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# Efficacy of intravenous Dexmedetomidine in attenuation of hemodynamic response to laryngoscopy and endotracheal intubation

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## ABSTRACT

**Introduction:** The process of laryngoscopy and endotracheal intubation are noxious stimuli and is associated with intense sympathetic activity which may precipitate intra-operative complications. **Aim:**To study the efficacy of intravenous dexmedetomidine in attenuation of hemodynamic response to laryngoscopy and endotracheal intubation. **Materials and Methods:** This was a prospective study conducted in a group of hundred patients. The study population was divided randomly into two groups.Group C – Control group (n=50) – received 10 ml of normal saline intravenously over 10 minutes, 10 minutes before induction and Group D – (n=50) – received Dexmedetomidine in the dose of 0.6 µg/kg body weight diluted in 10 ml of normal saline. **Results:** There was marked decrease in HR 10 minutes after Dexmedetomidine administration. 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 all times which was statistically significant. There was reduced thiopentone requirement and no increased sedation scoring in patients belonging to Dexmedetomidine group compared to control group. There were minimal side effects noted in the Dexmedetomidine group which were managed easily. **Conclusion:** Dexmedetomidine at a dose of 0.6µ/kg body weight given 10 minutes before induction significantly attenuated the hemodynamic response to laryngoscopy and tracheal intubation and there was reduction in dose requirement of thiopentone sodium without significant side effects

**Key words:** Laryngoscopy and intubation, General anesthesia, Hemodynamic response, Dexmedetomidine, Reduced thiopentone sodium requirement

## Introduction

The process of laryngoscopy and endotracheal intubation are noxious stimuli and therefore constitute a period of extreme hemodynamic stress and is associated with intense sympathetic activity marked by tachycardia and hypertension[1]. These changes are usually transient and insignificant in healthy individuals but may be hazardous to those with cardiovascular or cerebrovascular diseases[2].Pressor response may result in intra-operative myocardial infarction, acute left ventricular failure, intracranial bleed[3] and dysrhythmias[4].Intravenous anesthetic induction agents do not adequately or predictably suppress the circulatory responses induced by endotracheal intubation[5]. So, prior to initiating laryngoscopy, additional pharmacological measures are taken. Various studies have found that Dexmedetomidine can decrease the hemodynamic response to laryngoscopy and intubation[6]. The present study is aimed at attenuation of hemodynamic response to laryngoscopy and endotracheal intubation with Dexmedetomidine at 0.6µg/kg body weight.We study the efficacy of intravenous dexmedetomidine in attenuation of hemodynamic response to laryngoscopy and endotracheal intubation.

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## Materials and methods

This study was a Prospective Randomized Double Blind Controlled Clinical study conducted at Prathima Institute of Medical Sciences, Karimnagar over a

period of one year. The study group consisted of hundred patients. Informed consent was taken from all the patients.

**Inclusion criteria:** Adult patients aged between 18 and 50 years of both sex, posted for elective surgeries under general anesthesia and belonging to ASA physical status I and II, and Mallampatti grade I and II.

**Exclusion criteria:**Patients with hypertension, cardiac, coronary, renal, hepatic, cerebral diseases, peripheral vascular diseases and endocrinal diseases, heart rate less than 60 bpm, systolic blood pressure less than 100 mm of Hg, presence of heart block, difficult airway and obese patients (BMI>30).

**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 was asked to pick up the envelope. The envelope was opened by senior anesthesiologist 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 0.6µg/kg body weight diluted to 10 ml normal saline intravenously over 10 min, 10 minutes prior to induction using syringe pump.

**Preoperative period**: Investigations done were: complete blood picture, complete urine examination, electrocardiogram, X-ray chest, blood sugar, blood urea, serum creatinine. Pre-anesthetic evaluation was done in the evening before the day of surgery.

**Intra operative period:** The patients were connected to multiparameter monitor. The baseline systolic, diastolic blood pressure, mean arterial pressure and heart rate were recorded. The cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II.

