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Efficacy of Dexmedetomidine in the Dose of 0.5 ug/kg as a Single Bolus Dose in Attenuating Hemodynamic Response to Laryngoscopy and Tracheal Intubation in Adult Patients

Samrajni Ganguly[1], Suhasini Sonavdekar[2], RL Gogna[3]

[1]Senior Resident, [2]Associate Professor, [3]Professor, Department of Anesthesia, MGM Medical College, Kamothe, Navi Mumbai, Maharashtra 410209, India.

Abstract

Aims and objectives: To evaluate efficacy of dexmedetomidine in the dose of 0.5 ug/kg as a single bolus dose in attenuating hemodynamic response to laryngoscopy and tracheal intubation in adult patients. *Materials and Methods:* We conducted a prospective, randomized, double-blind study, in which 60 patients scheduled for elective surgery under general anesthesia were enrolled for the study. Patients were randomly distributed in two groups (30 in each group). Group D received a bolus dose of 0.5 ug/kg dexmedetomidine group and Group C received 10 ml of normal saline (control group). *Results:* There was significant decrease in heart rate in Group D as compared to Group C from 1 minute after induction till 80 minutes ($p < 0.05$). There was significant decrease in SBP, DBP and MAP in Group D as compared to Group C from laryngoscopy till postextubation ($p < 0.05$). Complications like hypotension, hypertension, bradycardia, tachycardia, agitation and coughing was observed in 0%, 80%, 0%, 83.33%, 23.33% and 40% of Group C patients respectively while it was present in 10%, 0%, 10%, 0%, 0% and 13.33% of Group D patients respectively. *Conclusion:* Single bolus dose of dexmedetomidine 0.5 µg/kg prior to laryngoscopy and endotracheal intubation attenuates the airway reflexes and hemodynamic responses effectively during induction of anesthesia providing smooth intubation and provides adequate sedation and delays the need for analgesia in the postoperative period.

Keywords: Dexmedetomidine; Laryngoscopy; Tracheal intubation.

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Introduction

Laryngoscopy and endotracheal intubation are often employed to secure the airway during general anesthesia. However both laryngoscopy and intubation are noxious stimuli and are associated with stress and hemodynamic responses in the form of laryngosympathetic stimulation which is manifested as hypertension, tachycardia and arrhythmias. These hemodynamic responses are well tolerated in otherwise healthy individuals, but in patients with hypertension, coronary heart disease, cerebrovascular disease and intracranial aneurysm these transient changes can result in potentially deleterious effects like left ventricular failure, pulmonary edema, myocardial ischemia, ventricular dysrhythmias and cerebral hemorrhage.[1]

Corresponding Author: Suhasini Sonavdekar, Professor, Department of Anesthesia, MGM Medical College, Kamothe, Navi Mumbai, Maharashtra 410209, India.

E-mail: suhasinee.dr@gmail.com

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Intravenous dexmedetomidine, a central alpha-2 agonist is being used in anesthesia practice as a premedicant. The advantages of dexmedetomidine as premedicant in anesthesia setting include sedation, analgesia, anxiolysis and improved hemodynamic stability. Because of these beneficial properties it has been found that the minimum alveolar concentration (MAC) of volatile anesthetics also decreases significantly up to 90% and hence decreases the requirement of anesthetics.[2] It has also been found that it can decrease the hemodynamic response to laryngoscopy and intubation.[3, 4]

The hemodynamic effects of dexmedetomidine result from peripheral and central mechanism. Alpha-2-adrenoreceptor agonists show a biphasic, dose-dependent, blood pressure effect at low doses the dominant action of α2-adrenoreceptor agonist activation is a reduction in sympathetic tone, mediated by a reduction of norepinephrine release at the neuroeffector junction, and an inhibition of neurotransmission in sympathetic nerves.[2] The net effect of dexmedetomidine action is a significant reduction in circulating catecholamines with a slight decrease in blood pressure and a modest reduction in heart rate.[5] This prospective, randomized, double blinded study was planned to evaluate the efficacy of dexmedetomidine in the dose of 0.5 ug/kg as a single bolus dose in attenuating hemodynamic response to laryngoscopy and tracheal intubation in adult patients.

