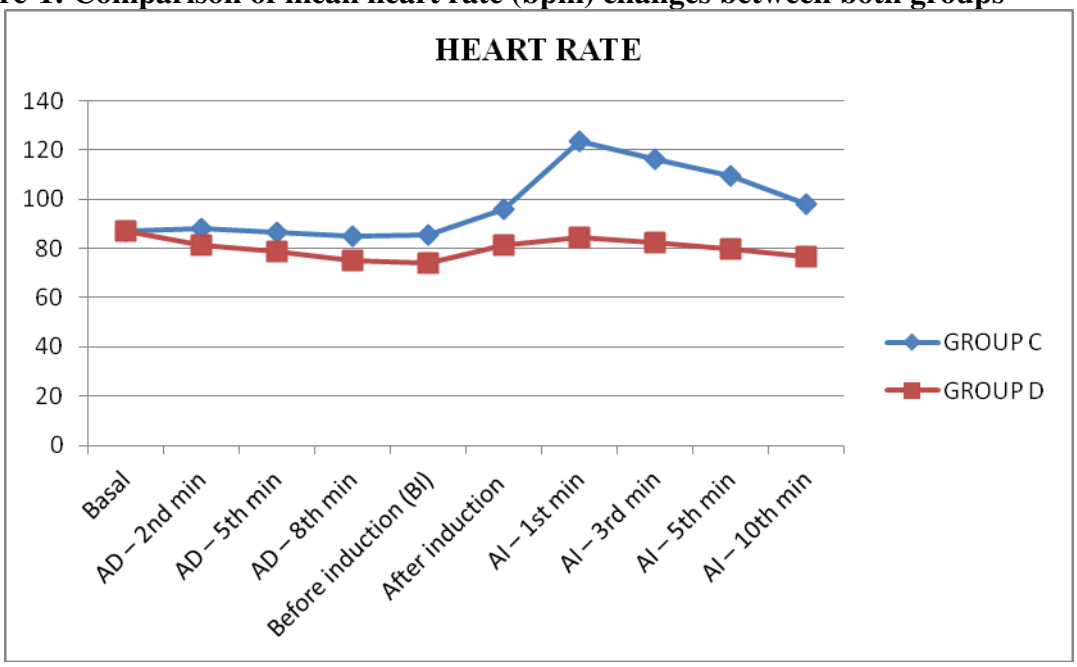
Type of surgery	Number of patients	
	Group C (Control)	Group D (Dexmedetomidine)
General surgeries	26	18
Gynecological surgeries	05	11
Orthopedic surgeries	9	7
ENTsurgeries	10	14
Duration of surgery (minutes)	90.08±10.7	88.92±11.8

In control group the mean duration of surgery was 90.08 minutes and in Dexmedetomidine group 88.92 minutes which was statistically not significant (p=0.078).

Table 3: Showing the intragroup and intergroup comparison of mean heart rate (bpm) changes between both groups

TIME	GROUP C			GROUP D			p-value C vs D	Remarks
	Heart Rate (HR)	p-value	Remarks	Heart Rate (HR)	p-value	Remarks		
Basal	87.36±9.96			87.36±13.58			1.000	NS
AD – 2nd min	88.18±9.77	0.086	NS	81.28±14.25	0.000	HS	0.006	HS
AD – 5th min	86.62±9.08	0.057	NS	78.64±13.47	0.000	HS	0.001	HS
AD – 8th min	84.82±9.92	0.090	NS	75.26±12.62	0.000	HS	0.000	HS
Before induction (BI)	85.30±9.81	0.072	NS	74.08±11.09	0.000	HS	0.000	HS
After induction	96.24±9.76	0.000	HS	81.26±12.08	0.000	HS	0.000	HS
AI – 1st min	123.6±10.46	0.000	HS	84.50±11.41	0.079	NS	0.000	HS
AI – 3rd min	116.4±9.16	0.000	HS	82.38±11.28	0.008	HS	0.000	HS
AI – 5th min	109.62±9.17	0.000	HS	79.88±11.93	0.000	HS	0.000	HS
AI – 10th min	98.30±9.82	0.000	HS	76.90±10.77	0.000	HS	0.000	HS

Figure-1: Comparison of mean heart rate (bpm) changes between both groups



The basal heart rate were comparable in both groups (p=1.000). Statistical evaluation between the groups showed a significant fall in HR in group D at 2, 5 and 8 minutes of drug administration and before and after induction. The mean HR increase observed at 1, 3, 5 and

10 minutes after intubation in group C was statistically highly significant compared to mean HR in group D (p=0.000).