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 0.6µg/kg body weight diluted in 10 ml normal saline intravenously over 10 min, 10 min before induction using syringe pump and in control group only normal saline 10 ml was given. The study drug was prepared by the senior anesthesiologist who was not involved with the study and observer as well as patient were blinded for the study. 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. Ninety seconds before intubation all patients received IV Lignocaine 1.5mg/kg body weight. Patient was induced with injection Thiopentone sodium 5 mg/ kg body weight, as a 2.5% solution till loss of eye lash reflex occurred and dose of Thiopentone sodium required for loss of eye lash reflex recorded. Endotracheal intubation was facilitated with injection Vecuronium 0.1mg/kg.Anesthesia was maintained appropriately. No surgical or any other stimulus was applied during 10 minutes of study period and Vecuronium was the only additional drug given during this 10 minutes 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.

**Monitoring**: The following cardiovascular parameters were recorded in all patients: Heart rate, Systolic blood pressure, Diastolic blood pressure, Mean arterial pressure [MAP] in mm of Hg in the following time interval – Basal before giving study drug, then 2 minutes, 5 minutes, 8 minutes after giving study drug, before induction, after induction, 1 minute, 3 minutes, 5 minutes, and 10 minutes after laryngoscopy and intubation.

**Side effects:** Hypotension was defined as SBP $\leq$ 20% of baseline value.[7] Tachycardia as HR > 25% of baseline value.[8] Bradycardia as HR < 60 beats/ minute.[5] Dysrhythmia as any ventricular or supra ventricular beat or any rhythm other than sinus rhythm.[8] The side effects of the study drug like hypotension, bradycardia and sedation were noted. Sedation scoring was as per Ramsay sedation scale.[9]

## Results

The minimum age in groups C and D were 20 and 18 years respectively. The maximum age in both groups was 55 years. The mean age in group C and D were 36.8±9.7 and 36.42± 9.36 respectively. There was no significant difference in the age of patients between the Group C and Group D. Both groups were similar with respect to age distribution (p=0.835).

**Table 1: Demographic distribution in study**

| Age (years) | Group C (Control) | | Group(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 | |
| Mean age in years±SD | 36.8±9.7 | | 36.42± 9.36 | |
| p-value | 0.835 (NS) | | | |
| Gender distribution | | | | |
| Male | 27 | 54 | 22 | 49 |
| Female | 23 | 56 | 28 | 56 |
| Total | 50 | 100 | 50 | 100 |
| 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 |
| Mean body weight in kg± SD | 56.12±6.15 | | 55.34±7.56 | |
| Minimum body weight in kg | 45 | | 43 | |
| Maximum body weight in kg | 70 | | 73 | |
| p-value | 0.189 (NS) | | | |

**Gender distribution**: In group C there were 27 (54%) males and 23 (56%) females. In group D there were 22 (49%) males and 28 (56%) female patients. Statistically there was no significant change in the gender in both the groups.

**Body weight**: The minimum body weight in groups C and D were 45 kg and 43 kg respectively. The maximum body weight in groups C and D were 70 kg and 73 kg respectively. The mean body weight in Group C was 56.12 ± 6.15 and in Group D it was 55.34 ± 7.56. There was no significant difference in the body weight of patients between the Group C and Group D (p=0.189).

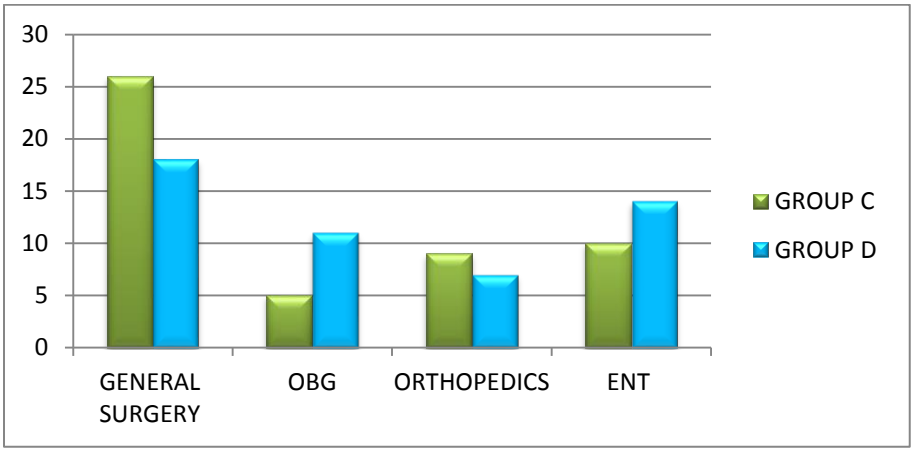



**Fig 1: Type of surgeries done in study**

**Type of surgery**: For group C and group D respectively, there were 26 cases and 18 cases who underwent general surgeries, 5 cases and 11 cases who underwent gynaecological surgeries, 69 cases and 7 cases who underwent orthopaedic surgeries and 10 cases and 14 cases who underwent ENT surgeries. The type of surgeries comprised of excision of lipoma, fibroadenoma, cervical rib, cervical lymph node, thyroglossal cyst excision, exploration of sinus tract, etc.

