

ORIGINAL ARTICLE

Comparison of Mean Hemodynamic Change with Different Doses of Intravenous Dexmedetomidine on Endotracheal IntubationTasneem Alam¹, Sanum Kashif^{2*}, Khalid Mehmood³, Umair⁴, Akhtar Hussain⁴**ABSTRACT**

Objective: To compare mean change in heart rate and arterial pressure in patients undergoing endotracheal intubation receiving 0.5 µg/kg dexmedetomidine versus 1.00 µg/kg dexmedetomidine.

Study Design: Randomized control trial.

Place and Duration of Study: The study was conducted in main Operation Theatre of Combined Military Hospital, Sialkot, Pakistan from 3rd February 2022 to 30th November 2022.

Materials and Methods: A total of 60 patients undergoing elective surgeries under general anesthesia using endotracheal intubation, aged 20–60 years of either gender were included. The included patients were divided into two groups using draw randomization. Randomization was done using lottery method. Group I patients received 0.5 µg/kg dexmedetomidine infusion and group II patients received 1.0 µg/kg dexmedetomidine infusion over 10 mins through a controlled infusion device. After endotracheal intubation, mean arterial pressure (MAP) and mean heart rate (HR) were noted after 3 minutes of intubation.

Non-probability convenience sampling was used for data collection and SPSS version 23 was used for data analysis.

Result: Out of 60 patients, 26 (43.33%) were males and 34 (56.67%) were females with male to female ratio of 1:1.3, the average age was 42.19 ± 9.87 years and BMI was found 28.88 ± 2.48 kg/m². In group I (0.5 µg/kg dexmedetomidine infusion), the mean HR and MAP before intubation was 108.43 ± 6.26 bpm, 112.20 ± 8.88 mmHg and after 3 minutes of intubation was 76.20 ± 4.16 bpm, 88.20 ± 3.93 respectively while in group II (1.0 µg/kg dexmedetomidine infusion), the mean HR and MAP before intubation 102.10 ± 10.20 bpm, 109.07 ± 8.35 mmHg and after 3 minutes of intubation was 85.97 ± 3.26 bpm, 97.90 ± 5.74 mmHg respectively.

Conclusion: Higher dose of dexmedetomidine (1.0 µg/kg) significantly attenuates hemodynamic response on endotracheal intubation as compare to lower dose (0.5 µg/kg).

Keywords: Dexmedetomidine, Endotracheal Intubation, Heart rate, Hemodynamic, Mean Arterial Pressure.

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Introduction

Health care providers must have a basic understanding of airway management, including the ability to intubate, especially in primary care settings in remote areas. Failure to secure a patent airway, causes inappropriate breathing and oxygenation, which causes hypoxic brain damage and death.¹ In order to maintain a secure airway while under general anesthesia, laryngoscopy and endotracheal intubation are often used procedures with specific purposes.² A painful stimulation brought on by endotracheal intubation triggers severe physiological reactions such as autonomic and activated brain stem reflexes.³ Laryngoscopy and

endotracheal intubation directly trigger a strong sympatho-adrenal reaction, which raises heart rate, catecholamine levels in the blood, arterial blood pressure, and in certain people, causes dysrhythmia.^{4,5}

Adrenergic blockers, vasodilators, calcium channel blockers, and alpha2 agonists have all been used to "blunt" this pressor response during tracheal intubation. Some researchers have utilized alpha2 agonists like clonidine and dexmedetomidine to reduce the stress response to laryngoscopy.^{6,7}

Dexmedetomidine is a highly selective agonist of the alpha 2-adrenergic receptors, with the potential to have sedative, sympatholytic, analgesic, and cardiovascular stability.⁸ When used in clinically effective doses, dexmedetomidine does not impair extubation by lowering respiratory rate.⁹ Dexmedetomidine has been shown to be beneficial in reducing the stress response to laryngoscopy and endotracheal intubation when administered at doses of 0.5 and 1 ug/kg in the literature.¹⁰

The aim of the study was to assess changes in mean heart rate and mean arterial pressure (MAP) in cases receiving two different dosages of dexmedetomidine (0.5 ug/kg versus 1.0 ug/kg) during endotracheal intubation.

Materials and Methods

A total of 60 American Society of Anesthesiology (ASA) I or II patients, 20-60 years, who were undergoing surgery under general anaesthesia with endotracheal intubation were included after receiving written informed consent and institutional review board approval (CMH Sialkot, ERC/1060/22). Patients having history of hypertension, diabetes mellitus, ASA III patients, those undergoing emergency surgery, and those with medication allergies were excluded. Using open-epi software, the sample size was determined by comparing the estimated MAPs of patients receiving 0.5 ug/kg of dexmedetomidine to those getting 1.0 ug/kg of dexmedetomidine at 3 minutes post-intubation: 101±9.1 mmHg versus 86±4.4 mmHg.

