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Intravenous Dexmedetomidine 0.6µg/kg and 1µg/kg for Attenuation of the Haemodynamic Response to Laryngoscopy and Intubation: A Clinical Study

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Abstract

Introduction: Laryngoscopy and intubation are noxious stimuli and are associated with haemodynamic responses in the form of laryngo-sympathetic stimulation which is manifested as hypertension, tachycardia. The magnitude of haemodynamic changes observed may be dependent on various factors such as the depth of anaesthesia, whether any measures are taken prior to airway manipulation, the anaesthetic agent used, the duration of laryngoscopy and intubation. *Aims*: The present study is aimed to comparing the effectiveness of two different doses of intravenous Dexmedetomidine, 0.6 µg/kg body weight and 1µg/kg body weight for attenuating haemodynamic response to laryngoscopy and endotracheal intubation and also to find out any adverse effects. *Materials and methods :* A Randomized, controlled study between 2 doses of intravenous dexmedetomidine–0.6 µg/kg body weight and 1 µg/kg body weight for attenuation of haemodynamic response to laryngoscopy and endotracheal intubation. was undertaken in 60 patients of either sex between 18 to and 55 yrs of age belonging to ASA-I undergoing elective general endotracheal anesthesia were selected for the study. *Results:* There was marked decrease in HR after dexmedetomidine administration. In group D-0.6 and group D-1 HR, SBP, DBP and MAP markedly increased at 1 minute following laryngoscopy and intubation in the control group where as in dexmedetomidine group there was a fall in HR, SBP, DBP and MAP at various intervals following intubation which was statistically significant. There was no statistical difference between the group D-0.6 and group D-1 with regard to haemodynamic parameters after laryngoscopy and endotracheal intubation. There was increased incidence of sedation in patients belonging to group D-1 when compared to group D-0.6 which was statistically significant. Incidence of bradycardia and hypotension was higher in group D-1 when compared to group D-0.6 which were managed easily. *Conclusion:* It is concluded that Dexmedetomidine obtunds the haemodynamic responses to laryngoscopy and endotracheal intubation and 0.6 µg/kg body weight is the ideal dose for the same.

Keywords: Dexmedetomidine; Haemodynamic Response; Laryngoscopy, Intubation

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Introduction

Laryngoscopy and endotracheal intubation are the most important and essential skills for an anaesthesiologist in maintaining the airway. However, both laryngoscopy and intubation are noxious stimuli and are associated with haemodynamic responses in the form of laryngo-

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sympathetic stimulation which is manifested as hypertension, tachycardia. The magnitude of haemodynamic changes observed may be dependent on various factors such as the 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 which may be the result of increase in catecholamine activity.

The increase in the heart rate and blood pressure are usually transitory, variable and unpredictable. Transitory hypertension and tachycardia are probably of no consequence in healthy individuals but either or both may be hazardous to those with hypertension, coronary artery disease or cerebrovascular diseases. This laryngoscopic reaction in such individuals may predispose to development of pulmonary edema, myocardial insufficiency, dysrrhythmias[9] and cerebrovascular accident.[1]

Intravenous anaesthetic induction agents do not adequately or predictably suppress the haemodynamic responses produced by endotracheal intubation.[10] So prior to initiating laryngoscopy, additional pharmacological measures like use ofvolatile anaesthetics, topical and intravenous lidocaine, opioids, vasodilators – sodium nitroprusside, Nitroglycerine, Calcium channel blockers and β-blockers have been tried by various authors. None of these drugs mentioned have been found to be effective to attenuate the sympathetic response to intubation.

Besides minimizing the cardiovascular response, anaesthesia induction for patients at risk must also satisfy the following requirements: it must be applicable regardless of the patient group, prevent impairment of cerebral blood flow and avoid awareness of the patient; it should neither be time consuming nor effect the duration or modality of the anaesthetic technique and also should not have any effect on the recovery characteristics of the patient.

Hence there is a need to find drugs which can suppress the cardiovascular response to intubation and also help in potentiating the effects of induction agents to meet the above requirement.

