*Original Article*

# Different doses of Dexmedetomidine in attenuating the pressor response to laryngoscopy in controlled hypertensive patient under general anesthesia

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## Abstract

***Background****: Laryngoscopic manipulation and endotracheal intubation are always a matter of concern which capable of producing tachycardia, arrhythmias and hypertension which is generally well tolerated in healthy patient. In Hypertensive patient cardiovascular response to laryngoscopy and intubation is exaggerated.*

***Aims:*** *To assess the effectiveness in attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation with different doses of intravenous dexmedetomidine in controlled hypertensive patients with no adverse effects.*

***Methods:*** *This prospective Randomized controlled trial was carried out with 60 patients belonging to American Society of Anesthesiologists (ASA) Physical Status II posted for elective general anaesthesia. Patients were randomly divided into three groups with fixed card sampling, where, patients who received IV dexmedetomidine 0.5 µg/kg diluted to 50 ml with normal saline as infusion over 10 min was considered as group A, patients who received IV dexmedetomidine 0.75 µg/kg diluted to 50 ml with normal saline was considered as group B and patients who received IV dexmedetomidine 1 µg/kg diluted to 50 ml with normal saline was considered as group C. The primary outcome measures were haemodynamic response at 1, 3 and 5 min after intubation. The secondary outcome measures were to note down any adverse effects associated with drugs.*

***Result:*** *The groups were well matched for their demographic data . Male to female ratio was 1:1 in all three group. The mean height, weight and BMI were almost similar among three groups. In this study baseline readings of SBP, DBP, MAP and HR were almost similar in all three groups and statistically not significant. Maximum intubation response was seen at 1 min post intubation in all the three groups. The mean SBP of group A varied from 144.8±8.4 mmHg to 118.5±4.4 mmHg that of group B varied from 134.8±4.1 to 122.0±4.2 mmHg and then group C varied from 126.5±15.5 mmHg to 103.8±8.4 mmHg during different evaluation period ($p<0.05$). The mean DBP of group A varied from 91.8±7.6 mmHg to 72.4±5.8 mmHg that of group B varied from 81.3±5.2 to 70.3±2.5 mmHg and then group C varied from 80.9±6.7 mmHg to 63.4±2.4 mmHg during different evaluation period ($p<0.05$). The mean MAP of group A varied from 109.0±5.6 mmHg to 87.5±4.4 mmHg that of group B varied from 98.7±2.5 to 86.3±3.4 mmHg and then group C varied from 95.5±9.2 mmHg to 76.5±3.4 mmHg during different evaluation period ($p<0.05$). The mean heart rate of group A varied from 94.5±12.7 bpm to 75.2±10.5 bpm that of group B varied from 87.3±8.3 to 75.0±6.6 bpm and then group C varied from 81.1±7.2 bpm to 66.2±8.1 bpm during different evaluation period ($p<0.05$).*

***Conclusion:*** *Dexmedetomidine in doses of 0.75 µg/kg was more effective compared to 0.05 µg/kg and 1µg/kg in attenuating haemodynamic response to laryngoscopy and endotracheal intubation without producing adverse effects in control hypertensive patients.*

***Keywords:*** *Efficacy; different doses; dexmedetomidine; haemodynamic response; laryngoscopy; controlled hypertensive patient; randomized control trial.*

## Introduction

The anaesthesiologist is mainly responsible for providing a secure airway for a proper ventilation of the patient during anaesthesia and surgery. No medication and anaesthtic method is reassuring, unless a secure airway is maintained with great efforts. Laryngoscopy and endotracheal intubation is a commonly used measure for the maintenance of a secure airway during general anesthesia and it has specific indications[15].

However both laryngoscopy and intubation are noxious stimuli and are associated with stress responses and haemodynamic responses in the form of laryngo-sympathetic stimulation which is manifested as hypertension, tachycardia and arrhythmias[15]. These haemodynamic responses are well tolerated in otherwise healthy individuals, but in patients with hypertension, coronary heart these transient changes can result in potentially deleterious effects like left ventricular failure, pulmonary edema, myocardial ischemia, ventricular dysrhythmias and cerebral hemorrhage[8].