Aims and Objectives

1. To evaluate the efficacy of dexmedetomidine in the dose of 0.5 ug/kg as a single bolus dose in attenuating hemodynamic response to laryngoscopy and tracheal intubation in adult patients.
2. To study changes in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial blood pressure associated with laryngoscopy and intubation; and to monitor the hemodynamic and vital parameters at the time of endotracheal intubation and at 5 minutes and 10 minutes after.
3. To study any adverse effects associated, such as perioperative hypotension and bradycardia.

Materials and Methods

After approval by hospital research ethical committee, informed written consent for anesthesia was taken. A complete pre-anesthetic assessment of the patients was done. This included history of any systemic diseases like hypertension, bronchial asthma, cardiac and/or pulmonary disorder, psychiatric disorder, substance abuse and allergy to any drugs. Additionally a thorough general and systemic examination was carried out for each patient enrolled. The study was conducted as a double-blind trial from May 2016 to May 2017 at Mahatma Gandhi Mission Institute of Medical Sciences, Kamothe, Navi Mumbai. Sixty patients scheduled for elective surgery under general anesthesia were randomized into two groups.

Sample size: Sixty patients were enrolled for the study (randomly distributed in two Groups D and C [n=30 in each group]. Group D dexmedetomidine group and Group C control group).

Patient of both sexes with age between 18 and 60 yearshaving ASA physical status class I or II. Patients posted for surgeries under general anesthesiawith weight and height both within 20% of their respective ideal values were included.

Patients with comorbid diseases (cardiac, pulmonary, neurological disease), allergy to the drug to be used. Pregnancy, patients on alpha-2 adrenergic receptor agonist/calcium channel blocker/angiotensin converting enzyme inhibitor/ beta-blocker therapy, patient taking antipsychotic drugs and difficult intubation (patients requiring 3 or more attempts at intubation) were excluded.

Methodology

The patients were randomly divided into two groups as designated above and demographic data was noted. Baseline vital parameters were also noted. Patients were premedicated with Tab. Alprazolam 0.25 mg a night before and Tab. Ranitidine 2 hours prior on the morning of surgery with a sip of water. In the pre-op room, a good intravenous access was secured and vital parameters observed and recorded, which included heart rate (HR), mean arterial blood pressure (MAP), electrocardiogram respiratory rate and pulse oximetry (SpO_2). All patients were administered Ringer's lactate solution. Thereafter the patients were shifted to the operation theatre and all the monitors attached and baseline parameters were recorded. *Dexmedetomidine group (Group 'D'):* received a bolus dose of 0.5 ug/kg diluted in normal saline to 10 ml and injected intravenously slowly over 10 minutes, 10 minutes before induction. *Control group (Group C):* patients received 10 ml of normal saline, 10 min before induction.

The double-blind design of study was ensured by the fact that an anesthesiologist, not further involved in the study, prepared syringes immediately before induction of anesthesia. The syringes were thereafter marked dexmedetomidine/placebo together with the name of the patient. Thus the anesthesiologist responsible for the anesthetic technique was kept unaware of the content of the syringes.

Patients of both the groups were premedicated with 0.004 mg/kg of glycopyrolate, 0.02 mg/kg midozolam and 0.6 mg/kg of pentazocine given intravenously before induction. Alongside this, our study drug dexmedetomidine and placebo were randomly administered to the subjects selected for the study in a double-blinded trial as stated above.

Induction of anesthesia was carried out with Inj. Propofol in a dose sufficient to abolish eyelash reflex followed by 0.1 mg/kg of Inj.Vecuronium Bromide to provide neuromuscular blockade. The patient was preoxygenated with 100% O_2 for the next 3 mins. Thereafter, laryngoscopy was performed with an adequate size Macintosh blade and intubation was done with a cuffed endotracheal tube of appropriate size with a strict and vigil monitoring of hemodynamic and respiratory parameters at regular intervals of 1 minute for the first 5 minutes and thereafter at 5 minute intervals till the completion of surgery. Response to skin incision was also observed and recorded in a similar manner. During surgery, anesthesia was maintained with isoflurane and 70% nitrous oxide in oxygen. At the end of the surgical procedure, residual neuromuscular blockade was reversed with Inj.Neostigmine 0.05 mg/kg mg and Inj.Glycopyrolate 0.008 mg/kg intravenously (IV). Extubation was carried out routinely.