Various studies have found that Dexmedetomidine decreases the haemodynamic response to laryngoscopy and intubation. It has been introduced recently in India (2009).[2]

Various studies have used Dexmedetomidine in the dose of 0.6 µg/kg body weight and 1µg/kg body weight as intravenous bolus for attenuating the haemodynamic response. There is conflicting reports as to which dose of the drug is ideal to suppress the intubation response and also have minimal adverse effects.

Hence there is a need to know whether 0.6 µg/kg body weight to 1 µg/kg body weight is the ideal dose for attenuation of haemodynamic response to larynogoscopy and endotracheal intubation. Hence, the present study is aimed to comparing the effectiveness of two different doses of intravenous Dexmedetomidine, 0.6 µg/kg body weight and 1µg/kg body weight for attenuating haemodynamic response to laryngoscopy and endotracheal intubation and also to find out any adverse effects.

Materials and Methods

A study is Randomized, controlled study between 2 doses of intravenous dexmedetomidine– 0.6 µg/kg body weight and 1 µg/kg body weight for attenuation of haemodynamic response to laryngoscopy and endotracheal intubation. was undertaken in Department of Anesthesiology and Critical care at Gandhi Medical College and Hospital, 2017 to 2018.

After Institutional ethical committee approval, 60 patients of either sex between 18 and 55 yrs of age belonging to ASA-I undergoing elective general endotracheal anesthesia were selected for the study .

Inclusion Criteria: Adult patients aged between 18 and 55 years of both sex, ASA class 1 and Elective surgeries under general end tracheal anesthesia

Exclusion Criteria: Patients with cardiac, coronary, renal, hepatic, cerebral diseases and peripheral vascular diseases, hypertension, with difficult airway and obese patients (BMI > 30), with endocrinal diseases like hyperthyroidism, hypothyroidism and diabetes mellitus etc.Pregnant females, time for laryngoscope and intubation exceeding 15 seconds.

During the pre-operative visit, all patients were clinically evaluated, assessed and investigated as per study protocol a written informed consent was taken .

The study population (60 patients) was randomly divided into two groups with 30 patients in each

group using sealed envelopes containing the name of the group and patient asked to pick up the envelope. The envelope was opened by senior anesthesiologist who was assigned to prepare the solutions and was not involved with the study.

Group D-0.6: (n=30) received injection Dexmedetomidine 0.6 µg/kg body weight- diluted to 10 ml of normal saline, administered intravenously over 10 min.

Group D-1: (n=30) received injection Dexmedetomidine 1 µg/kg body weight- diluted to 10 ml of normal saline, administered intravenously over 10 min.

Pre-anesthetic evaluation was done an evening before surgery. A routine preanesthetic examination was conducted assessing General condition of the patient, Airway assessment by Mallampatti grading and rule of 1-2-3, Nutritional status and body weight of the patient

A detailed systemic examination and basic investigation were done in all patients. 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. They were kept nil orally 10 pm onwards on the previous night.

On arrival of the patient in the operating room, an 18-gauge intravenous cannula was inserted under local anaesthetic infiltration and an infusion of 500 ml Ringer Lactate was started. The patients were connected to multiparameter monitor which records heart rate, non-invasive measurements of SBP, DBP, MAP, $EtCO_2$ and continuous ECG monitoring and oxygen saturation. The baseline systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate were recorded (basal parameters). The cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II.

After recording the baseline readings, patients in group D-0.6, received Dexmedetomidine 0.6µg/ kg body weight diluted in 10 ml normal saline intravenously over 10 min using syringe pump, 10 min before induction.

Patients in group D-1, received Dexmedetomidine 1µg/kg body weight, diluted in 10 ml normal saline intravenously over 10 min using syringe pump, 10 min before induction.

The study drug was prepared by the senior anaesthesiologist who was not involved in the study and as such, the observer as well as patient were blinded for the study.

All patients were premedicated with injection midazolam-0.02mg/kg body weight and injection fentanyl 1µg/kg body weight IV after drug administration, 3 min before induction. Then patients were preoxygenated for 3 minutes via a face mask with closed circuit. Anaesthesia was induced with injection. propofol 1-1.5 mg/kg which continued till the patient's verbal response was abolished and dose of propofol required was noted Endotracheal intubation was facilitated with 1.5 mg/kg IV succinylcholine one minute prior to laryngoscopy and intubation. Laryngoscopy and intubation were performed using Macintosh no. 3 blade lasting for not more than 15 seconds and after confirmation of bilateral equal air entry and $EtCO_2$, the endotracheal tube was fixed.