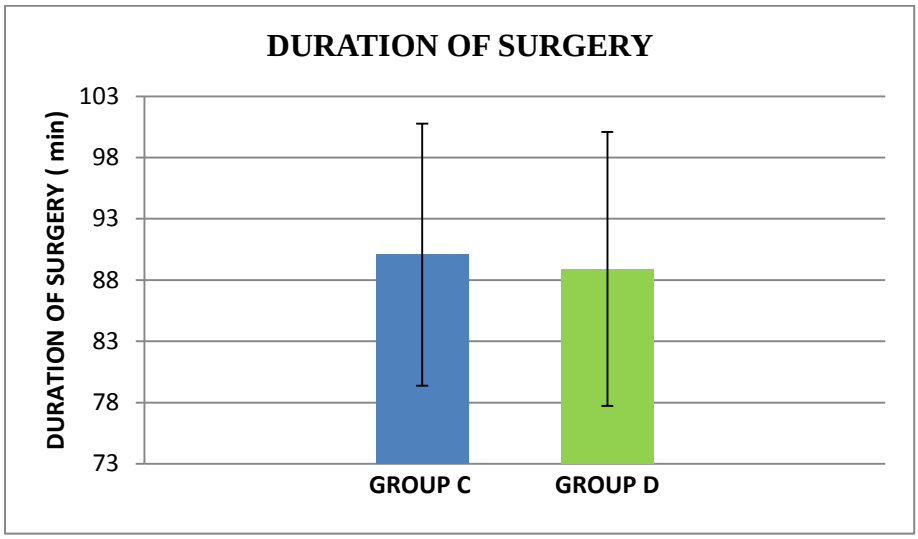
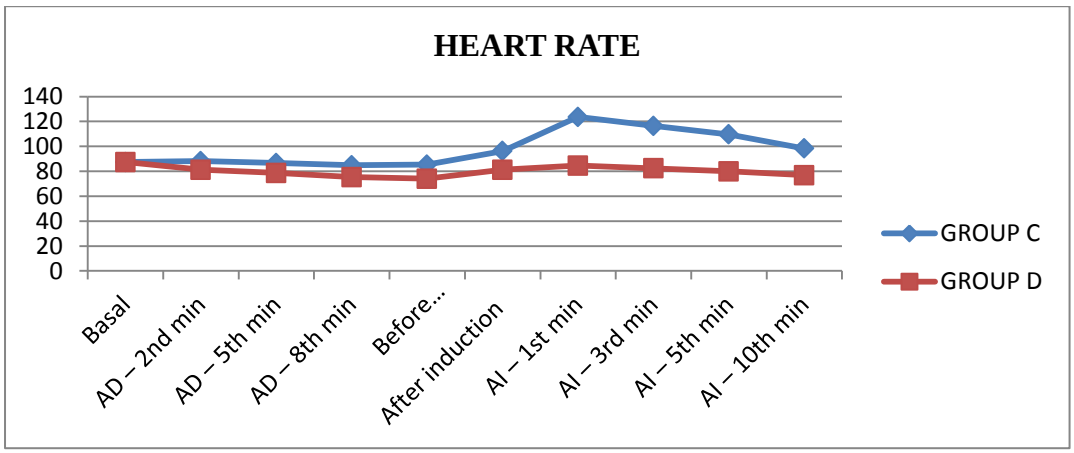



