

Evaluation of Dexmedetomidine-0.5 µg/kg and 1 µg/kg in Blunting the Responses to Laryngoscopy and Intubation

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ABSTRACT

Background: There is an on-going search for an ideal agent to blunt hemodynamic responses pertaining to tracheal intubation.

Objective: To compare the hemodynamic effects of intravenous (IV) dexmedetomidine in a dose of 0.5 µg/kg and 1 µg/kg body weight as premedicant.

Materials and Methods: 90 patients scheduled for various elective surgical procedures under general anesthesia belonging to ASA Class I and II and Mallampatti Grades I and II in the age group of 18 years to 60 years were included in the study. Patients with uncontrolled systemic disorders, difficult airways, and obesity were excluded from the study. The study population was randomly divided into three groups: (1) Group A: 30 Patients, receiving 10 ml of 0.9% saline IV, 10 min before induction. (2) Group B: 30 Patients, receiving 0.5 µg/kg dexmedetomidine IV, 10 min prior to induction (made to 10 ml with normal saline) (3) Group C: 30 Patients receiving 1 µg/kg dexmedetomidine IV 10 min prior to induction (made to 10 ml with normal saline). Patients of all the above-mentioned groups were premedicated with injection ondansetron 0.08 mg/kg IV and injection fentanyl 1.5 µg/kg IV prior to starting of infusion of the study drug.

Result: In our study, both the doses of IV dexmedetomidine blunted hemodynamic responses, however, IV dose of 1 µg/kg body weight was found more optimal with minimal incidence of side effects.

Conclusion: We conclude that dexmedetomidine in a loading dose of 1 µg/kg body weight significantly attenuates response to laryngoscopy and intubation with minimal incidence of side effects as compared with dexmedetomidine in a dose of 0.5 µg/kg which was found insufficient in majority of cases to cause complete attenuation of sympathetic response.

Key words: Intubation Blunting, IV Dexmedetomidine, General anaesthesia & Dexmedetomidine

INTRODUCTION

Laryngoscopy and tracheal intubation are invariably accompanied by an increase in arterial blood pressure and heart rate (HR). The peak rise in blood pressure and HR is usually transient, occurring 30 s after intubation and lasting for <10 min.¹

The magnitude of hemodynamic changes observed may be dependent on various factors such as depth of anesthesia, whether any measures are taken prior to airway manipulation, the anesthetic agent used, the duration of laryngoscopy and intubation. Until date, the exact mechanism of hemodynamic responses to laryngoscopy and intubation has not been clarified. The principle mechanism in hypertension and tachycardia is the sympathetic response^{2,3} which may be the result of increase in catecholamine activity.⁴

The increase in the pulse rate and blood pressure are usually transitory, variable, and unpredictable. Transitory hypertension and tachycardia are probably

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of no consequence in healthy individuals but either or both may be hazardous to those with hypertension, myocardial insufficiency or cerebrovascular diseases.⁴ This laryngoscopic response in such individuals may predispose them to development of pulmonary edema, myocardial insufficiency, and cerebrovascular accident.^{5,6}

Intravenous (IV) anesthetic induction agents do not adequately or predictably suppress the circulatory responses caused by endotracheal intubation.⁷ Hence, before initiating laryngoscopy, additional pharmacological measures such as use of volatile anaesthetics,⁸ topical and IV lidocaine,^{9,10} opioids,¹¹ vasodilators - sodium nitroprusside,¹² nitroglycerine,¹³ calcium channel blockers,¹⁴ and β -blockers¹⁵ have been tried by various authors.

None of the drugs mentioned above have been found to be completely effective in attenuating the sympathetic response to intubation.