Anaesthesia was maintained using 66% nitrous oxide and 33% of oxygen with 1% sevoflurane. After the patients recovered from succinylcholine, further neuromuscular blockade was maintained with vecuronium 0.05 mg/kg body weight initially and 0.5 mg increments as and when required. At the end of the procedure, total dose of vecuronium required for the surgery was recorded and patients were reversed with inj. Neostigmine- 0.05 mg/kg body weight and inj. Glycopyrolate- 0.01 mg/kg body weight. Sedation was assessed at the end of the surgery using Ramsay sedation score. The time for recovery was also noted, (the time from giving the reversal agent to extubation).

The cardiovascular parameters were recorded as Heart rate [HR] in beats per minute, Systolic blood pressure [SBP] in mm of Hg, Diastolic blood pressure [DBP] in mm of Hg and Mean arterial pressure [MAP] in mm Hg

The above cardiovascular parameters were monitored at time interval Basal-before giving study drug, 2, 5, 8, before, after, One, five and ten minutes after laryngoscopy and intubation

Incidences of side effects were recorded in all the three groups. The side effects of the study drug like hypotension, bradycardia and sedation were noted. Hypotension was treated using 3mg increments of IV mephenteramine and fluids. Bradycardia was treated using 0.6mg of IV atropine. Sedation scoring was done as per Ramsay sedation scale.

SPSS for windows (version 17.0) was employed for data analysis. $p < 0.005$ was considered as significant and $p < 0.01$ was considered as highly significant

Results

Age distribution of the patients in all two groups. The minimum age in group D-0.6, group D-1 were 18 years, 20 years, respectively. Maximum age in group D-0.6, group D-1 were 50 years, 46 years respectively. All two groups were similar with respect to age distribution and there is no statistical significance between the groups (p = 0.2972) (Table 1).

Table 1: Showing the demogrphic distribution

	Group D-0.6	Group D-1
	No of patients	**No of patients**
Age in years		
18 -20	3 (10)	1(3.33)
21-30	11(36.7)	11(36.7)
31-40	10(33.3)	14(46.66)
41-55	6(20)	4(13.33)
Total	30(100)	30(100)
Mean age in years ± SD	35.5667 ± 8.15	33.466 ± 6.79
p- value	0.2972(NS)	
Gender		
Male	16 (53.3)	15 (50)
Female	14 (46.7)	15 (50)
p-value	1.0000 (INS)	
Body Weight		
40-44	0 (0)	1 (3.3)
45-49	8 (26.7)	7 (23.3)
50-54	5(16.7)	7 (23.3)
55-59	9 (30.0)	7 (23.3)
60-64	3 (10.0)	5(16.7)
65-69	2 (6.7)	3 (10.0)
70 +	3 (10.0)	0 (0)
Main Body weight in kg ± SD	53.83 ± 8.54	53.83 ± 7.40
Minimum Body weight in kg	45	40
Maximum Body weight in kg	81	68
p-value	0.3269(NS)	