Dexmedetomidine is effective during intubation and maintained intraoperative cardiovascular stability. These drugs decrease tachycardia, hypertension, and sympathetic activity, which are beneficial for the cases with a presence of myocardial ischemia[2].

Various researchers of different countries have suggested that dexmedetomidine is significantly reduced the haemodynamic responses during laryngoscopy and endotracheal intubation among the hypertensive patients[3]. Choudhury et al[4] and Sulaiman et al[3] have both performed two separate studies by using dexmedetomidine with a dose of 0.05 μg/kg for reduction of blood pressure responses during laryngoscopy and endotracheal intubation. The pretreatment with dexmedetomidine 0.05 μg/kg attenuate the stress responses, but did not totally abolish the cardiovascular and catecholamine surge responses to tracheal intubation. Smitha et al[5] have used dexmedetomidine with the dose of 0.5 and 1μg/kg and have found that the dose of 1μg/kg is more effective for the reduction of stress response to laryngoscopy and endotracheal intubation. However, this is a promising results; furthermore the dose of 1 μg/kg is associated with some incidence of cardiovascular system. Sebastian et al[6] have used dexmedetomidine (0.75 μg/kg) for reduction of stress response to laryngoscopy and endotracheal intubation. In 0.75 μg/kg group, intubation responses is completely obtunded when compared to 0.5 μg/kg without any adverse effects.

Appropriate premedication can prevent the associated risks of heamodynamic pressure response to laryngoscopy and intubation in controlled hypertension patients; which is essential to prevent negative outcomes such as tachycardia, hypertension, myocardial ischemia, ventricular arrhythmias. Dexmedetomidine is highly specific and selective potent alpha-2 agonist which potentially offer a superior effect in attenuating stress-induced sympathoadrenal responses during laryngoscopy. Therefore, the search of effective dose of dexmedetomidine premedication for controlled hypertensive patients uncovering the possibilities for better management of those patient with less side effects in perioperative period and reduce mortality and morbidity.

## Methodology

**Study Population and Settings:** This single blind, parallel randomized controlled trial was conducted in Department of Anaesthesia, Analgesia, Palliative and intensive Care Medicine, Dhaka Medical College Hospital, Dhaka from August 2016 to July 2018 for a period of two (02) years. Data was gathered after approval of protocol by ethical review committee. Patients who were categorized as American society of Anesthesiology (ASA) class II, patients who had a history of essential hypertension for which they were being treated and controlled as well as posted for elective surgeries under general anesthesia were selected as study population. Any anticipated difficult intubation or patients who had a history of bronchial asthma, drug or alcohol abuse, patients who had a history of cerebrovascular, neurologic, respiratory or ischemic heart disease and renal or hepatic dysfunction, patients who were physically dependent on narcotics, known drug allergy to dexmedetomidine, patients on antidepressants, anxiolytics, anticonvulsant or antipsychotics, pregnant or nursing woman, participation in another drug study during the preceding 1 month period were excluded from this study.

**Randomization and Blinding:** A total number of sixty (60) patients belonging to ASA Physical Status II posted for elective general anaesthesia was finally selected. All the information were recorded in a prefixed data sheet. Patients were randomly divided into three groups and each group had twenty patients. Randomization allocated by fixed card sampling. One assigned anesthesiologist performed the grouping. Data were collected by the volunteer anaesthesiologist who was expert enough take data and was fully unaware of the study.