α -2 agonists have been used for attenuating the sympathetic response¹⁶ and both clonidine and dexmedetomidine appear to fulfill all the above criteria. Both Clonidine and dexmedetomidine have actions on both α -1 and α -2 receptors, but dexmedetomidine is highly specific and selective α -2 adrenoceptor agonist with α 2: α 1 binding selectivity ratio of 1620:1 compared to 220:1 for clonidine.¹⁷

Various studies have also found that dexmedetomidine can decrease the hemodynamic response to laryngoscopy and intubation.^{18,19}

The advantages of IV dexmedetomidine as premedicant in anesthesia setting include anxiolysis, analgesia, sedation, improved hemodynamic stability, and no/minimal respiratory depression. These beneficial properties also result in decreased minimum alveolar concentration (MAC) of volatile anesthetics which decreases significantly up to 90% and hence decreases the requirement of anesthetics.²⁰

The present study, however, was aimed at attenuation of the hemodynamic response to laryngoscopy and intubation in adult patients posted for various surgeries under general anesthesia, with single IV bolus dose of 0.5 mcg/kg and 1 mcg/kg given over 10 min before induction.

Pharmacology

Dexmedetomidine

Dexmedetomidine is the S-enantiomer (dextroisomer) of medetomidine, a widely used anesthetic in veterinarian practice. Dexmedetomidine was first approved for use in 1999 by the FDA as a short term (<24 h) sedative and analgesic for use in the ICU in patients being mechanically ventilated.²¹

IV/Intramuscular dexmedetomidine causes anxiolysis, sedation, analgesia, and sympatholysis produced with minimal respiratory depression. It is used in ICU setting, as a premedication, as a sole anesthetic agent, in regional anesthesia, and in MAC.

Dose: Dexmedetomidine is initiated as 0.5-1 μ g/kg IV over 10 min followed by a maintenance infusion of 0.2-0.7 μ g/kg/h for a period not <24 h.

MATERIALS AND METHODS

A total of 90 patients scheduled for various elective surgical procedures under general anesthesia belonging to ASA Class I and II and Mallampatti Grades I and II in the age group of 18 years to 60 years were included in the study.

The detailed preanesthetic checkup was done on all patients, and relevant hematological, biochemical, and radiological investigations were carried out for all patients as per surgical requirements.

The study population was randomly divided into three groups:

Group A: 30 Patients, receiving 10 ml of 0.9% saline IV, 10 min before induction.

Group B: 30 Patients, receiving 0.5 μ g/kg dexmedetomidine IV, 10 min before induction (made to 10 ml with normal saline).

Group C: 30 Patients receiving 1 μ g/kg dexmedetomidine IV 10 min before induction (Made to 10 ml with normal saline).

Anesthetic Procedure

Patients of all the above-mentioned groups were premedicated with injection Ondansetron 0.08 mg/kg IV and injection fentanyl 1.5 μ g/kg IV prior to starting of infusion of the study drug.

Anesthesia was induced after the administration of the study drug using injection Propofol 2 mg/kg IV and injection Vecuronium 0.08 mg/kg IV. This was followed by 3 min of ventilation. Laryngoscopy and endotracheal intubation were done. After intubation, the patient was maintained on O₂:N₂O (1:1) and isoflurane (isoflurane was started 10 min post-intubation). The concentration of isoflurane was adjusted to maintain systolic blood pressure (SBP) within 20% of the preoperative values. Further neuromuscular block was maintained by injection Vecuronium 0.02 mg/kg body weight.

The SBP, diastolic blood pressure (DBP), mean arterial blood pressure (MAP), and HR were recorded. All these parameters were recorded as a baseline value,

T = 0 min, 5 min after starting dexmedetomidine infusion, preintubation (T = 10 min), immediately post-intubation, post-intubation 1, 3, 5 min, and at 10 min.

Adverse Effects

1. Hypotension was defined as SBP <90 mmHg or a DBP of <60 mmHg or MAP of <50 mmHg on any reading.
2. Bradycardia was defined as HR <50 b/min.
3. Arrhythmia was defined as supraventricular or a ventricular beats >3/min or a rhythm other than sinus.

