

DEXMEDETOMIDINE FOR ATTENUATION OF PRESSOR RESPONSE OF LARYNGOSCOPY AND INTUBATION

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ABSTRACT: BACKGROUND: Laryngoscopy and tracheal intubation causes intense autonomic reflex responses consisting of increased circulating catecholamines, tachycardia, hypertension, myocardial oxygen demand, and dysarrhythmias. To obtund haemodynamic response lignocaine, opioids, nitroprusside, nitroglycerine, verapamil, nifedipine, esmolol, clonidine and recently, dexmedetomidine have been studied. **AIMS AND OBJECTIVES:** We investigated whether dexmedetomidine a α_2 agonist could attenuate sympathoadrenal response (Heart rate and MAP) to laryngoscopy and intubation. **MATERIALS AND METHODS:** Eighty patients, ASA grade I/II, undergoing routine general anesthesia were randomly premedicated by i. v. dexmedetomidine 0.6 μ g or saline. Heart rate (HR), mean arterial pressure (MAP), were measured before, after the premedication, after thiopental, after succinylcholine at laryngoscopy, immediately after intubation and then 1 min. 3 min. and 5 min after intubation. **STATISTICAL ANALYSIS:** Descriptive and inferential statistics using chi-square test, z-test and wilcoxon sign rank test was done. Software used in the analysis was SPSS 17.0 version and Graph Pad Prism 5.0. Data was reported as mean value \pm SD & p-value <0.05 is considered as level of significance. **RESULTS:** The demographic profile was comparable. After intubation the MAP in the control group (z=.535, p=<0.05 at laryngoscopy and z=9.95, p<0.05 after intubation) was higher than that in the dexmedetomidine group (z=8, p=0.000) and exceeded the baseline value(p<0.05) The heart rate also showed less fluctuation in the dexmedetomidine group than in the control group. Though there was rise in both the groups, it was more in control group than dexmedetomidine group (z=7.73, p<0.05 at laryngoscopy and z=9.22, p<0.05 after intubation). Thus the pressor response to laryngoscopy and intubation were effectively decreased by dexmedetomidine and were highly significant on comparison (p<0.05). **CONCLUSION:** i v dexmedetomidine 0.6 μ g premedication is advantageous as it is found to be effective and beneficial in attenuating the haemodynamic response of laryngoscopy and intubation to prevent its consequences.

KEYWORDS: α_2 adrenoreceptor, dexmedetomidine, sympathoadrenal response, tracheal intubation.

INTRODUCTION: The attendant danger of hypertension and tachycardia observed during laryngoscopy and intubation, needs considerable attention to prevent their consequences such as arrhythmia and myocardial ischemia. In patients with cardiovascular disease these hemodynamic changes may lead to life threatening complications like acute heart failure, cerebrovascular accidents.¹ To obtund the cardiovascular responses various drugs used are lignocaine in various

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forms, high doses of opioids like fentanyl, alfentanyl, sufentanyl, buprenorphine etc, vasodilators like sodium nitroprusside, nitroglycerine, hydralazine, calcium channel blockers like verapamil, nifedipine,² nitradepine, manidepine etc, alpha adrenergic blocker droperidol, beta adrenergic blockers metoprolol, esmolol,³ magnesium sulphate,⁴ alpha adrenergic agonist like clonidine⁵ and recently dexmedetomidine.⁶ However, no modality is devoid of drawbacks and limitations. Dexmedetomidine, a highly selective

α -2 adrenergic agonist has sedative and analgesic effects^{7,8} and produces hyperpolarization of noradrenergic neurons and suppression of neuronal firing in the locus cerelous leading to decreased systemic noradrenalin release that results in attenuation of sympathoadrenal responses and hemodynamic stability during laryngoscopy and tracheal intubation.⁹ The aim of our study is to evaluate the efficacy of IV dexmedetomidine pre-medication on attenuation of haemodynamic response (HR and MAP) to laryngoscopy and intubation.