Table 4: Showing the intragroup and intergroup comparison of mean systolic blood pressure (SBP in mm of hg) changes between both groups

TIME	GROUP C			GROUP D			p-value C vs D	Remarks
	Mean SBP	p-value	Remarks	Mean SBP	p-value	Remarks		
Basal	128.00±6.09			127.38±11.30			0.734	NS
AD – 2nd min	127.72±6.65	0.678	NS	129.26±12.95	0.165	NS	0.456	NS
AD – 5th min	127.76±5.62	0.382	NS	116.92±11.99	0.000	HS	0.000	HS
AD – 8th min	127.18±5.84	0.134	NS	111.32±11.80	0.000	HS	0.000	HS
Before induction (BI)	127.90±6.86	0.906	NS	110.02±10.03	0.000	HS	0.000	HS
After induction	121.30±5.11	0.000	HS	103.68±11.15	0.000	HS	0.000	HS
AI – 1st min	158.02±4.41	0.000	HS	111.52±9.95	0.000	HS	0.000	HS
AI – 3rd min	149.02±8.14	0.000	HS	105.04±11.13	0.000	HS	0.000	HS
AI – 5th min	138.70±8.26	0.000	HS	103.04±12.38	0.000	HS	0.000	HS
AI – 10th min	128.78±6.35	0.449	NS	101.30±11.12	0.000	HS	0.000	HS

The mean SBP were comparable in both groups (p=0.734). After 2 min of drug administration the change in SBP was not significant (0.456). The mean SBP values at 5 and 8 minutes of drug administration, before and after induction were significantly low (p=0.000) in group D compared to group C. The increase in SBP in group C at 1, 3, 5 and 10 minutes after intubation was statistically highly significant (p=0.000) compared to group D.

Figure-2: Showing intergroup comparison of mean systolic blood pressure (SBP in mmHg) between both groups

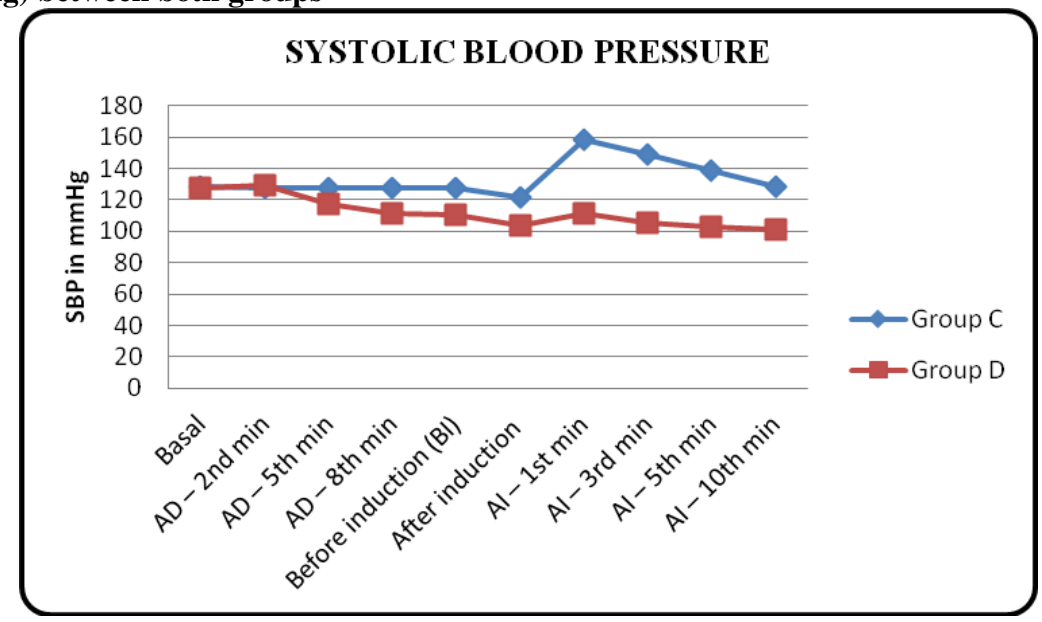
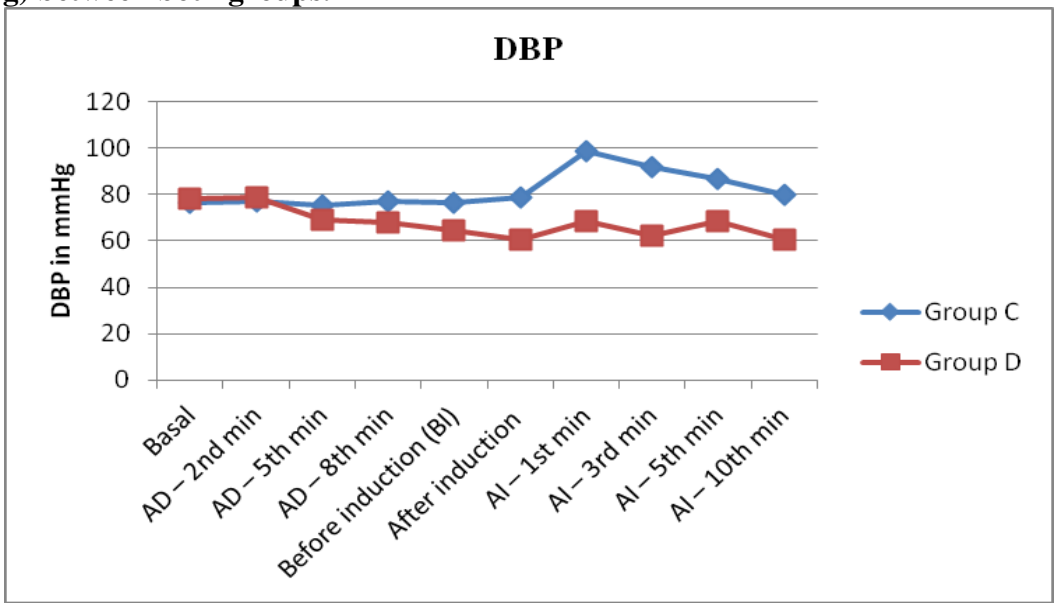