Exclusion Criteria

- Patient refusal
- Patients with ASA Class III and above
- Patients on preoperative beta blockers
- Age <18 years and >60 years
- Pregnant or nursing women
- Any history of drug reactions
- Patients with anticipated difficult intubation
- Patient having hypovolemia, hypotension, and bradycardia
- Duration of laryngoscopy and endotracheal intubation >30 s
- Patients with systemic diseases, obesity.

DISCUSSION

Baseline Comparison of Groups

Demographic parameters

The study included patients in the age group ranging from age 18 to 60 years for all 3 groups. The mean values of the age were 38.33 ± 11.67 , 38.30 ± 9.45 , and 34.13 ± 11.42 years for Groups A, B, and C, respectively (Table 1). There was no statistical significance difference between the 3 groups with respect to age ($P = 0.234$).

Distribution of sex was also comparable (Table 2). In our study, Group A, 63.3% were males and 36.7% were females. In Group B, 46.7% were males and 53.3% were females. In Group C, 46.7% were males and 53.3% were females. No statistically significant difference was observed in sex distribution of the cases between the 3 groups ($P = 0.328$).

The mean weights of patients were 64.63 ± 10.42 kg, 61.87 ± 10.21 kg, 59.20 ± 10.17 kg in Group A, Group B, and Group C, respectively (Table 3). No statistically significant difference was observed in weight distribution of the cases between the 3 groups ($P = 0.128$).

Hemodynamic parameters

SBP

In Group A control, the mean baseline SBP was 124.13 ± 12.66 , which decreased by 3.1 mmHg after

completion of administration of study drug. Post-intubation, there was a rise in the mean SBP values from the pre-laryngoscopic values (T-10) by 29.64 mmHg immediately after intubation, 22.14 mmHg at 1 min, 15.3 mmHg at 3 min, 6.54 mmHg at 5 min, and 2.37 mmHg at 10 min (Table 4).

In Group B, the mean baseline SBP was 126.03 ± 11.62 which decreased by 6.66 mmHg after completion of administration of study drug. Post-intubation there was a rise in the mean SBP values from the Pre-laryngoscopic values (T-10) by 17 mmHg immediately after intubation, 12 mmHg at 1 min, 6.76 mmHg at 3 min, 1.3 mmHg at 5 min and decreased by 0.2 mmHg at 10 min as compared with pre-laryngoscopic values (Table 4).

In Group C, the mean baseline SBP was 126.67 ± 9.01 which decreased by 15.94 mmHg after completion of administration of the study drug. Post-intubation there was a rise in the mean SBP values from the pre-laryngoscopic values (T-10) by 7.87 mmHg immediately after intubation, 7.14 mmHg at 1 min, 1.90 mmHg at 3 min, and decreased by 0.3 mmHg at 5 min and 1.6 mmHg at 10 min as compared with pre-laryngoscopic values. However, at no point of time during the post-intubation period did the mean SBP rise above the Baseline mean SBP value of the study population in this group (Figure 1).

Group C demonstrated a better suppression of the pressor response to intubation compared with other two groups.

DBP

In Group A control, the mean baseline DBP was 78.00 ± 8.60 , which decreased by 0.4 mmHg after completion of administration of study drug. Post-intubation there was a rise in the mean SBP values from the Pre-laryngoscopic values (T-10) by 18.23 mmHg immediately after intubation, 13.77 mmHg at 1 min, 6.87 mmHg at 3 min, 1.73 mmHg at 5 min, and decreased by 0.17 mmHg at 10 min as compared with pre-laryngoscopic values (Table 5).

In Group B, the mean baseline DBP was 76.97 ± 8.28 which decreased by 1.2 mmHg after completion of administration of study drug. Post-intubation there was a rise in the mean DBP values from the pre-laryngoscopic values (T-10) by 11.96 mmHg immediately after intubation, 8.43 mmHg at 1 min, 1.66 mmHg at 3 min, and decreased by 0.24 mmHg at 5 min and decreased by 1.54 mmHg at 10 min as compared with pre-laryngoscopic values (Table 5).