MATERIALS AND METHOD: In this double-blind, randomized, controlled clinical trial, after approval from institutional ethical committee, and with written informed consent, eighty normotensive patients of either sex, aged 25-65 yrs, ASA class I or II scheduled for elective surgeries to be performed under general anaesthesia (GA) requiring endotracheal intubation, were recruited. Patients were excluded if had body weight more than 150% of their ideal body weight using Broca's index, had Contraindication to study drug, with obvious intubation difficulty, Pregnant or lactating patient, Hypertensive patients receiving any antihypertensive, had Psychiatric disorders. Randomization was done by computer generated randomization table using Software "Minitab", random sequence was generated by random allocation software. Patients were allocated in 2 groups; Group D (n=40) to receive single bolus IV dose of dexmedetomidine ($0.6 \mu\text{gkg}^{-1}$) diluted upto 20 ml as pre-medication or Group C (n=40) to receive IV saline (20 ml) as pre-medication. Syringe containing pre-medication (either dexmedetomidine or normal saline) was prepared by a team member who was not involved in the data recording. Intraoperative monitoring included three lead ECG, plethysmographic pulse oximetre, capnometry, non-invasive arterial pressure was performed. The observations were made by the same observer in all the patients so as to avoid observer bias. Patients were pre-medicated with a single dose of dexmedetomidine $0.6\mu\text{gkg}^{-1}$ IV using 20ml syringe pump over 10 min in group D and the same amount of saline was given to the patient in the control group. Both the groups were also pre-medicated with IV fentanyl $1\mu\text{gkg}^{-1}$ After pre-oxygenation for 3 min both groups were induced with injection thiopental 5mgkg^{-1} and Succinylcholine 2mgkg^{-1} was administered. After cessation of fasciculations gentle laryngoscopy was done by trained anaesthesiologist and patient was then intubated with proper size cuffed endotracheal tube under direct vision. Correct position of tracheal tube was verified by auscultation of chest and by capnometry. It was decided to exclude those patients from study who require more than one attempt for intubation. However all the patients in our study were intubated in first attempt and there was no exclusion. Intraoperative lactated Ringer solution was administered at $4\text{mlkg}^{-1} \text{h}^{-1}$ iv. Further management of cases was done according to institutional protocol for general anaesthesia. Hypotension (MAP $\leq 30\%$ from baseline) was treated with IV ephedrine 6 mg and bradycardia (heart rate $\leq 30\%$ from baseline)

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was treated with IV glycopyrolate 0.2 mg. Mean arterial pressure (MAP), heart rate (HR) readings were taken at following time interval:

- T1: Baseline on OT table
- T2: 10 min after pre-medication.
- T3: 30 seconds after thiopental.
- T4: 30 second after succinylcholine at laryngoscopy.
- T5: immediately after intubation.
- T6: at 1 min after intubation.
- T7: at 3 min after intubation
- T8: at 5 min after intubation

STATISTICAL ANALYSIS: Sample size was calculated by using EPI info software (3.4.3) in consultation with statistician, minimum sample size was calculated 40 in each group, considering confidence interval of 95 %, power 90 % with 1:1 Unpaired T – test was used to find out significance between two samples. Data was reported as mean value \pm SD & p-value <0.05 is considered as level of significance. All the data were entered into the excel database from paper proforma. During the data entry, data was checked for any error or missing data. After resolution of all issues, the database was analyzed. Statistical analysis was done by using descriptive and inferential statistics using chi-square test, z-test and wilcoxon sign rank test. The software used in the analysis were SPSS 17.0 version and Graph Pad Prism 5.0. Randomization was done using software 'Minitab' (computer software) computer generated randomization. The comparison of normally distributed continuous variables between the groups was performed by Z-test and comparison of groups from baseline was done by Wilcoxon sign rank test. Nominal categorical data among study groups were compared using the chi-square test.