**Fig 2: Mean duration of surgery**

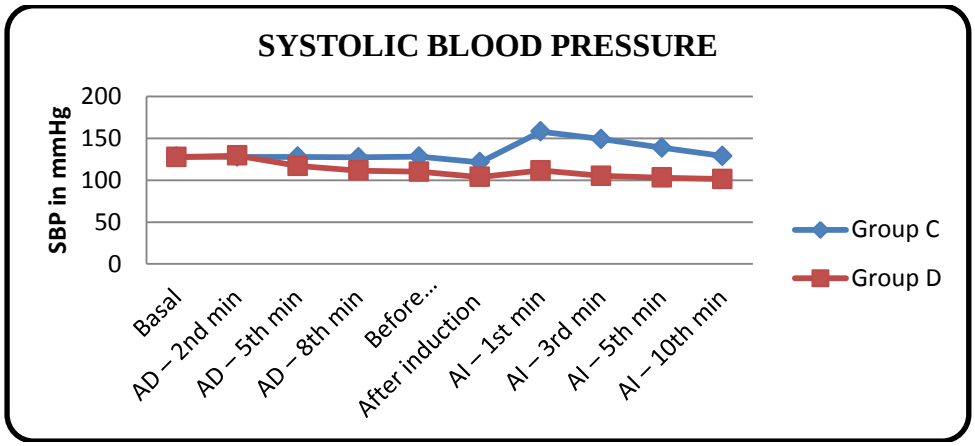
In control group the mean duration of surgery was 90.08±10.7 minutes and in Dexmedetomidine group 88.92±11.8 minutes which was statistically not significant (p=0.078).(Fig 2)




(p<0.01) – Highly significant (HS); (p<0.05) – Significant (S);
(p>0.05) – Not significant (NS); AD-After drug administration; AI- After intubation

**Fig 3: Intergroup comparison of mean heart rate (bpm) changes in response to laryngoscopy and intubation**

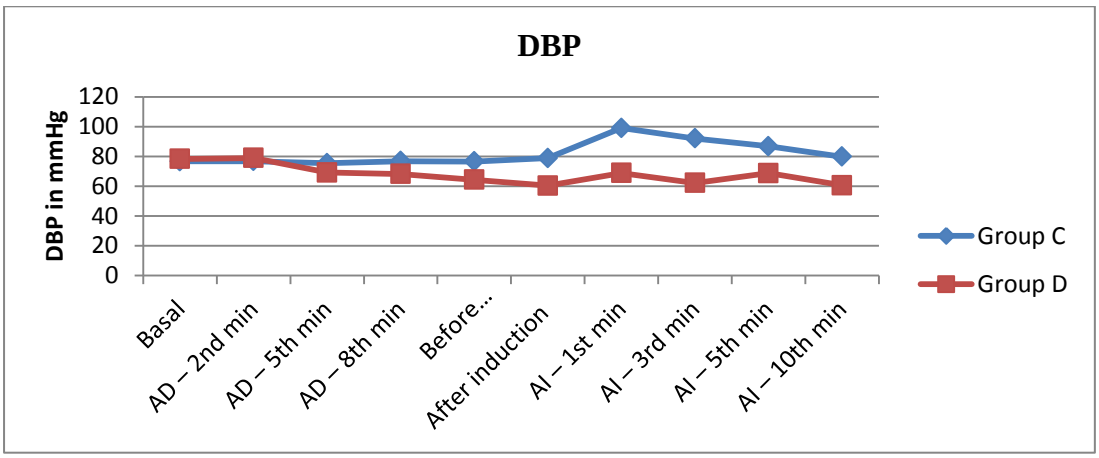
The basal heart rate was comparable in both groups (p=1.000). There was a significant fall in HR in group D at 2, 5 and 8 minutes of drug administration and before and after induction. The mean HR increase observed at 1, 3, 5 and 10 minutes after intubation in group C was statistically highly significant compared to mean HR in group D (p=0.000).(Fig 3)




(p<0.01) – Highly significant (HS); (p<0.05) – Significant (S);
(p>0.05) – Not significant (NS); AD-After drug administration; AI-After intubation

**Fig 4: Intergroup comparison of mean systolic blood pressure (SBP in mmHg) changes in response to laryngoscopy and intubation**