**Intervention:** Group A consisted of twenty (20) patients who were received IV dexmedetomidine 0.5 μg/kg diluted to 50 ml with normal saline as infusion over 10 min. Group B consisted of twenty (20) patients who were received IV dexmedetomidine 0.75 μg/kg diluted to 50 ml with normal saline as infusion over 10 min. Group C consisted of twenty (20) patients who were received IV dexmedetomidine 1 μg/kg diluted to 50 ml with normal saline as infusion over 10 min. All infusions were started 10 minutes prior induction of general anesthesia. All patients were evaluated a day before surgery. The patients were kept fasting overnight after 10:00 pm and was received tablet Ranitidine 150 mg orally and tablet midazolam 7.5 mg orally as premedication at night before surgery. All patients monitored with non-invasive blood pressure monitor (philips sure signs VS3). An IV line was secured, and the patients were administered 500ml of IV fluid Ringer's lactate. Baseline systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP) and heart rate (HR) were measured by one volunteer anaesthesiologists by non-invasive blood pressure monitor and pulse oximetry(phillips sure signs VS3). Then study drug infusion was given over 10 min by principal investigator. All the patients were pre-oxygenated for 3 minutes. Then, patients was induced with IV Thiopental Sodium 5 mg/kg body weight, IV fentanyl 1 μg/kg, endotracheal intubation was facilitated by IV succinylcholine 1.5 mg/kg body weight. Laryngoscopy and intubation was done by principal investigator.

**Follow up and Outcome Measures:** Following laryngoscopy and endotracheal intubation, the parameters recorded was SBP, DBP, MAP and HR at 1, 3 and 5 min after intubation by non-invasive blood pressure monitor. After adequate recovery, patients were shifted to post-anaesthesia care unit and monitored for 2 hours. The principle investigator was assessing the patients directly postoperatively in the recovery room and was also personally follow-up the patients in the ward for monitoring purposes. If any unforeseen complication occurs in the ward, the principle investigator was available to come and examine the patient, address and manage any problems.

**Statistical analysis**: All the parameters were expressed as mean and standard deviation (mean ± SD) and percentage. ANOVA for repeated measures followed by post hoc analysis with Least Significance Difference (LSD) was performed for comparing continuous variables within the groups at different time points. For intragroup comparison at the same time point, between the groups, Students 't' test was applied. $p$ value <0.05 was accepted as level of significance. Statistical analyses were performed by using a computer based statistical program SPSS (Statistical Package for Social Sciences) Version 22 with the help of a biostatistician.

## Result

This present study was carried out with an aim to find out the specific dose of Dexmedetomidine in attenuation of haemodynamic responses to laryngoscopy and tracheal intubation in controlled hypertensive patients without adverse effects. The groups were well matched for their demographic data. Male to female ratio was 1:1 in all three group. The basal readings of SBP, DBP, MAP and HR were similar in all the three groups. Maximum intubation response was seen at 1 min post-intubation in all the three groups. Regarding the side effects it was observed that Hypotension was found in 6(30.0%) and bradycardia was found in 4(20.0%) cases in group C and hypertension was found in 4(20.0%) cases of group A. The difference was statistically significant (p<0.05) among three groups. The mean age were 45.2±3.1 years in group A, 50.3±7.4 years in group B and 47.5±7.9 years in group C. Male was found 10(50.0%) in group A, 10(50.0%) in group B and 10(50.0%) in group C. The difference was statistically not significant (p>0.05) among three groups (Table 1).

**Table I** *Age and Gender Distribution of the Study Participant (n=60)*

| Variables | Group A (n=20) | Group B (n=20) | Group C (n=20) | P value |
|---|---|---|---|---|
| **Mean Age** (Years) | 45.2±3.1 | 50.3±7.4 | 47.5±7.9 | [a]0.053[ns] |
| Range(min-max) | 33 to 43 | 40 to 60 | 35 to 60 | |
| Gender | | | | |
| • Male | 10(50.0%) | 10(50.0%) | 10(50.0%) | [b]1.000[ns] |
| • Female | 10(50.0%) | 10(50.0%) | 10(50.0%) | |

ns= not significant; [a]p value reached from ANOVA test; [b]p value reached from Chi- square test; Data are expressed as Mean±SD

Baseline mean Systolic blood pressure was found 130.2±6.5 (mmHg) in group A, 135.6±4.3 (mmHg) in group B and 134.9±11.9 (mmHg) in group C. The Baseline difference was statistically not significant (p>0.05) among three groups. The mean SBP of group A varied from 144.8±8.4 mmHg to 118.5±4.4 mmHg that of group B varied from 134.8±4.1 to 122.0±4.2 mmHg and then group C varied from 126.5±15.5 mmHg to 103.8±8.4 mmHg during different evaluation period. The difference was statistically significant (p<0.05) among three groups (Table II).