RESULTS: With regard to age, weight, gender, MPC grading, and baseline HR, MAP there were no significant differences between two groups (Table 1). There was statistically significant initial fall in HR in group D after dexmedetomidine (p=0.000). There was rise in HR, after laryngoscopy and intubation, remained raised for 3 min postintubation in both the groups (p=0.000 & 0.017). But this rise was statistically significantly more in control group from T2-T6 as compared to group D (p=0.000; 0.001; 0.003; 0.000; 0.0140 at various levels from T2-T6). Heart rate in both groups was almost near to the baseline values at T7 and T8 (p=1.000). The MAP was increased significantly compared with preoperative value after intubation in the group C (p=0.000) and was significantly higher than in group D (p=0.000).

In the group D, MAP was not significantly higher than the preoperative value at all times. No incidence of hypotension or bradycardia requiring intervention was reported in both groups.

DISCUSSION: The pressor response associated with laryngoscopy and intubation is due to reflex sympatho-adrenal discharge provoked by epilaryngeal and laryngotracheal stimulation. The magnitude of the response is greater with increasing force and duration of laryngoscopy.¹⁰ The elevation in arterial pressure typically starts within five seconds of laryngoscopy, peaks in 1-2 min and returns to control levels within 5 min.

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The present study was undertaken to evaluate the effect of Dexmedetomidine which was compared with control group where saline was given. Findings of each group are discussed in comparison with their preoperative values and with control group at different time intervals with regard to MAP and Heart rate.

MAP: We observed highly significant rise in MAP at laryngoscopy and immediately after intubation ($p < 0.05$) in control group (Table 2) Dexmedetomidine group showed significant fall after giving study drug, it remained status co and there was no rise after laryngoscopy & intubation. (Table 3 and 4) as compared to control group. This observation is in agreement with previous studies.^{6, 11, 12, 13.}

HEART RATE: There was significant rise in heart rate control group starting from T2 till T5, but it was more pronounced at T4 and T5 i.e., After laryngoscopy and intubation ($p < 0.05$) (Table 5) Dexmedetomidine group showed initial fall and then significantly less rise after laryngoscopy and intubation as compared to control group. (Table 6 and 7) This observation is in agreement with previous studies.^{6, 11, 12, 13.}

Thus, in our study Dexmedetomidine premedication attenuated haemodynamic response of laryngoscopy and intubation where MAP and HR increased significantly after intubation in group C whereas in group D Dexmedetomidine premedication prevented rise in heart rate after laryngoscopy and intubation, similarly MAP did not cross the base value after laryngoscopy and intubation but remained on lower side. Several previous studies have reported the blunting effect of Dexmedetomidine on the sympathetic response to laryngoscopy and intubation^{6,11,12,13} and our findings are in accordance with them. This could be due to centrally mediated sympatholytic effects of α_2 agonists and by its decreasing norepinephrine release via peripheral presynaptic α_2 receptors^{14,15} Increased risk of hypotension and bradycardia often observed in young healthy volunteers on rapid bolus administration have been reported.^{15,16} We did not observe hypotension or bradycardia in any patient.

The α -adrenoceptors are involved in regulating the autonomic nervous system and cardiovascular systems. α_2 -adrenoceptors are located on blood vessels, where they mediate vasoconstriction and on sympathetic presynaptic terminals where they inhibit epinephrine and nor-epinephrine release.¹³ α_2 -adrenoceptors are also located within the central nervous system and their activation leads to sedation, a reduction of tonic levels of sympathetic outflow and an augmentation of Vagal activity. This can result in a decrease in HR and cardiac output. The use of α_2 -agonists in the peri-operative period has been associated with reduced anesthetic requirements and attenuated HR and blood pressure responses to stressful events.^{16,17}