NS- Not significant

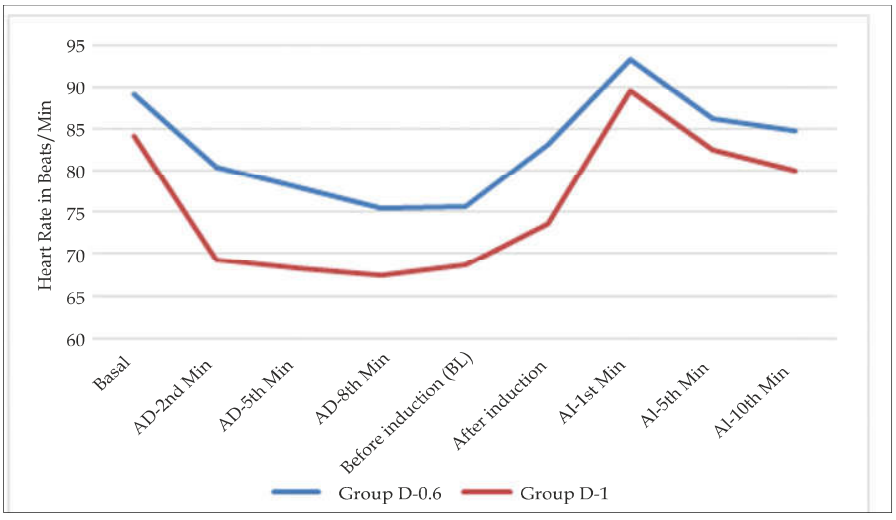

Fig. 1: Intergroup comparision of mean heart rate (bpm) changes in response to laryngoscopy and intubation between two groups

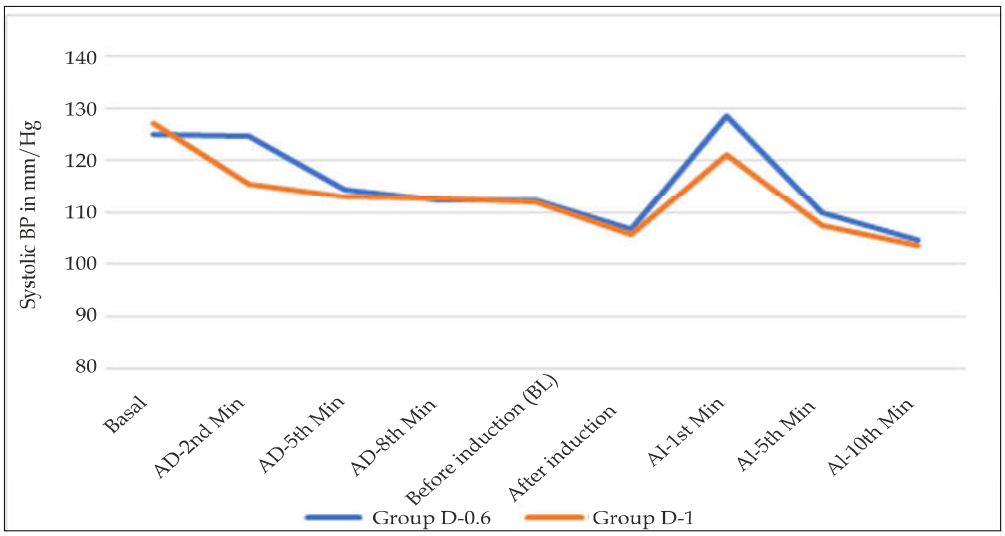

Fig. 2: Comparison of systolic blood pressure changes (mmHg) changes in response to laryngoscopy and intubation between two Groups

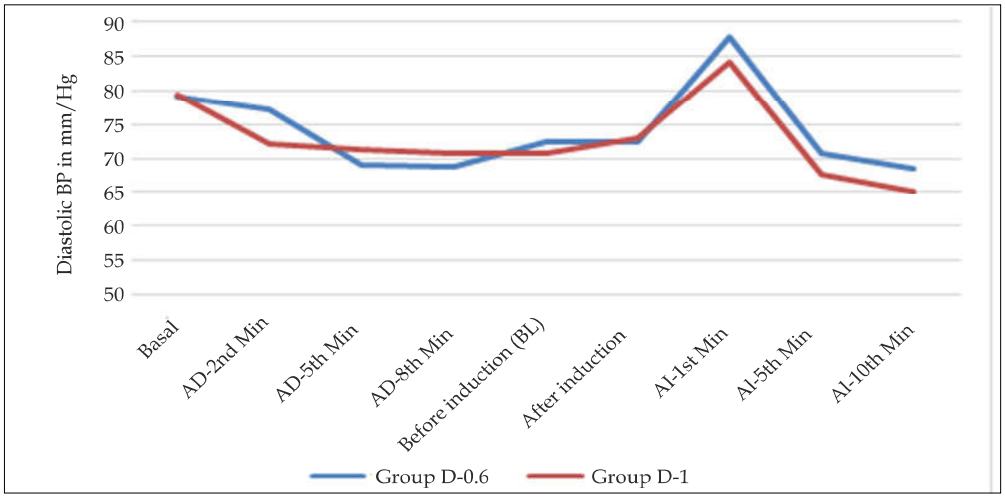

Fig. 3: Comparison of diastolic blood pressure changes (mmHg) changes in response to laryngoscopy and intubation between two Groups