Table 5: Showing the intragroup and intergroup comparison of mean diastolic blood pressure (DBP in mm of hg) changes between both groups

TIME	GROUP C			GROUP D			p-value C vs D	Remarks
	Mean DBP	p-value	Remarks	Mean DBP	p-value	Remarks		
Basal	76.64±6.08			78.32±7.53			0.223	NS
AD – 2nd min	76.78±5.99	0.824	NS	78.88±6.65	0.456	NS	0.674	NS
AD – 5th min	75.32±7.64	0.175	NS	69.14±6.81	0.000	HS	0.015	S

AD – 8th min	76.78±5.99	0.820	NS	68.16±7.00	0.000	HS	0.000	HS
Before induction (BI)	76.58±5.45	0.928	NS	64.32±9.85	0.011	S	0.009	HS
After induction	70.68±3.33	0.022	S	60.32±10.63	0.001	HS	0.000	HS
AI – 1st min	98.98±3.72	0.000	HS	68.78±9.41	0.000	HS	0.000	HS
AI – 3rd min	92.06±7.39	0.000	HS	62.20±10.70	0.000	HS	0.000	HS
AI – 5th min	86.18±8.04	0.000	HS	68.68± 9.48	0.000	HS	0.000	HS
AI – 10th min	79.76±8.12	0.039	S	60.60±8.93	0.000	HS	0.000	HS

Figure-3: Showing intergroup comparison of mean diastolic blood pressure (DBP in mmHg) between both groups.



The mean basal DBP are comparable in both groups (p=0.223). The mean DBP, 2 min after drug administration was statistically not significant (p=0.674). The mean DBP values at 5 and 8 minutes of drug administration, before and after induction were significantly low (p=0.000) in group D compared to group C. The increase in DBP in group C at 1, 3, 5 and 10 minutes after intubation was statistically highly significant (p=0.000) compared to group D.

Table 6: Showing the intragroup and intergroup comparison of mean arterial pressure (MAP in mm of hg) between both groups

TIME	GROUP C			GROUP D			p-value C vs D	Remarks
	Mean MAP	p-value	Remarks	Mean MAP	p-value	Remarks		
Basal	91.84±3.44			90.84±3.44			1.000	NS
AD – 2nd min	92.08±4.85	0.689	NS	91.88±3.40	0.942	NS	0.812	NS
AD – 5th min	91.54±3.47	0.680	NS	86.24±4.47	0.000	HS	0.000	HS
AD – 8th min	91.48±4.04	0.647	NS	81.70±6.02	0.000	HS	0.000	HS
Before induction (BI)	92.48±3.29	0.378	NS	83.32±7.00	0.000	HS	0.000	HS
After induction	84.80±4.85	0.000	HS	81.02±8.11	0.000	HS	0.000	HS
AI – 1st min	118.26±3.42	0.000	HS	88.86±7.65	0.000	HS	0.000	HS
AI – 3rd min	110.18±6.94	0.000	HS	80.44±7.84	0.000	HS	0.000	HS
AI – 5th min	102.84±7.24	0.000	HS	79.34±8.02	0.000	HS	0.000	HS
AI – 10th min	95.96±6.61	0.000	HS	80.54±8.85	0.000	HS	0.000	HS