Results

Both the groups were comparable in terms of age, weight, height, gender ratio and ASA physical status.

At baseline the mean heart rate among the two groups was comparable (p = 0.402). There was significant decrease in heart rate in Group D as compared to Group C from 1 minute after induction till 80 minutes ($p < 0.05$). After that mean heart rate among the two groups was comparable ($p > 0.05$) (Table 1).

At baseline the mean SBP among the two groups was comparable (p = 0.593). There was significant decrease in SBP in Group D as compared to Group C from laryngoscopy till post-extubation ($p < 0.05$) (Table 2).

At baseline the mean DBP among the two groups was comparable (p = 0.402). There was significant decrease in DBP in Group D as compared to Group C from laryngoscopy till 90 minutes ($p < 0.05$) (Table 3).

Table 1: Comparison of Mean Heart Rate between Different Study Groups at Various Time Intervals

Heart rate	Group C		Group D		*p* value
	Mean	SD	Mean	SD	
Baseline	82.93	5.589	82.73	5.420	0.402
Before induction	83.13	5.244	80.07	4.741	0.412
1 min after induction	79.40	6.891	78.33	4.302	0.043
At laryngoscopy	103.53	6.642	72.40	3.802	0.004
At intubation	109.93	6.533	71.80	3.253	0.0001
5 min	102.53	5.649	71.73	3.991	0.0001
At skin incision	100.87	5.987	69.93	3.342	0.023
10 min	99.53	4.455	71.33	3.689	0.0001
20 min	96.33	6.707	72.93	3.393	0.016
30 min	96.60	6.790	71.33	3.536	0.0001
40 min	92.60	5.468	72.00	3.523	0.0001
50 min	88.13	5.557	73.67	3.763	0.006
60 min	86.73	4.770	72.07	3.084	0.0001
70 min	84.40	5.076	68.73	3.513	0.0001
80 min	83.47	5.029	69.93	3.393	0.002
90 min	82.07	4.017	69.13	3.137	0.256
120 min	81.80	4.156	76.07	3.473	0.085
Post-extubation	98.07	2.149	74.60	3.729	0.500

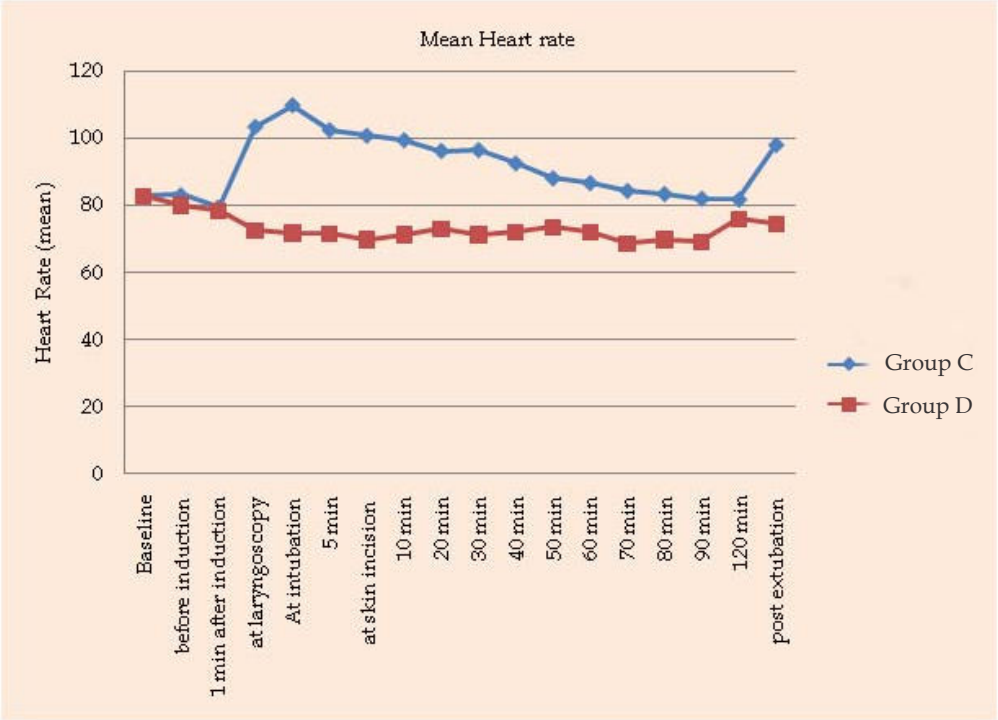

Fig. 1: Comparison of mean heart rate between different study group at various time intervals.