The basal mean HR were comparable in both the groups. statistical evaluation between the groups shows statistical significance in the mean HR between group D-0.6 and groups D-1, after study drug administration at 2nd min, 5th min & 8th min and before induction and after induction. The changes in mean HR were comparable in both the groups and statistically not significant, after intubation at 1st min 5th min and 10th min (Fig. 1).

The basal mean SBP were comparable in both the groups. In group D-1, there is statistically significant decrease in mean SBP after drug administration at 2nd min only, when compared to groupD-0.6. statistical evaluation shows no significance in the mean SBP values at 5th min and 8th min after drug administration and before and after induction and also at various intervals after intubation between the two groups (Fig. 2).

The basal mean DBP were comparable in both the groups. In group D-1, there is statistically significant decrease in mean DBP after drug administration at 2nd min only, when compared to group D-0.6. statistical evaluation shows no significance in the mean DBP values at 5th min and 8th min after drug administration and before and after induction and also at various intervals after intubation between the two groups (Fig. 3).

The basal mean MAP was comparable in both the groups. In group D-1, there is statistically significant decrease in mean MAP after drug administration at 2nd min only, when compared to groupD-0.6.

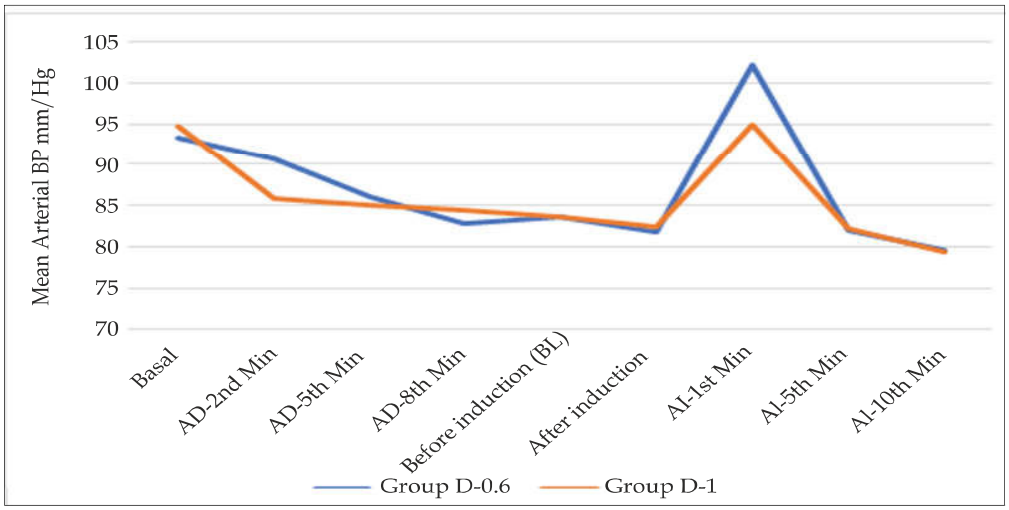

Fig. 4 Comparison of Mean Arterial pressure changes (mmHg) changes in response to laryngoscopy and intubation between two Groups

Statistical evaluation shows no significance in the mean MAP values at 5th min and 8th min after drug administration and before and after induction and also at various intervals after intubation between the two groups (Fig. 4).

Statistical evaluation between the groups showed increased incidence of sedation in groupD-1 than in droupD-0.6 which statistically highly significant .Recovery time is similar in two groups. There is no statistical significance between the two groups with respect to recovery time (Table 2).