Baseline mean diastolic blood pressure baseline (DBP) was found 79.8±2.2 (mmHg) in group A, 80.6±3.7 (mmHg) in group B and 82.2±4.5 (mmHg) C. The Baseline difference was statistically not significant (p>0.05) among three groups. The mean DBP of group A varied from 91.8±7.6 mmHg to 72.4±5.8 mmHg that of group B varied from 81.3±5.2 to 70.3±2.5 mmHg and then group C varied from 80.9±6.7 mmHg to 63.4±2.4 mmHg during different evaluation period. The difference was statistically significant (p<0.05) among three groups (Table III).

**Table II** *Distribution of the Study Participant by Systolic Blood Pressure (n=60)*

| Systolic blood pressure (mmHg) | Group A (n=20) | Group B (n=20) | Group C (n=20) | P value |
|---|---|---|---|---|
| **Baseline** | 130.2±6.5 | 135.6±4.3 | 134.9±11.9 | 0.086[ns] |
| Range(min-max) | 120 to 140 | 130 to 142 | 127 to 158 | |
| After drug administration | 118.5±4.4 | 129.0±5.5 | 124.5±10.9 | 0.001[s] |
| Range(min-max) | 110 to 122 | 120 to 137 | 110 to 143 | |
| 1 min | 144.8±8.4 | 134.8±4.1 | 126.5±15.5 | 0.001[s] |
| Range(min-max) | 130 to 155 | 130 to 140 | 105 to 145 | |
| 3 min | 128.6±5.8 | 126.3±5.7 | 112.2±8.9 | 0.001[s] |
| Range(min-max) | 118 to 135 | 120 to 135 | 100 to 124 | |
| 5 min | 119.1±8.0 | 122.0±4.2 | 103.8±8.4 | 0.001[s] |
| Range(min-max) | 105 to 128 | 118 to 130 | 90 to 120 | |

s=significant; ns=not significant; p value reached from ANOVA test; Data are expressed as Mean±SD

**Table III** *Distribution of the study participant by diastolic blood pressure (n=60)*

| Diastolic BP (mmHg) | Group A (n=20) | Group B (n=20) | Group C (n=20) | P value |
|---|---|---|---|---|
| Baseline | 79.8±2.2 | 80.6±3.7 | 82.2±4.5 | 0.108[ns] |
| Range(min-max) | 75-82 | 75-87 | 80-92 | |
| After drug administration | 72.4±5.8 | 73.7±4.2 | 78.1±5.7 | 0.003[s] |
| Range(min-max) | 60-80 | 70-82 | 70-86 | |
| 1 min | 91.8±7.6 | 81.3±5.2 | 80.9±6.7 | 0.001[s] |
| Range(min-max) | 79-100 | 78-85 | 71-90 | |
| 3 min | 86.3±6.5 | 75.7±2.9 | 73.1±6.7 | 0.001[s] |
| Range(min-max) | 75-95 | 70-80 | 68-90 | |
| 5 min | 77.8±3.9 | 70.3±2.5 | 63.4±2.4 | 0.001[s] |
| Range(min-max) | 70-82 | 65-75 | 60-68 | |

s=significant; p value reached from ANOVA test; Data are expressed as Mean±SD

Baseline mean MAP was found 96.8±4.1 (mmHg) in group A, 98.3±3.8 (mmHg) in group B and 100.3±7.1 (mmHg) in Group C. The Baseline difference was statistically not significant (p>0.05) among three groups. The mean MAP of group A varied from 109.0±5.6 mmHg to 87.5±4.4 mmHg that of group B varied from 98.7±2.5 to 86.3±3.4 mmHg and then group C varied from 95.5±9.2 mmHg to 76.5±3.4 mmHg during different evaluation period. The difference was statistically significant (p<0.05) among three groups (Table IV).

Baseline mean heart rate was found 83.4±9.6 bpm in Group A, 90.2±13.4 bpm in Group B and 84.9±3.9 bpm in Group C. The baseline difference was statistically not significant (p>0.05) among three groups. The mean heart rate of group A varied from 94.5±12.7 bpm to 75.2±10.5 bpm that of group B varied from 87.3±8.3 to 75.0±6.6 bpm and then group C varied from 81.1±7.2 bpm to 66.2±8.1 bpm during different evaluation period. The difference was statistically significant (p<0.05) among three groups.