In Group C, the mean baseline DBP was 78.87 ± 6.66 which decreased by 9.74 mmHg after completion of administration of the study drug. Post-intubation there was a rise in the mean SBP values from the pre-laryngoscopic values (T-10) by 6.04 mmHg immediately after intubation,

4.0 mmHg at 1 min, 0.4 mmHg at 3 min, and decreased by 0.7 mmHg at 5 min and 2.13 mmHg at 10 min as compared with pre-laryngoscopic values (Table 5). However, at no point in time during the post-intubation period did the mean DBP rise above the baseline mean DBP value of the study population in this group (Figure 2).

Group C demonstrated a better suppression of the pressor response to intubation compared to other two groups.

MAP

In Group A control, the mean baseline MAP was 93.30 ± 8.76, which decreased by 1.0 mmHg after completion of administration of study drug. Post-intubation there was

a rise in the mean MAP values from the pre-laryngoscopic values (T-10) by 21.87 mmHg immediately after intubation, 16.3 mmHg at 1 min, 9.43 mmHg at 3 min, 3.23 mmHg at 5 min, and by 0.5 mmHg at 10 min (Table 6). In Group B, the mean baseline MAP was 92.97 ± 8.15 which decreased by 2.65 mmHg after completion of administration of study drug. Post-intubation there was a rise in the mean MAP values from the pre-laryngoscopic values (T-10) by 12.95 mmHg immediately after intubation, 8.71 mmHg at 1 min, 3.31 mmHg at 3 min, 0.25 mmHg at 5 min, and decreased by 1.11 mmHg at 10 min (Table 6).

In Group C, the mean baseline MAP was 94.53 ± 6.66 which decreased by 11.66 mmHg after completion of administration of the study drug. Post-intubation there was a rise in the mean MAP values from the pre-laryngoscopic values (T-10) by 6.83 mmHg immediately after intubation, 5.26 mmHg at 1 min, 1.1 mmHg at 3 min, and decreased by 0.24 mmHg at 5 min and 1.97 mmHg at 10 min as compared with pre-laryngoscopic values (Table 6). However, at no point of time during the post-intubation period did the mean MAP rise above the Baseline mean MAP value of the study population in this group (Figure 3).

Group C demonstrated a better suppression of the pressor response to intubation compared to other two groups.

HR

In Group A control, the mean baseline HR was 79.8 ± 11.06, which remained the same after completion of administration of study drug. Post-intubation there was a rise in the mean HR values from the pre-laryngoscopic values (T-10) by 28.37 beats/min immediately after intubation, 21.67 beats/min at 1 min, 16.9 beats/min at 3 min, 9.07 beats/min at 5 min, and by 3.54 beats/min at 10 min (Table 7).

In Group B, the mean baseline HR was 80.73 ± 10.65 which decreased by 7.36 beats/min after completion of administration of study drug. Post-intubation there was a rise in the mean HR values from the Pre-laryngoscopic

Table 1: Age distribution of patients studied

Age groups (years)	Frequency (%)			P value
	Group A	Group B	Group C	
18-30	11 (36.7)	7 (23.3)	12 (40.0)	
31-40	8 (26.7)	12 (40.0)	11 (36.7)	
41-50	7 (23.3)	10 (33.3)	5 (16.7)	
51-60	4 (13.3)	1 (3.3)	2 (6.7)	
Total	30 (100)	30 (100)	30 (100)	
Mean±SD	38.33±11.67	38.30±9.45	34.13±11.42	0.234

SD: Standard deviation

Table 2: Gender distribution of patients studied

Sex	Frequency (%)			P value
	Group A	Group B	Group C	
Male	19 (63.3)	14 (46.7)	14 (46.7)	0.328
Female	11 (36.7)	16 (53.3)	16 (53.3)	
Total	30 (100)	30 (100)	30 (100)	