Table 2: Showing the statistical singnificant in GroupD-1 compare to GroupD-0.6 in different variables

Variables	GroupD-0.6	GroupD-1	*p*-Value
SBP 2nd min	124.66 ± 13.7	115.7 ± 10.06	0.0006 (HS)
DBP 2nd min	77.10 ± 8.19	72.03 ± 10.41	0.041 (S)
MAP 2nd min	90.70 ± 6.48	85.9 ± 9.55	0.027(S)
MHR AD-2nd min	80.46 ± 12.1	69.26 ± 9.41	0.000 (HS)
MHR AD-5th min	78.30 ± 11.8	68.36 ± 11.08	0.0001 (HS)
MHR AD-8th min	75.50 ± 11.5	67.33 ± 10.54	0.006 (HS)
Before Induction	75.60 ± 10.4	68.76 ± 10.29	0.0014 (S)
After Induction	83.16 ± 13.1	73.66 ± 12.5	0.006 (HS)
Sedation score	2.13 ± 0.43	2.63 ± 0.18	0.0001
Recovery time	2.82 ± 0.3	3.01 ± .02	0.30 (NS)

In group D-0.6, one patient had bradycardia which was treated. In group D-1 bradycardias who were treated (Table 3).

Table 3: Showing the side effects between two groups

	Nil	Bradycardia	Hypotension	Treatment required
GroupD-0.6	49	0	0	1
GroupD-1	45	3	2	5
p value	0.10 (NS)			

Discussion

Dexmedetomidine has been found by various authorsto blunt the haemodynamic response for laryngoscopy and intubation. Dexmedetomidine has been recently introduced in India (only in 2009). Not many studies have been done to know the effectiveness of Dexmedetomidine in attenuating the haemodynamic response to laryngoscopy and intubation in India. Hence it has been selected as our study drug.

Various authors have employed IV Dexmedetomidine for blunting haemodynamic responsestolaryngoscopyandintubationindifferent doses. Different doses of Dexmedetomidine have been used to find the effectiveness for blunting haemodynamic responses to laryngoscopy and intubation, with conflicting results. It has been used in the doses of 0.3 µg/kg, 0.4 µg/kg, 05 µg/kg, 0.6 µg/kg and 1 µg/kg body weight, 0.3 µg/kg to 0.5 µg/kg body weight does was not very effective in blunting the response.Both 0.6 µg/kg and 1 µg/kghave been found to be effective.[3] It is not yet found which one of this dose is effective with minimal side effects. Hence, in our study these two

doses of Dexmedetomidine have been compared to know the minimum effective does of the drug for this purpose with least side effects.

In the present study dexmedetomidine was diluted in 10 ml of normal saline and given intravenously over 10 minutes using syringe pump. Rapid administration of bolus dose of dexmedetomidine, initially results in transient increase in blood pressure and reflex decrease in HR. The initial reaction is due to peripheral α-2B adrenoceoptors stimulation of vascular smooth muscle and can be attenuated by a slow infusion over 10 minutes. Hence in our study we administered the bolus dose over 10 minutes.The administration of the test drugs over 10 minutes in our study, is similar to the studies conducted by Mowafi et al.[2], Basar et al.[4] and Kunisawa et al.[5] and Jarineshin H.[6]

From the pharmacokinetic profile, it is seen that the distribution half life of intravenous dexmedetomidine is approximately 6 minutes. Various authors Aho et al.[7], Scheinin et al.[8], Jakola et al.[9], Mowafi et al.[2] and Keniya et al.[10] have administered dexmedetomidine 10 minutes before induction. Hence, in the present study dexmedetomidine was administered 10 minutes before induction to blunt the haemodynamic response to laryngoscopy and intubation.

In our study, it was observed that there was a statistically highly significant decrease in the mean HR after the administration of 0.6 µg/kg body weight and 1 µg/kg body weight of Dexmedetomidine before induction which is similar to the findings of Scheinin et al.[8], Jaakola et al.[9], Basar et al.[4], Keniya et al.[10] and Chirag Patel et al.[4]

Compared to, group D-0.6, it was observed that there is a statistically highly significant decrease in mean HR in group D-1. The same thing has also been observed in the studies conducted by Martina Aho et al.[7] and Sagiroglu et al.[11] who have found that higher doses of Dexmedetomidine produces more decrease in the HR.