The mean basal MAP are comparable in both groups (p=1.000). After 2 min of drug administration the change in MAP was statistically not significant (p=0.812). There was a significant difference in MAP values at 5th min, 8th min after drug administration and before and after induction which was statistically highly significant (p=0.000). The increase in MAP

in group C was statistically highly significant at 1 min and 3, 5 and 10 minutes after intubation (p=0.000) compared to group C.

Figure-4: Showing intergroup comparison of mean arterial blood pressure (MAP in mmHg)between both groups.

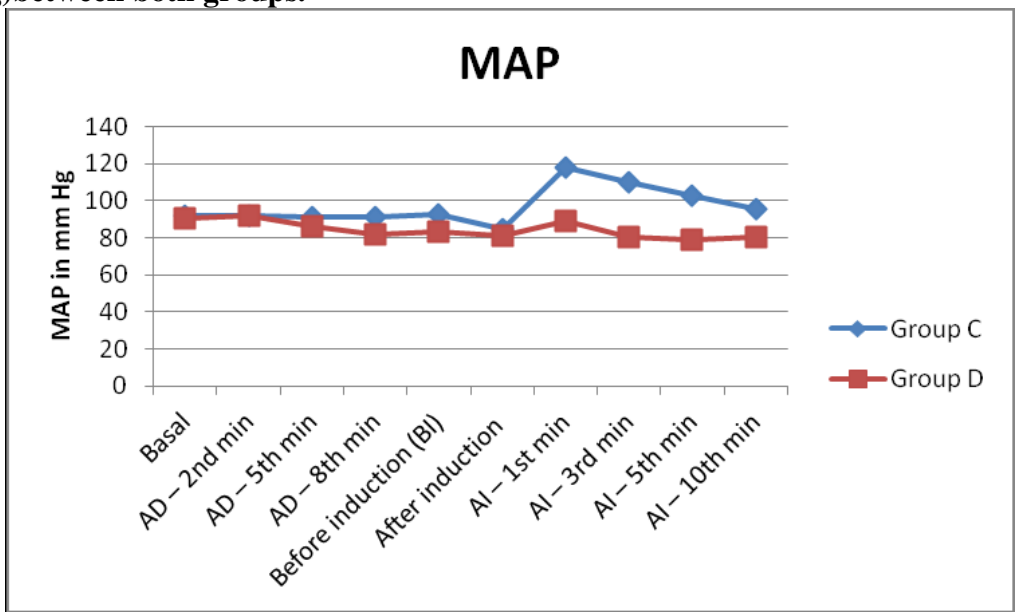


Table-7: Comparision of Sedation score in both groups

	Group C	Group D	p-value
Sedation score	2.62±0.49	2.52±0.43	0.087 (NS)

In group C sedation score was 2.62±0.49 and in group D the score was 2.52±0.43. Statistical evaluation showed no difference in the sedation score between the two groups.

Table-8: Comparision of side effects between both groups

	Nil	Bradycardia	Hypotension	Bradycardia and hypotension	Treatment required
Group C	50	0	0	0	
Group D	42	4	3	1	1
p-value	0.034 (S)				

In group C, none of the patients had side effects like bradycardia and hypotension. In group D, 4 patients had bradycardia, 3 had hypotension and one patient had both bradycardia and hypotension.