The dose of dexmedetomidine pre-medication administered in our study (0.6 $\mu\text{g}/\text{kg}$) was based on the previous clinical studies^{6,9,11} where the selected dose resulted in a significant reduction in HR and MAP in response to intubation and the pressor response to laryngoscopy and endotracheal intubation was also significantly attenuated. Higher doses of dexmedetomidine were associated with hypotension and bradycardia^{18,19} With the use of lower dose of dexmedetomidine, the increase in BP during intubation could not be attenuated.²⁰

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Studies suggest that perioperative use of dexmedetomidine may result in a decreased risk of adverse cardiac events, including myocardial ischemia.²¹ α adrenoreceptors stimulation can beneficially modulate coronary blood flow during myocardial ischemia by preventing transmural redistribution of blood flow away from the ischemic endocardium, by specific epicardial vasoconstrictive effects, leading to improvement in endocardial perfusion (the reverse steal effect) and by decreasing heart rate. This property along with hemodynamic stability and attenuation of intubation response makes dexmedetomidine an ideal anesthetic adjuvant, particularly for patients undergoing coronary bypass grafting. Dexmedetomidine has been also administered in infant following open heart surgery²² with a decrease in heart rate during the immediate postoperative time together with an effective and safe sedation.

A biphasic cardiovascular response has been described after the administration of dexmedetomidine.²³ A bolus of 1 mcg/kg results in a transient increase in arterial blood pressure and reflex decrease in heart rate in young healthy patients. Initial response is due to α_2 receptor stimulation of vascular smooth muscle. This response can be markedly decreased by slow infusion over 10 min. In our study, this effect was not noticed due to the slow infusion of the drug over 10 min.

The major limitations of our study being a single center study was that it was not feasible to validate our conclusion as the sample size was small (n 40). Hemodynamic stability offered by dexmedetomidine would have been better established by measuring plasma catecholamine level which was not practically feasible in our institute. Additionally to substantiate the cardiovascular stability of such drug larger meta-analytical studies would be required to be done instead of small study of ours. Hence, future study on large number of patients could strongly prove the hypothesis. The rapid speed of infusion also determines the higher incidence of side effects like apnoea and irregular ventilation which occurs due to increased central sedation rather than direct respiratory depressant effect.^{24,25}

We conclude that IV dexmedetomidine $0.6 \mu\text{gkg}^{-1}$ in 10 ml NS, as premedication given slowly over 10 min before induction can be effective and safe drug in attenuating the hemodynamic response of laryngoscopy and intubation which can be more beneficial to the patients who are already at risk of deleterious cardiac effects as myocardial ischemia might occur during the induction. Intubation sequence in patients with coronary artery disease and intraoperative ischemia has been associated with a high rate of perioperative myocardial infarction. Hence, prevention is definitely mandatory in such cases.

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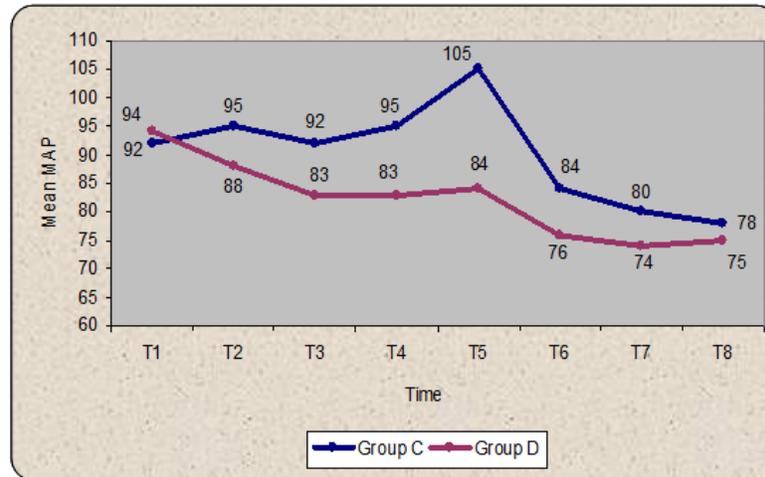


Fig. 1

Fig. 1: Changes in MAP in the control and dexmedetomidine groups. Measurements were recorded before premedication (T1), 10 min after premedication (T2), 30 secs after thiopental (T3), 30 secs after succinylcholine at laryngoscopy (T4), immediately after intubation (T5) and 1,3,5, min after intubation (T6-8). Significant difference of MAP between control and dexmedetomidine group at T2-T7. Rise was maximum at T5 in control group.