After induction of anaesthesia, compared to pre induction values, it was found that HR increased by nearly 12 bpm in the control group. In group D-0.6 there is a increase in HR Of 8 bump and in group D-1 there is a increase in HR of 5 bpm which is statistically highly significant. In all the 3 groups there is an increase in the HR after the administration on thiopentone. This is in accordance to the property of Dexmedetomidine; the baroreceptor activity is being well preserved.

In the present study, following laryngoscopy and intubation at 1 minute, the mean HR increased by 36 bpm in the control group whereas in group D-0.6 the mean HR increased by only 4 bpm and in group D-1 the mean HR increased by only 5 bpm which is statistically highly significant (p = 0.000) when compared to control group. But the mean change in HR after intubation at various intervals in between group D-0.6 and group D-1 was not statistically significant. Various authors have found similar response to IV dexmedetomidine at 1 min after intubation.

Aho et al.[7] noted that following laryngoscopy and intubation HR at 1 minute increased by 35 bpm in control group and by 15 bpm in 0.6 µg/kg dexmedetomidine group which was statistically significant and compares with our study. The 15 bpm increase in their study is higher than the group D-0.6 in our study (4 bpm). This is probably because in their study all the patients were pre-treated with glycopyrolate.

The increase in mean heart rise in control group sustained even at 5th minute and was 23 bpm whereas in group D-0.6 and group D-1 there is a decrease in HR by 3 and 4 bpm respectively which is statistically highly significant (p = 0.000). Similar observations were made by Scheinin et al.[8] and Jakola et al.[9] with both control and with Dexmedetomidine – 0.6 µg/kg. In the study done by Sagiroglu AE et al.[11], the decrease in HR at 5th min with 1 µg/kg Dexmedetomidine was 18 bpm which his higher than our study. This is probably because all the patients in our study were preloaded with 500 ml of Ringer Lactate which was not done in the study mentioned above.

At 10th minute in our study even at 10th minute, there was increase in HR by 13 bpm in control group compared to decrease in the HR by 4 bpm in both group D-0.6 and group – 1 which was statistically highly significant (p = 0.000). Our study compares with the studies done by Basar et.al. 4 and Chirag Patel et al.[3], who also observed a decrease of 5 bpm and 12 bpm at the end of 10th min. In our study, compared to 0.6 µg/kg body weight there was no significant difference in the mean HR at 1st min, 5th min and 10th min after intubation with 1 µg/kg body weight. Both were equally effective in obtunding the HR response. Same thing has also been observed by sagiroglu AE et al.[11]

Changes in Systolic Blood Pressure (SBP) after Dexmedetomidine Administration

After administration of Dexmedetomidine, there is a gradual reduction in blood pressure till induction in both group D-0.6 and group D-1, which was statistically highly significant. Aho et al.[7] and Keniya et al.[10] found a continuous gradual reduction of SBP as in our study. There was no reduction in SBP in control group till induction which was statistically not significant.

After induction there was a reduction of 10 mmHg of SBP in group D-0.6 and reduction of 22 mmHg in group D-1 and 10 mmHg in control group compared to basal value which is statistically highly significant. Similar observations were made by Kunisawa et al[12] where in there was a decrease in SBP by 12 mmHg in Dexmedetomidine group.

In our study, it is seen that there is highly significant fall in the SBP in group D-0.6 and group D-1 at 1st min 5th min and 10th min following laryngoscopy and intubation compared to control group (p = 0.000) wherein there was an increase of SBP of 29 mmHg, 11 mmHg and 1 mmHg at 1st min, 5th Min and 10 min following laryngoscopy and intubation respectively. Studies done by Scheinin et al.[11], Jaakola et al.[9] and Keniya et al.[10]found similar results that compares with our study. Comparing the SBP at various time intervals between group D-0.6 and group D-1, there was no statistical significant difference. This is consistent with the studies conducted by Sagiroglu et al.[11]

Changes in Diastolic Blood Pressure (DBP) after dexmedetomidine administration there is a gradual decrease of DBP after drug administration at 2nd min, 5th min and 8th min, till induction in both group D-0.6 and group D-1, which is statistically significant. In control group there is not much of variation in DBP till induction. Similar observations were also found by Aho et al.[7], Kunisawa et al.[5] and Keniya et al.[10] where there was a decrease in DBP in dexmedetomidine group and no change in control group.