Table 3: Distribution of weight in three groups of patients studied

Weight (in Kgs)	Mean±SD			P value
	Group A	Group B	Group C	
Weight	64.63±10.42	61.87±10.21	59.20±10.17	0.128

SD: Standard deviation

Table 4: Comparison SBP mmHg in three groups of patients studied

SBP	Mean±SD			Group A vs. Group B	Group A vs. Group C	Group B vs. Group C
	Group A	Group B	Group C			
Baseline	124.13±12.66	126.03±11.62	126.67±9.01	0.789	0.657	0.974
T0	128.83±11.07	131.00±10.53	130.87±9.53	0.699	0.730	0.999
T5	124.40±10.18	124.20±12.66	117.30±11.43	0.997	0.048	0.057
T10	121.03±9.58	119.37±15.39	110.73±12.25	0.866	0.006	0.026
TP1	150.67±11.14	136.37±15.49	118.60±10.78	<0.001	<0.001	<0.001
T1	143.17±12.21	131.37±13.73	117.87±11.71	0.001	<0.001	<0.001
T3	136.33±11.24	126.13±12.03	112.63±10.76	0.002	<0.001	<0.001
T5	127.57±12.26	120.67±11.41	110.43±10.04	0.052	0.002	<0.001
T10	123.40±12.88	119.17±11.43	109.13±9.51	0.323	<0.001	0.003

SD: Standard deviation, SBP: Systolic blood pressure

Table 5: Comparison DBP mmHg in three groups of patients studied

DBP	Mean±SD			Group A vs. Group B	Group A vs. Group C	Group B vs. Group C
	Group A	Group B	Group C			
Baseline	78.00±8.60	76.97±8.28	78.87±6.66	0.868	0.905	0.621
T0	80.30±8.53	77.87±8.03	81.43±5.67	0.425	0.829	0.163
T5	77.33±9.60	74.87±9.30	72.73±8.01	0.540	0.123	0.630
T10	77.60±8.99	75.77±11.78	69.13±8.32	0.750	0.003	0.028
TP1	95.83±10.54	87.73±9.77	75.17±8.25	0.004	<0.001	<0.001
T1	91.37±9.52	84.20±9.06	73.13±9.68	0.011	<0.001	<0.001
T3	84.47±7.85	77.43±9.67	69.53±9.17	0.008	<0.001	0.003
T5	79.33±9.42	75.53±9.47	68.43±9.10	0.261	<0.001	0.011
T10	77.43±11.07	74.23±9.80	67.00±8.66	0.426	<0.001	0.016

DBP: Diastolic blood pressure, SD: Standard deviation

Table 6: Comparison MAP mmHg in three groups of patients studied

MAP	Mean±SD			Group A vs. Group B	Group A vs. Group C	Group B vs. Group C
	Group A	Group B	Group C			
Baseline	93.30±8.76	92.97±8.15	94.53±6.66	0.985	0.817	0.722
T0	96.43±8.26	95.30±7.56	97.87±5.31	0.813	0.719	0.351
T5	92.60±9.32	91.00±8.82	87.50±8.27	0.762	0.070	0.278
T10	92.30±8.33	90.32±12.22	82.87±9.05	0.725	0.001	0.014
TP1	114.17±10.20	103.27±10.62	89.70±8.60	<0.001	<0.001	<0.001
T1	108.60±9.56	99.03±9.48	88.13±9.87	0.001	<0.001	<0.001
T3	101.73±8.05	93.63±9.09	83.97±9.19	0.002	<0.001	<0.001
T5	95.53±9.56	90.57±9.19	82.63±8.88	0.099	<0.001	0.004
T10	92.80±10.86	89.21±9.33	80.90±8.25	0.317	<0.001	0.003