**Table IV** *Distribution of the study participant by MAP (n=60)*

| MAP (mmHg) | Group A (n=20) | Group B (n=20) | Group C (n=20) | P value |
|---|---|---|---|---|
| Baseline | 96.8±4.1 | 98.3±3.8 | 100.3±7.1 | 0.113$^{ns}$ |
| Range(min-max) | 91 to 106 | 93 to 105 | 95 to 114 | |
| After drug administration | 87.5±4.4 | 91.9±3.7 | 92.6±7.7 | 0.007$^{s}$ |
| Range(min-max) | 80 to 94 | 88 to 99 | 83 to 104 | |
| 1 min | 109.0±5.6 | 98.7±2.5 | 95.5±9.2 | 0.001$^{s}$ |
| Range(min-max) | 101 to 117 | 96 to 103 | 84 to 108 | |
| 3 min | 99.9±4.3 | 91.8±4.2 | 85.9±5.4 | 0.001$^{s}$ |
| Range(min-max) | 94 to 106 | 85 to 97 | 80 to 96 | |
| 5 min | 91.4±3.4 | 86.3±3.4 | 76.5±3.4 | 0.001$^{s}$ |
| Range(min-max) | 84 to 96 | 80 to 93 | 70 to 82 | |

s=significant; p value reached from ANOVA test; Data are expressed as Mean±SD.

**Table V** *Distribution of the Study Participant by Heart Rate (n=60)*

| Heart Rate (bpm) | Group A (n=20) | Group B (n=20) | Group C (n=20) | P value |
|---|---|---|---|---|
| Baseline | 83.4±9.6 | 90.2±13.4 | 84.9±3.9 | 0.078$^{ns}$ |
| Range(min-max) | 60 to 94 | 80 to 120 | 76 to 90 | |
| After drug administration | 75.2±10.5 | 83.6±12.5 | 73.6±6.7 | 0.006$^{s}$ |
| Range(min-max) | 54 to 90 | 67 to 110 | 65 to 86l | |
| 1 min | 94.5±12.7 | 87.3±8.3 | 81.1±7.2 | 0.006$^{s}$ |
| Range(min-max) | 78 to 120 | 75 to 98 | 70 to 90 | |
| 3 min | 87.4±11.7 | 79.4±5.8 | 75.0±7.8 | 0.002$^{s}$ |
| Range(min-max) | 70 to 112 | 70 to 90 | 60 to 85 | |
| 5 min | 78.7±6.9 | 75.0±6.6 | 66.2±8.1 | 0.001$^{s}$ |
| Range(min-max) | 66 to 90 | 65 to 87 | 52 to 74 | |

s=significant; p value reached from ANOVA test; Data are expressed as Mean±SD
Note: ADA= after drug administration

**Table VI** *Distribution of the study participant by side effects (n=60)*

| Side effects | Group A (n=20) | | Group B (n=20) | | Group C (n=20) | | P value |
|---|---|---|---|---|---|---|---|
| | n | % | n | % | N | % | |
| Hypotension | 0/20 | 0.0 | 0/20 | 0.0 | 6/20 | 30.0 | 0.001[s] |
| Hypertension | 4/16 | 20.0 | 0/20 | 0.0 | 0/20 | 0.0 | 0.014[s] |
| Bradycardia | 0/20 | 0.0 | 0/20 | 0.0 | 4/16 | 20.0 | 0.013[s] |

s= significant, p value reached from chi square test

Table VI shows side effects of the study patients, it was observed that bradycardia had 4(20.0%) in group C. The difference was statistically significant (p<0.05) among three groups.