Table 2: Comparison of Mean SBP between Different Study Group at Various Time Interval.

SBP	Group C		Group D		*p* value
	Mean	SD	Mean	SD	
Baseline	129.93	11.356	128.40	10.705	0.593
Before induction	123.47	10.342	122.87	10.170	0.822
1 Min after induction	107.20	9.535	106.73	4.813	0.081
At laryngoscopy	156.60	10.036	99.93	6.741	0.0001
At intubation	145.53	9.864	100.47	5.600	0.0001
5 Min	134.07	7.565	103.47	3.919	0.0001
At skin incision	133.33	7.884	101.80	4.475	0.0001
10 min	132.20	9.445	102.20	3.253	0.0001
20 min	128.60	8.520	98.73	4.472	0.0001
30 min	126.20	8.735	96.13	5.198	0.0001
40 min	129.53	7.820	97.20	4.859	0.0001
50 min	132.53	7.873	100.07	5.139	0.0001
60 min	126.67	9.238	96.40	4.673	0.0001
70 min	128.87	8.431	98.67	2.832	0.0001
80 min	130.40	8.728	99.47	4.539	0.0001
90 min	132.33	8.023	105.27	4.690	0.0001
120 min	131.60	9.250	108.27	6.787	0.0001
Post-extubation	155.00	8.383	111.53	6.932	0.0001

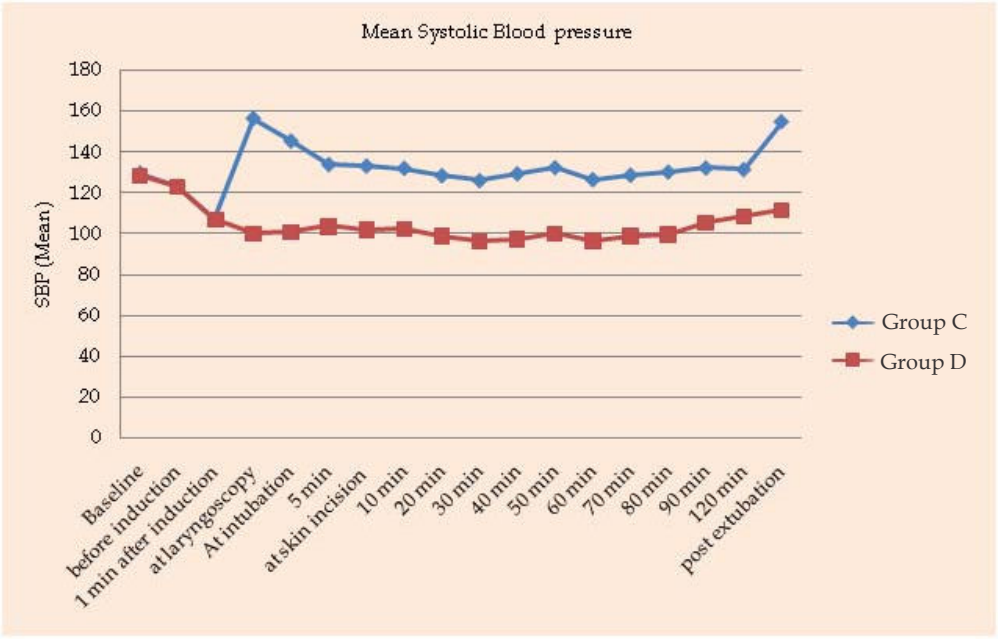

Fig. 2: Comparison of mean SBP between different study group at various time interval

Table 3: Comparison of Mean DBP between Different Study Groups at Various Time Interval.