MAP: Mean arterial blood pressure, SD: Standard deviation

Table 7: Comparison of HR (bpm) in three groups of patients studied

HR	Mean±SD			Group A vs. Group B	Group A vs. Group C	Group B vs. Group C
	Group A	Group B	Group C			
Baseline	79.80±11.06	80.73±10.65	83.73±12.15	0.945	0.373	0.561
T0	87.00±11.16	83.23±13.52	88.53±13.51	0.491	0.888	0.248
T5	82.73±10.68	77.60±12.50	75.53±10.35	0.185	0.039	0.756
T10	79.83±9.97	73.37±12.23	68.50±9.50	0.054	<0.001	0.185
TP1	108.20±15.21	93.60±10.12	75.10±9.42	<0.001	<0.001	<0.001
T1	101.50±12.95	88.40±10.88	75.17±9.42	<0.001	<0.001	<0.001
T3	96.73±10.52	85.90±11.32	72.93±9.94	<0.001	<0.001	<0.001
T5	88.90±10.38	82.73±11.00	70.97±9.67	0.060	<0.001	<0.001
T10	83.37±10.50	79.77±10.34	69.83±9.78	0.364	<0.001	0.001

HR: Heart rate, SD: Standard deviation

Table 8: Comparison of side effects in three groups of patients studied

Side effect	Frequency (%)			P value
	Group A	Group B	Group C	
None	30 (100)	28 (93.3)	28 (93.3)	0.351
Bradycardia	0 (0.0)	2 (6.7)	2 (6.7)	
Hypotension	0 (0.0)	0 (0.0)	0 (0.0)	
Arrhythmias	0 (0.0)	0 (0.0)	0 (0.0)	
Total	30 (100)	30 (100)	30 (100)	

values (T-10) by 20.23 beats/min immediately after intubation, 15.03 beats/min at 1 min, 12.53 beats/min

at 3 min, 9.36 beats/min at 5 min, and 6.4 beats/min at 10 min (Table 7).

In Group C, the mean baseline HR was 83.73 ± 12.15 which decreased by 15.23 beats/min after administration of the study drug. Post-intubation there was a rise in the mean HR values from the pre-laryngoscopic values (T-10) by 6.6 beats/min immediately after intubation, 6.67 beats/min at 1 min, 4.43 beats/min at 3 min, 2.47 beats/min at 5 min and 1.33 beats/min at 10 min (Table 7). However, at no point of time during the post-intubation period did the mean HR rise above the T0 min mean HR value of the study population in this group (Figure 4).

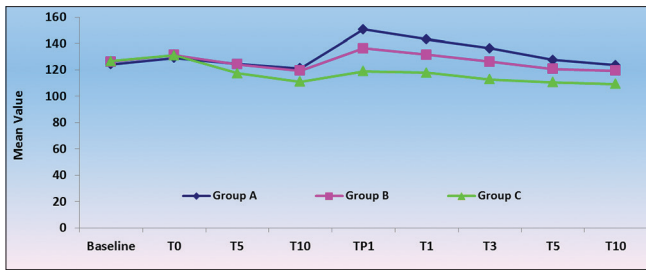


Figure 1: Comparison of mean systolic blood pressure among groups at different time points

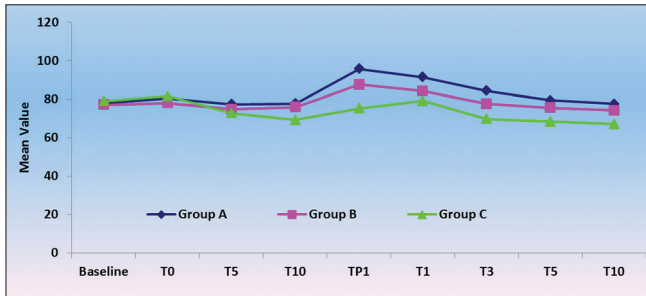


Figure 2: Comparison of mean diastolic blood pressure among groups at different time points