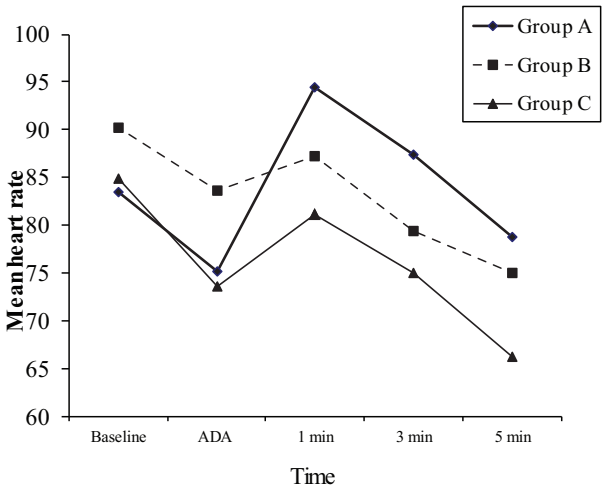


**Figure 1:** *Line graph showing heart rate of the study populations*

## Discussion

In this present study, the mean age was 45.2±3.1 years in group A, 50.3±7.4 years in group B and 47.5±7.9 years in group C. The difference was statistically not significant (p>0.05) among three groups. Samala et al[8], Pramanick et al[9] and Smitha et al[5] found almost similar mean age and age ranged in their respective studies. On the other hand Sebastian et al[6] found the mean age was 32.50±9.12 years in Group A, 36.96±10.33 years in Group B, 31.20±9.30 years in Group C, which is smaller with the present study. The lower mean age and age range maybe due to geographical variations, racial, ethnic differences, and genetic causes.

In this study, dexmedetomidine is effective significantly in blunting the increase in mean SBP and DBP due to laryngoscopy and intubation. However, the baseline difference of mean SBP and DBP is not statistically significant (p>0.05) among the group A, B and C. Though mean SBP and DBP have been increased at 1 minute after intubation in all three groups. The haemodynamic variables fell below the base line in group B and C all the time. The findings indicates that there is a disparity of SBP and DBP in group A and group C but in group B it was almost unswerving from baseline to 5 minutes follow-up. Inter group comparison revealed statically significant among three groups (p<0.05). Smitha et al[5] compared the effect of 0.5 and 1 µg/kg of dexmedetomidine with normal saline in attenuating stress response. They found out that dexmedetomidine 1 µg/kg was more effective than dexmedetomidine 0.5 µg/kg in controlling haemodynamic responses to tracheal intubation. The intergroup comparison revealed a statistically significant (p<0.05). Similar observations regarding the SBP and DBP were also Samala et al[8], Pramanick et al[9] and Sebastian et al[6].

At baseline, the MAP (P=0.113) is almost same among the group A, B and C. However, it has been also found that group B and group C have a significantly lower MAP (p=0.001) to 5 min follow up. Dexmedetomidine attenuates sympathato-adrenal response by activation of presynaptic á2 receptors in sympathetic nerve endings resulting in decreased release of noradrenaline. Moreover, stimulation of postsynaptic á2 receptors of locus coeruleus causes inhibition of norepinephrine release[10]. Patel et al[11] have administered dexmedetomidine intravenously as loading dose of 1 ìg/kg over 10 min prior to induction in group B and observed, dexmedetomidine significantly attenuated stress response to intubation with lesser increase in systolic (6% vs. 23%) and diastolic (7% vs. 20%) blood pressure as compared to the control group (P <0.05).

In this current study, the baseline mean MAP is 96.8±4.1 (mmHg) in group A; however, in group B and group C the MAP are slightly higher that group A which are 98.3±3.8 (mmHg) and 100.3±7.1 (mmHg) respectively. The baseline difference of mean arterial pressure among these three groups is not statistically significant (p>0.05). Maximum intubation response is found at 1 min post-intubation among the three groups. In group B, they approached near the baseline by 3 minutes. Interestingly, the variables fell below the baseline by 3 min in group C. In group A statistically higher values of SBP, MAP at all-time intervals are found in post-intubation when compared to group B and group C. Therefore, it can be inferred that the haemodynamic response is better observed in group B and group C, when it is compared with group A. However, the parameters fell below the baseline value at 1 min after intubation in group C. This clearly indicates that the dexmedetomidine in a dose of 0.75 µg/kg and 1 µg/kg is superior to dexmedetomidine in a dose of 0.5 µg/kg.