DBP	Group C		Group D		*p* value
	Mean	SD	Mean	SD	
Baseline	85.50	5.283	84.90	5.307	0.662
Before induction	85.20	5.499	84.20	5.467	0.483
1 min after induction	85.13	5.425	83.13	6.425	0.536
At laryngoscopy	94.67	5.762	78.67	6.127	0.0001
At intubation	95.47	5.380	76.60	5.315	0.0001
5 min	94.40	5.103	74.07	4.806	0.0001
At skin incision	93.80	5.762	73.73	4.417	0.0001
10 min	94.87	5.987	70.93	4.660	0.0001
20 min	85.33	5.287	78.73	4.653	0.0001
30 min	84.33	5.827	76.33	4.521	0.0001
40 min	84.67	5.762	78.87	4.133	0.0001
50 min	86.67	5.511	79.93	4.315	0.0001
60 min	84.87	5.865	78.33	3.871	0.0001
70 min	85.33	5.287	84.67	5.020	0.001
80 min	86.00	5.452	85.60	5.315	0.018
90 min	84.07	5.953	83.93	4.891	0.030
120 min	83.00	5.960	82.47	6.361	0.739
Post-extubation	84.13	5.198	83.87	5.063	0.841

At baseline the mean MAP among the two groups was comparable (p = 0.294). There was significant decrease in MAP in Group D as compared to Group C from laryngoscopy till post-extubation ($p < 0.05$) (Table 4).

Complications like hypotension, hypertension, bradycardia, tachycardia, agitation and coughing was observed in 0%, 80%, 0%, 83.33%, 23.33% and 40% of Group C patients respectively while it was present in 10%, 0%, 10%, 0%, 0% and 13.33% of Group D patients respectively. There was statistically significant difference between various complications amongst different study groups (Table 5).

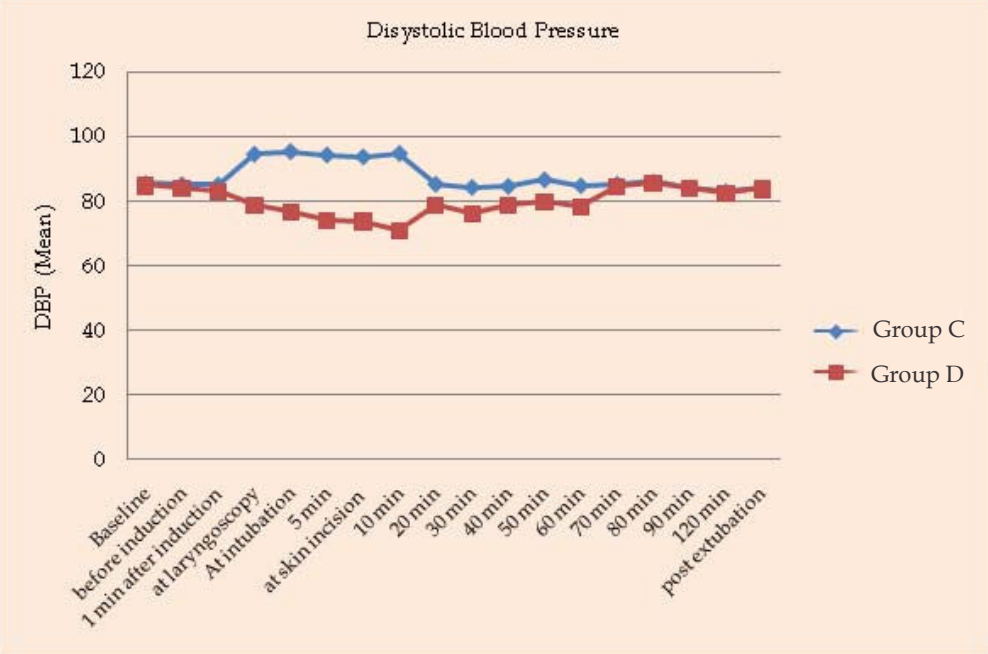

Fig. 3: Comparison of mean DBP between different study group at various time interval.

Table 4: Comparison of Mean MAP between Different Study Groups at Various Time Intervals.