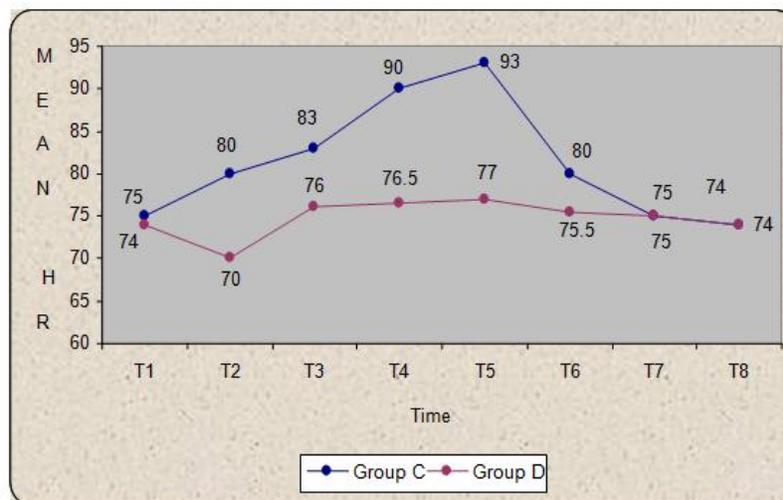


Fig. 2

Fig. 2: Changes in HR in the control and dexmedetomidine groups. Measurements were recorded before premedication (T1), 10 min after premedication (T2), 30 secs after thiopental (T3), 30 secs after succinylcholine (T4), immediately after intubation (T5) and 1,2,3, min after intubation (T6-8). Significant difference of HR between control and dexmedetomidine group at T2-T6. In control group significant continuous rise from T2-T5 and stability in dexmedetomidine group all throughout.

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	Group C	Group D	P - value
Age (yr)	39.0 ±10.23	42.22±12.25	0.22NS,p>0.05
MPC Grade I/II	23/17;57.5%/42.5%	20/20;50%/50%	
Weight (kg)	53.25±7.77	53.10±8.50	0.92,NS,0>0.05
Gender-Male/Female	19/21;47.5%/52.5%	16/24;40%/60%	0.22,NS,p>0.05
Preoperative HR (beat min ⁻¹)	75 7.13	74 8.07	0.55;NS,p>0.05
Preoperative MAP (mm hg)	92 ± 12.52	94 ±8.76	0.42;NS P>0.05

Table 1: Patient Characteristics

	Mean	N	Std. Deviation	Std. Error Mean	z-value	p-value
T1	92.00	40	12.52	1.98	-	-
T2	95.00	40	12.55	1.98	10.64	0.000 S, p<0.05
T3	92.00	40	12.13	1.91	0.00	1.000 NS, p>0.05
T4	95.00	40	11.19	1.76	6.31	0.000 S, p<0.05
T5	105.00	40	10.21	1.61	17.04	0.000 S, p<0.05
T6	84.00	40	8.56	1.35	6.72	0.000 S, p<0.05
T7	80.00	40	8.25	1.30	9.46	0.000 S, p<0.05
T8	78.00	40	7.78	1.23	10.84	0.000 S, p<0.05

Table 2: Comparison of MAP from T2 to T8 as compared to baseline in group C using Wilcoxon sign rank test