DISCUSSION

The haemodynamic changes brought about by laryngoscopy and intubation was first described by Reid and Brace.[4] The haemodynamic response is initiated within seconds of direct laryngoscopy and further increases with endotracheal intubation. The response is initiated within 5 s of laryngoscopy, peaks in 1–2 min and returns to normal levels by 5 min.[5] These changes are usually short lived and well tolerated by normal patients. In patients with cardiovascular disease, it can incite harmful effects such as myocardial ischaemia, ventriculardysrrhythmias, ventricular failure and pulmonary oedema. It can also lead to cerebrovascular accidents in cerebrovascular disease patients.[6]

Various drug regimens and techniques such as lignocaine, opioids, nitroglycerine, calcium channel blockers such as diltiazem and β-blockers such as esmolol have been tried for

obtunding the stress response. α-2 receptor agonists mediate their action through α-2A receptors located in locus caeruleus, the predominant noradrenergic nuclei of upper brainstem. The presynaptic activation of α-2A receptors in the locus caeruleus inhibits the noradrenaline release and brings about sedation and hypnosis. Post-synaptic activation of α-2 receptors in central nervous system brings about decreased sympathetic activity leading to bradycardia and hypotension.[7]

Dexmedetomidine has been found by various authorsto blunt the haemodynamic response for laryngoscopy and intubation. Dexmedetomidine is eight times more potent α-2 receptor agonist than clonidine. The action of dexmedetomidine is short lived with an elimination half-time of 2 h. Dexmedetomidine has a reversal drug for its sedative effect called as atipamezole. These factors make dexmedetomidine superior to clonidine.[8,9]

The present study was done to evaluate the role of dexmeditomidine in a dose of 0.5µg/kg in suppressing the haemodynamic response to laryngoscopy and endotracheal intubation.A total of hundred patients were selected and randomized into two groups of fifty patients each. Group C received 10 ml of normal saline intravenously over 10 min, 10 minutes prior to induction. Group D received injection Dexmedetomidine 0.5µg/kg body weight diluted to 10 ml normal saline intravenously over 10 min, 10 minutes prior to induction. Baseline parameters like Heart rate,Systolic blood pressure,Diastolic blood pressure and Mean arterial pressure were recorded in all patients before giving studydrug, 2 ,5 and 8 minutes after studydrug, just beforeinduction, immediately after induction, 1,3,4,10 minute after laryngoscopy andintubation.

Both the groups were comparable and there was no statistically significant difference with regards to mean age, weight, sex and duration of surgery.

Bon Sebastian et al. in their study have compared between intravenous dexmedetomidine and normal saline for attenuation of the haemodynamic stress response to laryngoscopy and endotracheal intubation. The intergroup comparison reveal a statistically significant reduction in heart rate and MAP by dexmedetomidine than normal saline.[10]

In another study,Keniya VM et al observed that perioperative infusion of dexmedetomidine is effective in attenuating sympathoadrenal response to tracheal intubation. After tracheal intubation, maximal average increase are 8% in systolic and 11% in diastolic blood pressure in dexmedetomidine group, as compared to 40% and 25%, respectively, in the control group. Similarly, average increase in heart rate are 7% and 21% in the dexmedetomidine and control groups, respectively.[11]

In another study by Tanskanen et al,the role of dexmedetomidine as an anaesthetic adjuvant in intracranial tumour surgery is evaluated. It was observed that IV DEX can blunt the hypertensive and tachycardic responses to intubation and extubation.[12]

In a similar study by Sulaiman et al, 60 adult patients scheduled for elective off-pump coronary artery bypass surgery have been randomly allocated to receive dexmedetomidine (0.5 mcg/kg)or normal saline 15 min before intubation. Patients have been compared for haemodynamic changes (heart rate, arterial blood pressure and pulmonary artery pressure) at baseline, 5 min after drug infusion, before intubation and 1, 3 and 5 min after intubation. The dexmedetomidine group has a better control of haemodynamics during laryngoscopy and endotracheal intubation.[13]

The findings of all the above studies closely correlate with findings in our study.

In group C mean sedation score immediately after extubation was 2.62 and 2.52 in control group and Dexmedetomidine group which was statistically not significant (p=0.087). There was no difference found in both groups with respect to sedation and recovery which was similar to observation noted by Aanta et al.[14]Dexmedetomidine in a dose of 1 µg/kg has been shown to cause increased sedation levels and need for oxygen supplementation by few authors.[15,16] The lesser sedation scores in group D could be related to using a lower dose (0.5

μg/kg) of dexmeditomidine.
In Dexmedetomidine group, 4 patients developed bradycardia which was after 30 minutes of the drug administration and significant hypotension in 3 patients which was 20 minutes after intubation. One patient required inj. Atropine for bradycardia and no patient required vasopressors for correction of blood pressure. Hypotension was managed by decreasing volatile anaesthetic concentration and infusing intravenousfluids.

CONCLUSION

In the present study, Dexmedetomidine at a dose of 0.5μ/kg body weight given 10 minutes before induction significantly attenuated the haemodynamic responses to laryngoscopy and tracheal intubation without significant side effects.

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