MAP	Group C		Group D		*p* value
	Mean	SD	Mean	SD	
Baseline	79.20	3.773	78.20	3.537	0.294
Before induction	77.80	3.773	76.73	3.704	0.274
1 min after induction	74.33	5.492	73.47	5.164	0.531
At laryngoscopy	97.60	5.882	68.73	5.669	0.0001
At intubation	101.80	5.886	67.07	4.996	0.0001
5 min	102.60	4.875	66.27	4.563	0.0001
At skin incision	99.13	5.218	68.07	4.127	0.0001
10 min	97.73	4.510	66.73	4.118	0.0001
20 min	97.37	4.958	65.60	3.802	0.0001
30 min	96.80	5.209	64.93	3.778	0.0001
40 min	98.27	5.644	62.47	3.711	0.0001
50 min	99.40	5.781	66.40	3.793	0.0001
60 min	96.53	5.655	66.53	3.702	0.0001
70 min	98.00	4.136	68.27	4.118	0.0001
80 min	99.53	4.353	71.27	3.982	0.0001
90 min	100.67	5.738	74.73	4.051	0.0001
120 min	103.80	5.886	75.73	5.349	0.0001
Post-extubation	110.80	5.592	76.33	5.365	0.0001

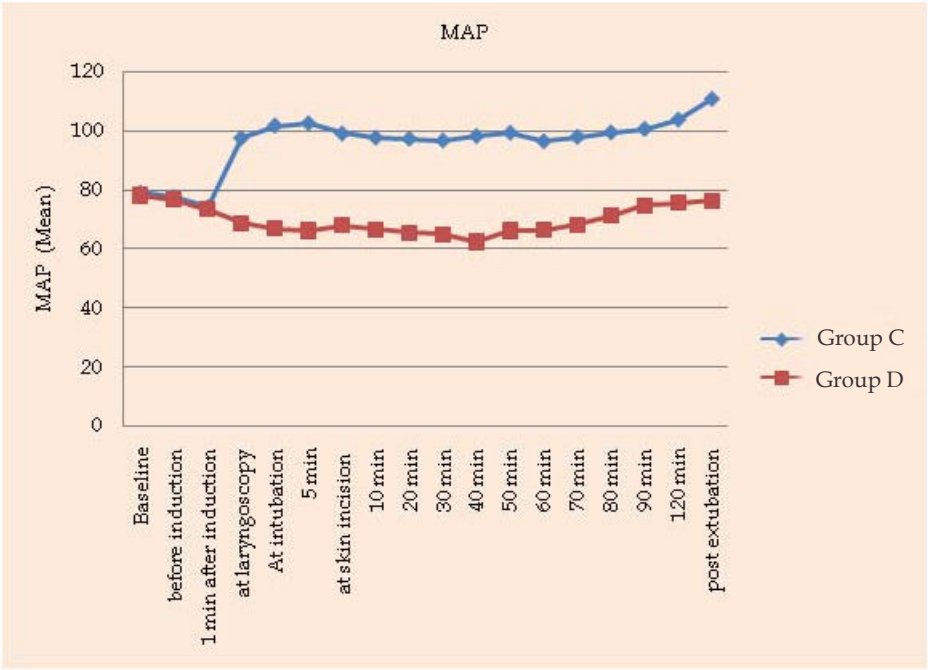

Fig. 4: Comparison of mean MAP between different study group at various time interval.

Table 5: Comparison of Various Complications amongst Different Study Groups.

Complications	Group C	Group D	*p* value
Hypotension	0 (0%)	3 (10%)	0.0001
Hypertension	24 (80%)	0 (0%)	0.0001
Bradycardia	0 (0%)	3 (10%)	0.0001
Tachycardia	25 (83.33%)	0 (0%)	0.0001
Agitation	7 (23.33%)	0 (0%)	0.0001
Coughing	12 (40%)	4 (13.33%)	0.0001

Discussion

Direct vision laryngoscopy for tracheal intubation stimulates the pharyngeal tissues and leads to a hypertensive pressor response due to reflex sympathetic discharge. Though these hemodynamic changes are short lived, yet they may be undesirable in patients with pre-existing myocardial or cerebral insufficiency.

The major stimuli to cardiovascular change during laryngoscopy and tracheal intubation are the forces exerted by the laryngoscope blade on the base of the tongue while lifting the epiglottis.[6] These include a pressor response and tachycardia along with an increase in catecholamine concentrations.[7]

The major part of this sympathoadrenal response is believed to arise from stimulation of supra-glottic region by the laryngoscope blade. Tracheal tube placement and cuff inflation cause minor additional stimulation.[8] It is also known that hemodynamic changes during laryngoscopy can cause unexpected adverse effects like cardiac dysrhythmias, acute surge of systolic blood pressure, left ventricular failure, or even pulmonary edema.