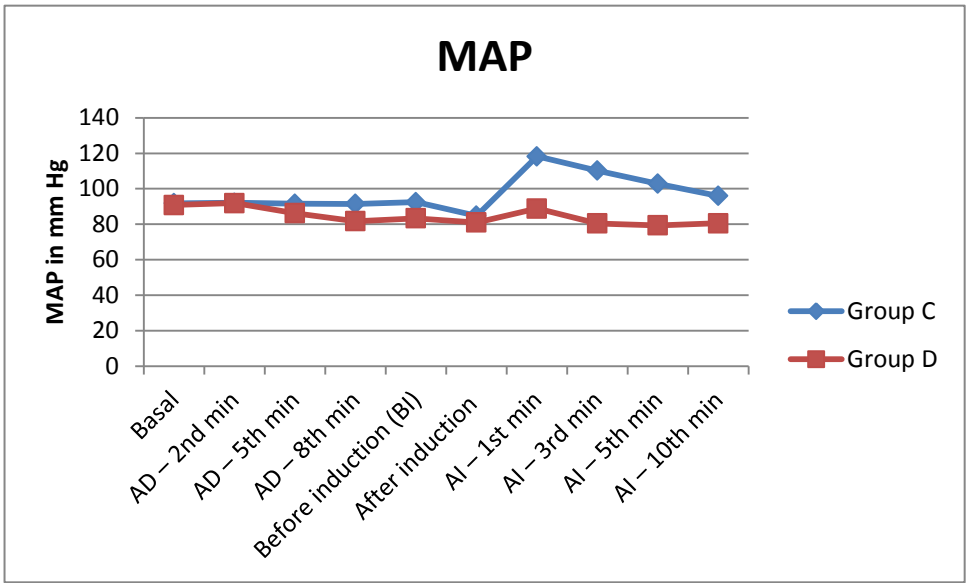
The mean SBP were comparable in both groups (p=0.734). The mean SBP values at 5 and 8 minutes of drug administration, before and after induction were significantly low in group D (p=0.000) compared to group C. The increase in SBP in group C at 1,3, 5 and 10 minutes after intubation was statistically highly significant (p=0.000) compared to group D.(Fig 4)




(p<0.01) – Highly significant (HS); (p<0.05) – Significant (S);
(p>0.05) – Not significant (NS); AD-After drug administration; AI-After intubation

**Fig 5: Intergroup comparison of mean diastolic blood pressure (DBP in mmHg) changes in response to laryngoscopy and intubation**

The mean basal DBP are comparable in both groups (p=0.223). The mean DBP values at 5 and 8 minutes of drug administration, before and after induction were significantly low (p=0.000) 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.(Fig 5)




(p<0.01) – Highly significant (HS); (p<0.05) – Significant (S);
(p>0.05) – Not significant (NS); AD-After drug administration; AI-After intubation

**Fig 6: Intergroup comparison of mean arterial pressure (MAP in mmHg) changes in response to laryngoscopy and intubation**

The mean basal MAP are comparable in both groups (p=1.000). 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 D(Fig 6)

**Table 3: Showing the dose of thiopentone required and sedation score**

| **Mean Dose of thiopentone required for induction(mg)** | |
|---|---|
| **Group C** | 278±34.49 |
| **Group D** | 170.5±30.17 |
| **p-value** | 0.000 (HS) |
| **Sedation score** | |
| **Group C** | 2.62±0.49 |
| **Group D** | 2.52±0.43 |
| **p-value** | 0.087 (NS) |

(p<0.01) – Highly significant (HS); (p<0.05) – Significant (S)(p>0.05) – Not significant (NS)
The mean dose of thiopentone sodium required for loss of eye lash reflex in group C and group D were 278±34.49 and 170.5±30.17 respectively.Statistical evaluation between the groups showed a statistically highly significant reduction in dose of thiopentone sodium required for induction (p=0.000).

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.