Similarly Sebastian et al[6] showed the mean of Mean Arterial Blood Pressure (MAP) in first minute 114.57±5.14 mmHg in Group A, 98.87±5.86 mmHg in Group B and 96.33±5.40 mmHg in Group C ($p$<0.001). In third minute 108.47±4.97 mmHg in Group A, 94.83±5.13 mmHg in Group B and 90.27±5.49 mmHg in Group C ($p$<0.001). In fifth minute 103.37±4.51 mmHg in Group A, 91.80±5.48 mmHg in Group B and 85.47±5.08 mmHg in Group C($p$<0.001). Similar observations regarding the MAP pressure was also reported by Smitha et al[5].

At baseline, the HR (P=0.078) was almost same among three groups. However, it was found that, group B and group C had a significantly lower HR just after intubation (P=0.006) to 5 min follow up. The heart rate showed that there is discrimination in group A and group C but in group B it was almost consistent from baseline to 5 minutes follow-up. Dexmedetomidine attenuates sympathoadrenal response by activation of presynaptic á2 receptors in sympathetic nerve endings resulting in decreased release of noradrenaline. Moreover, stimulation of postsynaptic á2 receptors of locus coeruleus causes inhibition of norepinephrine release[3]. Patel et al[11], administered dexmedetomidine intravenously as loading dose of 1 ìg/kg over 10 min prior to induction in group B and observed, dexmedetomidine significantly attenuated stress response to intubation with lesser increase in heart rate (10% vs. 17%).

The group A had statistically higher values of HR at all-time intervals post-intubation when compared to Group B and Group C. Hence, it can be inferred that the haemodynamic response was better obtained in Group B and Group C, when compared with Group A. In Group C, the parameters fell below the baseline value at 1 min after intubation. This indicates that dexmedetomidine in a dose of 0.75 µg/kg and 1 µg/kg was superior to dexmedetomidine in a dose of 0.5 µg/kg.

In this present study, hypotension was found in 6(30.0%) and bradycardia was found 4(20.0%) in group C. In group A hypertension has found in 4(20.0%) cases. The differences among the three groups is statistically significant (p<0.05). Smitha et al[5] have reported that different doses of dexmedetomidine has shown irregular breathing with varied episodes of apnoea. Furthermore, dexmedetomidine (1 µg/kg) has been associated with the increased incidence of adverse effects like bradycardia and hypotension observed by Kartik et al[12] and Menda et al[13]. It has been established that the activation of post-synaptic á-2 receptors in CNS brings the decreased sympathetic activity which can lead to bradycardia as well as hypotension[14]. Furthermore dexmedetomidine (1 ìg/kg) is associated with increased incidence of adverse effects[13].

Yallapragada et al[15] have been reported that the dexmedetomidine (1 µg/Kg) is effective on the blood pressure responses during laryngoscopy and intubation showed that BP increased by 4.0% initially but later declined by 11.0% following a 5 minute infusion of dexmedetomidine. After intubation the SBP rose only slightly above baseline. In four patients in dexmedetomidine Group hypotension (SBP <90 mmHg) was observed following induction of anaesthesia. In this study also six patients had developed hypotension in group C.

Patients have been shifted to the post anaesthesia care unit after complete clinical recovery and they have been observed for 2 hours for nausea, vomiting, bradycardia, hypotension and sedation.

In present study results suggested that to control blood pressure during laryngoscopy and tracheal intubation, Dexmedetomidine is a better drug and 0.75 ìg/kg dose is more effective than Dexmedetomidine with the dose of 0.5 ìg/kg and 1 ìg/kg which causes no significant side effects in controlled hypertensive patients. In this study no significant respiratory depression, apnea, muscle rigidity or decrease in $SpO_2$ was seen in any patient in post-operative period.

**Conclusion**

Under the condition of present study it can be concluded that Dexmedetomidine dose of 0.75 µg/kg is safe and effective in obtunding the blood pressure response to laryngoscopy and endotracheal intubation without producing side effects compared to doses of 0.5 µg/kg and 1 µg/kg

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