	Mean	N	Std. Deviation	Std. Error Mean	z-value	p-value
T1	94.00	40	8.76	1.38	-	-
T2	88.00	40	7.12	1.12	12.15	0.000 S, p<0.05
T3	83.00	40	8.15	1.29	10.19	0.000 S, p<0.05
T4	83.00	40	8.67	1.37	8.55	0.000 S, p<0.05
T5	84.00	40	8.58	1.35	8.00	0.000 S, p<0.05
T6	76.00	40	7.88	1.24	9.96	0.000 S, p<0.05
T7	74.00	40	7.13	1.12	11.65	0.000 S, p<0.05
T8	75.00	40	6.78	1.07	11.44	0.000 S, p<0.05

Table 3: Comparison of MAP from T2 to T8 as compared to baseline in group D using Wilcoxon sign rank test

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	Group C		Group D		z-value	p-value
	Mean	SD	Mean	SD		
T1	92.00	12.52	94.00	8.76	0.82	0.411 NS, p>0.05
T2	95.00	12.55	88.00	7.12	3.06	0.003 S, p<0.05
T3	92.00	12.13	83.00	8.15	3.89	0.000 S, p<0.05
T4	95.00	11.19	83.00	8.67	5.35	0.000 S, p<0.05
T5	105.0	10.21	84.00	8.58	9.95	0.000 S, p<0.05
T6	84.00	8.56	76.00	7.88	4.34	0.000 S, p<0.05
T7	80.00	8.25	74.00	7.13	3.47	0.001 S, p<0.05
T8	78.00	7.78	75.00	6.78	1.83	0.070 NS, p>0.05

Table 4: Comparison of MAP in both the groups using Z-test

	Mean	N	Std. Deviation	Std. Error Mean	z-value	p-value
T1	75.00	40	7.13	1.12	-	-
T2	80.00	40	8.17	1.29	8.79	0.000 S, p<0.05
T3	83.00	40	7.77	1.23	13.62	0.000 S, p<0.05
T4	90.00	40	8.07	1.27	24.77	0.000 S, p<0.05
T5	93.00	40	8.00	1.26	29.41	0.000 S, p<0.05
T6	80.00	40	7.37	1.16	8.22	0.000 S, p<0.05
T7	75.00	40	7.16	1.13	0.00	1.000 NS, p>0.05
T8	74.00	40	6.99	1.10	1.64	0.108 NS, p>0.05

Table 5: Comparison of Heart Rate from T2 to T8 as compared to baseline in group C using Wilcoxon sign rank test

	Mean	N	Std. Deviation	Std. Error Mean	z-value	p-value
T1	74.00	40	8.07	1.27	-	-
T2	70.00	40	7.92	1.25	17.23	0.000 S, p<0.05
T3	76.00	40	8.21	1.29	3.74	0.001 S, p<0.05
T4	76.50	40	7.52	1.18	3.19	0.003 S, p<0.05
T5	77.00	40	7.49	1.18	3.80	0.000 S, p<0.05
T6	75.50	40	7.33	1.16	2.57	0.014 S, p<0.05
T7	75.00	40	7.11	1.12	1.26	0.214 NS, p>0.05

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T8	74.00	40	6.63	1.04	0.00	1.000 NS, p>0.05
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Table 6: Comparison of Heart Rate from T2 to T8 as compared to baseline in group D using Wilcoxon sign rank test

	Group C		Group D		z-value	p-value
	Mean	SD	Mean	SD		
T1	75.00	7.13	74.00	8.07	0.58	0.559 NS, p>0.05
T2	80.00	8.17	70.00	7.92	5.55	0.000 S, p<0.05
T3	83.00	7.77	76.00	8.21	3.91	0.000 S, p<0.05
T4	90.00	8.07	76.50	7.52	7.73	0.000 S, p<0.05
T5	93.00	8.00	77.00	7.49	9.22	0.000 S, p<0.05
T6	80.00	7.37	75.50	7.33	2.43	0.017 S, p<0.05
T7	75.00	7.16	75.00	7.11	0.00	1.000 NS, p>0.05
T8	74.00	6.99	74.00	6.63	0.00	1.000 NS, p>0.05

Table 7: Comparison of HR in both the groups using Z-test

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