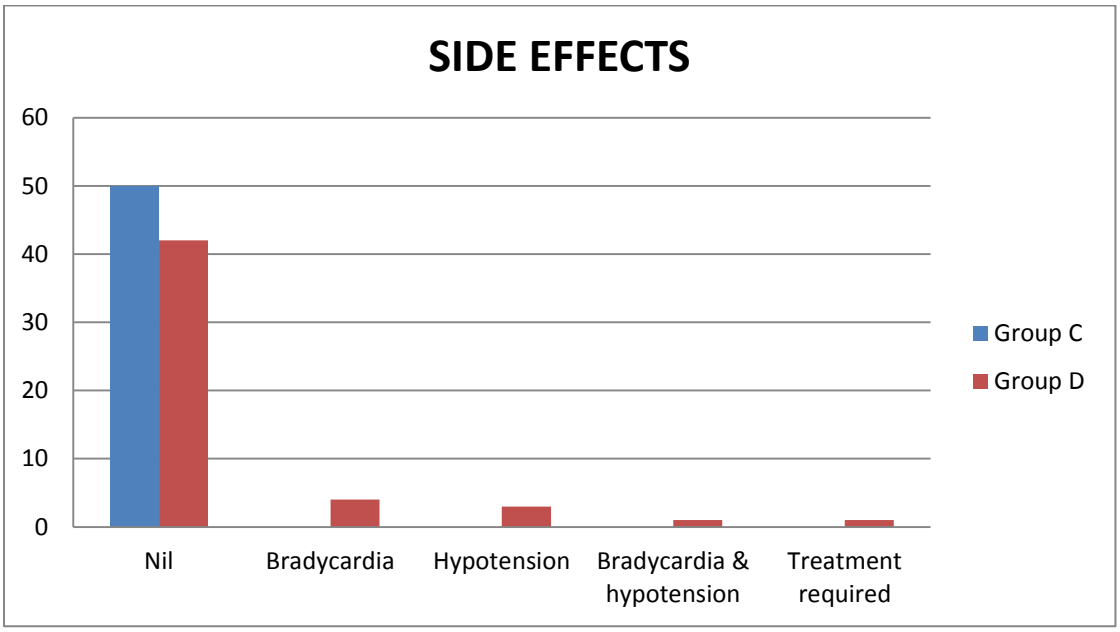



**Fig 7: Side effects between control and Dexmedetomidine group**

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. The p –value was 0.034 which is statistically significant. But all the cases were easily manageable.(Fig 7)

**Discussion**

Most of the general anesthesia procedures in the modern anesthetic practice are carried out with endotracheal intubation. Laryngoscopy and tracheal intubation are considered as the most critical events during administration of general anesthesia as they provoke transient but marked sympathoadrenal response manifesting as hypertension and tachycardia.[10]These responses are transitory, variable and may not be significant in otherwise normal individuals. But in patients with cardiovascular compromise like hypertension, ischemic heart disease, cerebrovascular disease and in patients with intracranial aneurysms, even these transient changes in hemodynamics can result in potentially harmful effects like left ventricular failure, pulmonary edema, myocardial ischemia,and cerebral hemorrhage.[3]

Many methods like use of inhalational anesthetic agents, lidocaine,[11] opioids,[12] and β-blockers[13] have been tried by various authors for blunting hemodynamic responses to laryngoscopy and intubation. But all such manoeuvres have their own limitations. Beta blockers blunt the heart rate response better than blood pressure response.[13] Hence, a drug which can blunt both the heart rate response and blood pressure response of laryngoscopy and intubation, without having any adverse effects like respiratory depression and post-operative nausea and vomiting (PONV), was required for the purpose. Dexmedetomidine is one such drug.[14]

Dexmedetomidine has been found by various authors[6, 7, 15, 16, 17, 18] to blunt the hemodynamic response for laryngoscopy and intubation. Dexmedetomidine has been recently introduced in India (in 2009) and not many studies have been done using Dexmedetomidine for suppression of intubation response. Hence, it was taken up as a study subject.

**Demographic criteria**

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

**Dose of Dexmedetomidine employed and administration**

Various authors have employed intravenous Dexmedetomidine for blunting hemodynamic response to laryngoscopy and intubation in different doses.

Kallio et al [19] showed that the maximum inhibition of sympathetic nervous system activity occurred at 50 and 75µg of Dexmedetomidine dose.

Kunisawa et al [17] used 1µg/kg body weight of Dexmedetomidine with fentanyl and found that, though there was a decrease in HR, the decrease in blood pressure was suppressed.

Esra et al [9] used 0.5 and 1µg/kg body weight of Dexmedetomidine for suppression of intubation response and did not find any significant change regarding HR in both doses.

Aho et al [7] observed that a dose of 0.6µg/kg body weight was more effective in blunting hemodynamic response to laryngoscopy and intubation than 0.3µg/kg body weight dose.