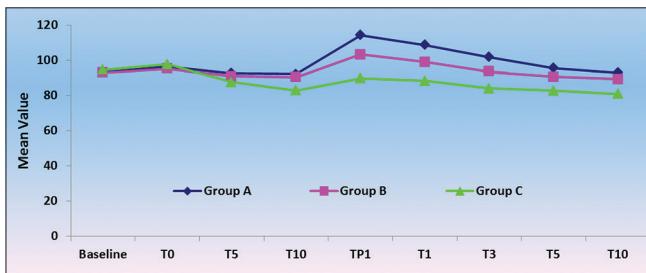


Figure 3: Comparison of mean arterial blood pressure among groups at different time points

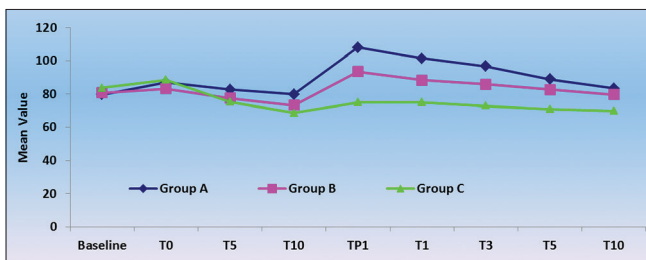


Figure 4: Comparison of mean heart rate among groups at different time points

Group C demonstrated a better suppression of the pressor response to intubation compared to other two groups.

Side effects

Group A, Group B, and Group C were statistically comparable in terms of side effects ($P = 0.351$). There

were 2 cases of bradycardia ($HR < 50/min$) in Group B, and 2 cases of bradycardia ($HR < 50/min$) in Group C, respectively. There were no other cases with hypotension or arrhythmias in terms of side effects (Table 8).

Comparison with Other Studies

In the study by Menda *et al.*,¹⁸ they found that the SBP, DBP, MAP, HR values were below the baseline values in the dexmedetomidine group at all measurement times,¹⁸ which was in accordance with our results.

Esra *et al.*²² observed in their study using dexmedetomidine $1 \mu g/kg$ that the mean SBP, DBP, MAP, and HR values were significantly lower at post-induction and 5 min after intubation compared to baseline values which were similar to the results of our study.

Our study results also concurred with the results of Keniya *et al.*²³ who observed an 8% increase in SBP and 11% for DBP, 7% for HR when compared with pre-laryngoscopic values in dexmedetomidine 1 mcg/kg group.

CONCLUSION

- Dexmedetomidine in a dose of 0.5 mcg/kg :
 - Is insufficient to cause complete blunting of the hemodynamic response to laryngoscopy and intubation for 10 min in majority of cases.
- Dexmedetomidine in a dose of 1 mcg/kg :
 - Significantly attenuates the hemodynamic response to laryngoscopy and intubation for 10 min.
 - Side effects observed were not statistically significant and comparable with side effects present in Group B.

No arrhythmias were seen during/after administration of study drug.

REFERENCES

1. Reid LC, Brace DE. Irritation of respiratory tract and its reflex effect on heart. *Surg Gynecol Obstet* 1940;70:157.
2. Kayhan Z, Aldemir D, Mutlu H, Oğüs E. Which is responsible for the haemodynamic response due to laryngoscopy and endotracheal intubation? Catecholamines, vasopressin or angiotensin? *Eur J Anaesthesiol* 2005;22:780-5.
3. Morin AM, Geldner G, Schwarz U, Kahl M, Adams HA, Wulf H, *et al.* Factors influencing preoperative stress response in coronary artery bypass graft patients. *BMC Anesthesiol* 2004;4:7.
4. Kovac AL. Controlling the hemodynamic response to laryngoscopy and endotracheal intubation. *J Clin Anesth* 1996;8:63-79.
5. Prys-Roberts C, Greene LT, Meloche R, Foëx P. Studies of anaesthesia in relation to hypertension. II. Haemodynamic consequences of induction and endotracheal intubation. *Br J Anaesth* 1971;43:531-47.
6. Dalton B, Guiney T. Myocardial ischemia from tachycardia and