Out of various approaches for attenuation of hemodynamic pressor responses of laryngoscopy, intubation and laparoscopic surgery, pharmacological approach was considered best as it could reduce the heart rate as well as the blood pressure. Many pharmacological methods have been studied for premedication or during induction of anesthesia to attenuate the extent of these hemodynamic events including high doses of opioids, intravenous local anesthetics, beta adrenergic blockers, α_2 adrenergic agonists, magnesium sulphate, and vasodilators like nitroglycerine.

The precise mechanism that leads to the hemodynamic responses to laryngoscopy and intubation involves intense sympathetic discharges and release of catecholamine. Dexmedetomidine promotes sedative and hypnotic effects. The

presynaptic and postsynaptic effects of α_2 adrenoreceptors agonist diminish norepinephrine release and inhibit the central sympathetic outflow.

Heart Rate

In the present study, it was observed that there was a significant decrease in heart rate in all patients after induction and the difference in heart rate changes between the groups was statistically significant (p value <0.001). The primary action of dexmedetomidine on heart is negative chronotropic effect by blocking the cardio-accelerator nerves as well as by augmenting vagal nerve. The decrease in heart rate can be attributed to reflex response for transient hypertension following injection and subsequently due to decrease in central sympathetic outflow.

In the present study, the baseline heart rate at the start of study drug administration was comparable in both groups. At baseline the mean heart rate among the two groups was comparable (p = 0.402). There was significant decrease in heart rate in Group D as compared to Group C from 1 minute after induction till 80 minutes (p < 0.05). After that mean heart rate among the two groups was comparable (p > 0.05).

In the present study, there was a rising trend in heart rate in Group C from baseline value of 82.93 ± 5.5 as compared to Group D in which there was no rise in heart rate but instead the heart rate fell below the baseline 82.73 ± 5.4 following injection of dexmedetomidine and this difference was statistically significant (p value is <0.05). This shows that dexmedetomidine attenuates the sympathetic response to intubation effectively.

It was observed that incidence of tachycardia was 83.33% in Group C vs 0% in Group D. The increase in heart rate in control group was more persistent than dexmedetomidine group. This finding was in agreement with the study done by D. Jain *et al.*[9]

Similarly in the study conducted by D. Jain *et al.*[9], it was observed that, significant decrease in the pulse rate was observed in dexmedetomidine group from 7–10 minutes after the start of dexmedetomidine (p < 0.05), but no intervention was required as this fall in pulse rate was transient and did not affect the blood pressure. The pulse rate in dexmedetomidine group remained below the pre-dexmedetomidine values (baseline value), at all-time intervals following extubation. On the contrary pulse rate rose significantly (p < 0.05) in normal saline (control) group following extubation.[9]

In our study, bradycardia was observed in 3 patients (10%) in Group D which responded to injection atropine. This finding correlates well with the observation by Aksu *et al.*[10]

Similarly in the study conducted by M.L. Jakola *et al.* it was observed that after intubation maximum heart rate was 18% less in dexmedetomidine group compared with placebo. They also noted that there was a significant decrease in blood pressure in dexmedetomidine group.[4]

Systolic Blood Pressure

At baseline the mean SBP among the two groups was comparable (p = 0.593). There was a significant decrease in SBP in Group D as compared to Group C from laryngoscopy till post-intubation (p < 0.05). In the present study the mean systolic blood pressure (SBP) of patients in Group D (dexmedetomidine group) prior to dexmedetomidine injection was 129.93 ± 11.35 mm hg and in Group C (control group) it was 128.40 ± 10.70 mm hg. On inter-group comparison, there was no statistically significant difference in the systolic blood pressure between the two groups prior to injection of the study drug as p = 0.593. At base line the mean SBP among the two groups was comparable (p = 0.593). There was significant decrease in SBP in Group D as compared to Group D from laryngoscopy till post intubation (p < 0.05).