Scheinin et al, **[14]** Jakola et al [6] and Mowafi et al [15] showed that bolus dose of 0.6µg/kg body weight Dexmedetomidine given 10 min before induction effectively attenuated the sympathoadrenal responses associated with laryngoscopy and tracheal intubation.In keeping with observation of above authors, we chose 0.6µg/kg body weight dose of dexmedetomidine for our study.

**Method of administration**

Rapid bolus dose of Dexmedetomidine, results in initial transient increase in blood pressure and reflex decrease in HR which is due to peripheral α-2 adrenoceptors stimulation of vascular smooth muscle and can be attenuated by a slow infusion over 10 minutes. The administration of drug over 10 minutes is similar to the studies by Mowafi et al [15] Basar et al [16] and Kunisawa et al [17]

The administration of Dexmedetomidine as 10 ml in the present study is similar to the administration by Scheinin et al [14] and Basar et al. [16]

**Timing of administration of Dexmedetomidine**

The distribution half-life of intravenous Dexmedetomidine is approximately 6 minutes.

Various authors, Aho et al [7] Scheinin et al [14] Jakola et al [6] Mowafi et al [15] and Keniya et al [20] have employed Dexmedetomidine, 10 minutes before induction and hence, we too adopted the same method.

**Comparative analysis of hemodynamic data between the Dexmedetomidine and control groups at various intervals**

**I. Changes in heart rate after Dexmedetomidine and saline administration**

Various authors have found[6,7, 17, 18] that Dexmedetomidine decreased the HR at various intervals of 2,5,8 and 10 minutes. Our study also found similar change in HR which is statistically highly significant.

In the control group, initially there was not much variation in HR after the administration of saline in the first 10 minutes whereas, in Dexmedetomidine group there was a continuous decrease in HR at 2, 5, 8 and 10 minutes which was statistically highly significant

**After induction:** values also showed increased heart rate in group C and reduced heart rate in group D. Our present study compares well with the findings of Kunisawa et al. [17]

**After laryngoscopy and intubation**

**At 1st min-** In the present study, following laryngoscopy and intubation at 1 minute, the mean HR increased in the control group and decreased in Dexmedetomidine group which was statistically highly significant (p=0.000). Various authors[15,16] have found similar response to intravenous Dexmedetomidine at 1 min after intubation.

**At 3rd min**- The increase in mean HR at 3rd minute in control group was 29.04 bpm whereas in Dexmedetomidine group the HR decreased by 4.98 bpm which is statistically highly significant (p=0.000).

**At 5th min**- The increase in mean heart rate in control group sustained even at 5th minute, whereas, in Dexmedetomidine group there was further decrease in HR which was statistically highly significant(p=0.000). Similar findings were reported by Jakola et al [6] and Scheinin et al. [14]

**At 10th minute**- In our study even at 10th minute, there was increase in HR in control group compared to basal level and in Dexmedetomidine group, the HR remained low by 10.46 bpm which was statistically highly significant (p=0.000).We could not compare out results with other authors as they have not studied the hemodynamic parameters at 10th minute.

Basar et al [16] observed increase in HR by 5 bpm in control group and decrease in HR by 5 bpm in Dexmedetomidine group.

In the control group, significant increase in HR occurred at various intervals after intubation at 1, 3, 5 and 10 minutes with maximum rise of 36.24 bpm (1 min after intubation). Similar findings were also noted by Aho et al [7] and Basar et al.[16]

In Dexmedetomidine group there was a decrease in HR at 1 min after intubation and was sustained till 10th minute after intubation which was statistically highly significant (p=0.000).

**II. Changes in systolic blood pressure (SBP)**

**After Dexmedetomidine administration**-After administration of Dexmedetomidine at 2nd minute there was a marginal and insignificant increase of SBP. Similar observation was made by Aho et al.[7]
From the 5th minute onwards, there was a gradual reduction in blood which was statistically highly significant. Aho et al [7] and Keniya pressure till induction et al[20] found a continuous gradual reduction of SBP as in our study.
**After induction**-After induction there was a reduction of 7 mmHg of SBP in control group compared to 24 mmHg in Dexmedetomidine group to basal value which is statistically significant. Similar observations were made by Kunisawa et al.[17]