- hypertension in coronary heart disease – Patients undergoing anaesthesia. Annual Meeting. Boston: American Society of Anesthesiologists; 1972. p. 201-2.
7. Stoelting RK, Hiller SC. Textbook of Pharmacology and Physiology in Anesthetic Practice. 4th ed. Philadelphia: Lippincott Williams and Wilkins; 2006. p. 340-5.
 8. King BD, Harris L, Greifenstein F, Elder J, Dripps RD. Reflex circulatory responses to direct laryngoscopy and intubation under general anaesthesia. *Anaesthesiology* 1951;12:556-66.
 9. Stoelting RK. Blood pressure and heart rate changes during short duration laryngoscopy for tracheal intubation: Influence of viscous or intravenous lignocaine. *Anaesth Analg* 1978;57:197-9.
 10. Stoelting RK. Circulatory changes during direct laryngoscopy and tracheal intubation: Influence of duration of laryngoscopy with or without prior lidocaine. *Anesthesiology* 1977;47:381-4.
 11. Martin DE, Rosenberg H, Aukburg SJ, Bartkowski RR, Edwards MW Jr, Greenhow DE, *et al.* Low-dose fentanyl blunts circulatory responses to tracheal intubation. *Anesth Analg* 1982;61:680-4.
 12. Stoelting RK. Attenuation of blood pressure response to laryngoscopy and tracheal intubation with sodium nitroprusside. *Anesth Analg* 1979;58:116-9.
 13. Fassoulaki A, Kaniaris P. Intranasal administration of nitroglycerine attenuates the pressor response to laryngoscopy and intubation of the trachea. *Br J Anaesth* 1983;55:49-52.
 14. Nishikawa T, Namiki A. Attenuation of the pressor response to laryngoscopy and tracheal intubation with intravenous verapamil. *Acta Anaesthesiol Scand* 1989;33:232-5.
 15. McCammon RL, Hilgenberg JC, Stoelting RK. Effect of propranolol on circulatory responses to induction of diazepam- nitrous oxide anesthesia and to endotracheal intubation. *Anesth Analg* 1981;60:579-83.
 16. Kulka PJ, Tryba M, Zenz M. Dose response effects of intravenous clonidine on stress response during induction of anaesthesia in coronary artery bypass graft patients. *Anesth Analg* 1995;80:263-8.
 17. Getler R, Brown CH, Mitchel H, Silvius N. Dexmedetomidine: A novel sedative analgesic agent. *Bayl Univ Med Cent Proc* 2001;14:13-21.
 18. Menda F, Koner O, Sayin M, Ture H, Imer P, Aykac B. Dexmedetomidine as an adjunct to anesthetic induction to attenuate haemodynamic response to endotracheal intubation in patients undergoing fast-track CABG. *Ann Card Anaesth* 2010;13:16-21.
 19. Yildiz M, Tavlan A, Tuncer S, Reisli R, Yosunkaya A, Otelcioglu S. Effect of dexmedetomidine on haemodynamic responses to laryngoscopy and intubation: Perioperative haemodynamics and anaesthetic requirements. *Drugs RD* 2006;7:43-52.
 20. Bloor BC, Ward DS, Belleville JP, Maze M. Effects of intravenous dexmedetomidine in humans. II. Hemodynamic changes. *Anesthesiology* 1992;77:1134-42.
 21. Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Young WL. *Miller's Anesthesia*. 7th ed. Philadelphia: Churchill Livingstone; 2014.
 22. Esra A, Celik M, Orhon Z, Yüzer S, Sen B. Different doses of dexmedetomidine on controlling haemodynamic responses to tracheal intubation. *Internet J Anaesthesiol* 2012;27.
 23. Keniya VM, Ladi S, Naphade R. Dexmedetomidine attenuates sympathoadrenal response to tracheal intubation and reduces perioperative anaesthetic requirement. *India J Anaesth* 2011;55:352-7.

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