Similarly in the study conducted by Martina Aho *et al.* reported that increase in BP and HR was significantly less in dexmedetomidine group which received 0.6 μg/kg than in the saline group. At the same time they noted that in patients receiving dexmedetomidine 0.3 μg/kg, the increase in HR and BP did not differ from that of the saline group. The major findings of this study were that dexmedetomidine administered before induction at a dose of 0.6 μg/kg blunted the tachycardia response during endotracheal intubation.[11]

This is in agreement with the study conducted by Jain D *et al.* in which study group patients received 1 μg/kg of dexmedetomidine and they did not observe any significant change (p) in the blood pressure in dexmedetomidine group throughout the study period. On the contrary, the systolic blood pressure rose significantly (p < 0.05) in control group following extubation (172.13 ± 17.35) as observed in our study which we achieved with 0.5 μg/kg of dexmedetomidine.[9]

In our study none of the patients in Group D had hypertension as against 80% in control group. (p < 0.001).

Diastolic Blood Pressure

The diastolic blood pressure at the start of study drug injection was taken as baseline for inter-group comparison of patients in each group. In our study, prior to injection the mean diastolic blood pressure (DBP) of patients in Group D was 84.90 ± 5.3 mm Hg and in Group C it was 85.50 ± 5.2 mm Hg there was no statistically significant difference in the diastolic blood pressure between the two groups at the start of study drug injection (p value=0.5967). At base line the mean DBP among the two groups was comparable (p = 0.402). There was significant decrease in DBP in Group D as compared to Group C from laryngoscopy till 90 minutes (p<0.05). Similar findings were observed by Guler G *et al.*, in which diastolic arterial pressure increased significantly during extubation in both dexmedetomidine and control groups, but diastolic blood pressure was significantly lower in dexmedetomidine group than in control group at all times starting from 5 min after drug administration.[12] D. Jain *et al.*, in their study have not studied the changes in the diastolic blood pressure.[9]

MAP (Mean Arterial Pressure)

At baseline the mean map among the two groups was comparable (p = 0.294). There was significant decrease in MAP in Group D as compared to Group C from laryngoscopy till post extubation (p < 0.05). Thus, the MAP values were significantly lower in Group D compared to baseline values at all times from the time of dexmedetomidine infusion to post-extubation 15 minutes. This is in conjunction with the study conducted by Jain *et al.* in which study group patients received 1 µg/kg of dexmedetomidine and they did not observe any significant change (p) in the blood pressure in dexmedetomidine group throughout the study period.[9]

Comparison of adverse effects (complications)

In the present study, various complications like hypotension, hypertension, bradycardia, tachycardia, agitation and coughing was observed in 0%, 80%, 0%, 83.33%, 23.33% and 40% of Group C patients respectively while it was present in 10%, 0%, 10%, 0%, 0% and 13.33% of of Group D patients respectively. There was statistically significant difference between various complications amongst different study groups. Our findings are in agreement with G. Guler *et al.* in which bradycardia occurred in one patient and hypotension in three, within 3 min of dexmedetomidine administration. Atropine was administered for bradycardia, and hypotension was treated within 2 min by giving fluid infusion and reducing inhalation agents; no vasopressor was required. In Group D, no patients developed tachycardia as against in Group C, 26 patients (26.6%) developed tachycardia after extubation, but it reverted back to baseline within 3–5 min. Which was statistically very significant (p < 0.05).[12]

Agitation was observed in 6 patients (20%) in Group C following extubation whereas none of patients were agitated in Group D. this is statistically and clinically significant (p < 0.001). This observation is in conjunction with study done by Guler G *et al.* who conducted a study on the effect of single-dose dexmedetomidine in reducing the agitation and providing smooth extubation after pediatric adenotonsillectomy.[12]

Conclusion

Our study demonstrates that single bolus dose of dexmedetomidine 0.5 µg/kg body weight administered as premedication over 10 minutes, prior to laryngoscopy and endotracheal intubation attenuates the airway reflexes and hemodynamic responses effectively during induction of anesthesia providing smooth intubation and provides adequate sedation, maintaining patient's arousability and delays the need for analgesia in the postoperative period. Hence we recommend the use of dexmedetomidine in patients under GA and further studies are needed in larger population before it can be recommended in patients of CAD, hypertension, cerebral vascular diseases and Neurosurgeries.

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