**After laryngoscopy and intubation**

**Table 5: Showing mean changes in SBP following laryngoscopy and intubation in Control and Dexmedetomidine group at various intervals**

| S. No | Author and year | Mean change in SBP in Control group | | | | Mean change in SBP in Study group | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 1 min | 3 min | 5 min | 10 min | 1 min | 3 min | 5 min | 10 min |
| **1** | Aho et al [7] 1991 | +48 | - | - | - | +48 | - | - | - |
| **2** | Scheinin et al [14] 1992 | - | - | -18 | - | - | - | -22 | - |
| **3** | Jakola et al [6] 1992 | - | - | + | - | - | - | -17 | - |
| **4** | Kunisawa et al[17] 2009 | +10 | - | - | - | -15 | - | - | - |
| **5** | Keniya et al[20] 2011 | +30 | - | 10 | - | -10 | - | -20 | - |
| **6** | **Present study** | **+30.02** | **+21.02** | **+10.7** | **+0.78** | **-15.8** | **-22.3** | **-24.3** | **-26.0** |

The sign (-) denotes decrease and (+) denotes increase in SBP. The spaces which have been left blank (' – '), are the parameters not studied by the authors.Dexmedetomidine blunts the increase in systolic blood pressure at 1, 3, 5 and 10 minutes following laryngoscopy and intubation compared to control group (p=0.000) which is statistically highly significant. Our findings are similar to those reported by Jakola et al, [6] Aho et al[7] and Scheinin et al. [14]

**III. Changes in diastolic blood pressure (DBP)**

**After Dexmedetomidine administration**-After 2nd minute there was a gradual decrease of DBP till induction which is statistically significant. In control group there was not much of variation in DBP till induction.
Similar observations were also found by Aho et al,[7] Kunisawa et al [17] and Keniya et al.[20] 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 6 mmHg in the control group and 18 mmHg in Dexmedetomidine group compared to basal value which compares well with the findings of Jakola et al.[6]
**After laryngoscopy and intubation**-In our study, there is an increase of DBP in control group which gradually decreased to near basal values by 10th minute. In Dexmedetomidine group, there is a decrease in DBP by 10th minute compared to basal values which is statistically highly significant. These findings are similar to findings of Jakola et al. [6]

**IV. Changes in mean arterial pressure (MAP)**

**After Dexmedetomidine administration**-From 5th minute onwards there is a continuous fall in MAP in Dexmedetomidine group till induction which is statistically significant. In control group not much of variation was observed regarding MAP till induction compared to basal values and to Dexmedetomidine group. Basar et al[16] also reported similar findings.
**After induction**-After induction, the reduction in MAP in Dexmedetomidine group was higher than in control group which is similar to findings of Mowafi et al.[15]
**After laryngoscopy and intubation**-Even at 10th minute the MAP was lower by 10 mmHg, compared to the basal values in Dexmedetomidine group while in the control group there was an increase in MAP which did not reach to basal values even after 10th minute. These findings concur with those of Mowafi et al [15] and Basar et al.[16].
**Dose of thiopentone required for induction**-In control group the mean dose of thiopentone for induction was 278 mg (4.96 mg/kg body weight) and in

Dexmedetomidine group it was 170.5 mg (3.08 mg/kg body weight) showing reduction of 1.88 mg/ kg body weight (38.66%) which is statistically highly significant (p=0.000).

Jakola et al**[**6]and Aanta et al [21] also observed 23 %and 37 % reduction in thiopentone dose requirements respectively in Dexmedetomidine group.

**Sedation scoring**-In group C mean sedation score immediately after extubation was 2.62 and 2.52 in Dexmedetomidine group which was statistically not significant (p=0.087).

There was no difference in both groups with respect to sedation and recovery which was similar to observation of Aanta et al [21]

**Side effects**-In Dexmedetomidine group, 4 patients developed bradycardia and 3 patients had significant hypotension which was managed by injection atropine and intravenous fluids.

## Conclusion

Laryngoscopy and endotracheal intubation are considered as the most critical events during administration of general anesthesia as they provoke transient but marked sympathoadrenal response which may result in intra-operative complications. Dexmedetomidine at a dose of 0.6µ/kg body weight given 10 minutes before induction significantly attenuated the hemodynamic responses to laryngoscopy and tracheal intubation. It also helps in reduction in dose requirement of thiopentone sodium. Side effects of Dexmedetomidine are minimal and can be managed easily.

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