After Induction in the present study, there was a reduction of 3 mmHg in the control group and 7 mmHg in group D-0.6 and 6 mmHg in group D-1 compared to basal value. Jakola et al.[9] found a decrease in DBP by 3 mmHg in control group and 15 mmHg in Dexmedetomidine group which compares with the present study.

After Laryngoscopy and Intubation in our study there is an increase of DBP by 21 mmHg in control group which gradually decreased to near basal values by 10th minute. In group D-0.6 and group D-1, there is an increase in DBP at 1st min by 8 mmHg and 5 mmHg respectively. However there is a decrease in DBP by 9mmHg and 11 mmHg at 5th min and 10th min in group D-0.6 and decrease in DBP by 8mmHg and 14 mmHg at 5th min and 10th min in group D-1 compared to basal values which is statistically highly significant. Jakola et al.[9] Kunisawa et al.[12] noted similar observations as in our study. In our study, comparing the DBP at various time intervals after laryngoscopy and intubation between group D-0.6 and group D-1, there was no statistical significant difference. This is consistent with the studies conducted by Sagiroglu et al.[11]

After administration of Dexmedetomidine, there is a continuous fall in MAP in both group D-0.6 and group D-1, till induction which is statistically significant. In control group not much of variation was observed in MAP till induction compared to basal values and to Dexmedetomidine group. Basar et al.[4] and Mowafi et al.[2] found which compares with our study.

After induction, there was a reduction in MAP by 12 mmHg in group D-0.6 and 12 mmHg in group D-1 which is statistically significant when compared to group C. Similarly Mowafi et al.[2] observed a decrease in MAP by 13 mmHg in Dexmedetomidine group which concurs with our study.

After Laryngoscopy and Intubation at 1st minute, in group D-0.6, there is an increase of MAP by 9 mmHg. whereas in group D-1, there is an increase in MAP by 1 mmHg compared to the basal values. However at 5th and 10th min the MAP in group D-0.6 was lower by 11 mmHg and 14 mmHg respectively, whereas in group D-1 it was lowered by 12 mmHg and 15 mmHg compared to the basal values which is statistically highly significant, similar to studies done by Basar et al.[4]

In our study, comparing the MAP at various time intervals after laryngoscopy and intubation between group D-0.6 and group D-1, there was no statistical significant difference. This is consistent with the studies conducted by Sagiroglu et al.[11], Talke et al.[13] studied the effect of dexmedetomidine on neuromuscular blockade and noted that dexmedetomidine increased the plasma concentration of rocuronium significantly ($p < 0.05$). The authors could not find a definitive reason for this effect. They hypothesized tht dexmedetomidine might have influcned the pharmacokinetics of rocuronium by decreasing both renal an hepatic blood flow. Similar observation was made by Ghada Ahmad et al.[14]

In group C, mean sedation score was 2.03. In group D-0.6 and group D-1, mean sedation score was 2.13 and 2.63 respectively which was statistically highly significant (p = 0.000) when compared to group C. Group D-1 patients were more sedated, when compared to group D-0.6 which was statistically highly significant. This is similar to observations done by Aho et al.[7], Yildiz et al.[15]and Chirag et al.[3]

Recovery time is similar in all three groups and statistically not significant. This is similar to observations made by Scheinin et al.[11] and Basar H et al.[4]. In group D-0.6, one patient had bradycardia which was treated with inj.atropnine. In group D-1 three patients had bradycardia and two patients had hypotension who were treated with inj. atropine and inj.mephenteramine. In group C none of the patients had bradycardia or hypotension.

Conclusion

Two different doses of Dexmedetomidine – 0.6 µg/kg body weight and 1 µg/kg body weight diluted in 10 ml saline, given 10 minutes before induction are equally efficacious in obtunding the haemodynamic responses to laryngoscopy and endotracheal intubation.However the incidence of sedation and side effects like hypotension and bradycardia is more with 1 µg/kg body weight of Dexmedetomidine.

We conclude that Dexmedetomidine obtunds the haemodynamic responses to laryngoscopy and endotracheal intubation and 0.6 µg/kg body weight is the ideal dose for the same.

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