The included patients were divided into two groups using draw randomization. Randomization was done using lottery method. Patients were shifted to the operating room, once their NPO status was confirmed. Systolic, diastolic, mean arterial pressure and mean heart rate measurements were taken at

baseline.

Group I patients received 0.5 µg/kg dexmedetomidine infusion over 10 minutes and group II patients received 1.0 µg/kg dexmedetomidine infusion over 10 minutes through a controlled infusion device at induction, followed by intravenous atracurium 0.5mg/kg. After 3 minutes of giving muscle relaxant, ETT attempted.

After 3 minutes of ETT intubation, MAP and heart rate were recorded. Baseline patient's characteristics such age, gender, and BMI were recorded before intubation.

Data compilation and analysis was done using SPSS version 23.0. Mean ±standard deviation were calculated for quantitative variables like age, height, weight, BMI, MAP and heart. Frequency and percentage were calculated for gender and ASA status. Independent sample t-test was applied to compare of MAP and heart between the groups at 3 minutes after intubation.

Effect modifiers such as age, gender, ASA status and BMI were controlled by stratification. Post-stratification independent sample t-test was applied to determine the effect of these effect modifiers on MAP and mean heart rate at 3 minutes after intubation between the groups. P-value ≤0.05 was taken as significant.

Results

The age of patients ranged between 20 to 60 years, with mean age of 42.19 ± 9.87 years. Out of 60 patients, 26 (43.33%) were males and 34 (56.67%) were females. Mean BMI was 28.88 ± 2.48 kg/m² (Table 1).

Table 1: Patient’s characteristics of both groups (n=60)

Patient’s characteristics		Group I	Group II	Total
		(n=30)	(n=30)	(n=60)
		No (%)	No (%)	No (%)
Age (years)	20-40	14(46.67)	15(50.0)	29(48.3)
	41-60	16(53.33)	15(50.0)	31(51.67)
Gender	male	11(36.67)	15(50.0)	26(43.33)
	female	19(63.33)	15(50.0)	34(56.67)
BMI (kg/m ²)	<27	10(33.33)	12(40.0)	22(36.67)
	>27	20(66.67)	18(60.0)	38(63.33)
ASA	I	12(40.0)	14(46.67)	26(43.33)
	II	18(60.0)	16(53.33)	34(56.67)

In group I (0.5 µg/kg dexmedetomidine infusion), the mean HR and MAP before intubation was 108.43 ± 6.26 bpm, 112.20 ± 8.88 mmHg and after 3 minutes of intubation was 76.20 ± 4.16 bpm, 88.20 ± 3.93

respectively while in group II (1.0 µg/kg dexmedetomidine infusion), the mean HR and MAP before intubation 102.10 ± 10.20 bpm, 109.07 ± 8.35 mmHg and after 3 minutes of intubation was 85.97 ±

3.26 bpm, 97.90 ± 5.74 mmHg respectively, mean hemodynamic changes were statistically significant (<0.05) between groups (Table 2).

Table 2: HR and MAP after 3 minutes of intubation with respect to age, gender, BMI and ASA status

Effect modifiers		Group I (n=30) HR after 3 min (Mean± SD)	Group II (n=30) HR after 3 min (Mean± SD)	Group I (n=30) MAP after 3 min (Mean± SD)	Group II (n=30) MAP after 3 min (Mean± SD)	P-value
Age (years)	20-40	74.79±3.62	86.53±3.98	89.14±4.02	97.07±5.56	0.0001
	41-60	77.44±4.30	85.40±2.35	87.38±3.77	98.73±5.99	0.0001
Gender	Male	76.18±4.83	85.60±3.11	89.18±3.95	96.60±6.72	0.0001
	Female	76.21±3.85	86.33±3.48	87.63±3.90	99.20±4.43	0.0001
BMI (kg/m ²)	≤27	77.10±3.31	85.33±3.47	88.60±5.50	99.83±6.95	0.0001
	>27	75.75±4.53	86.39±3.15	88.0±3.01	96.61±4.54	0.0001
ASA status	I	75.08±3.2	85.50±2.41	87.92±3.23	96.14±4.54	0.0001
	II	76.94±4.62	86.38±3.90	87.39±4.71	99.44±6.37	0.0001

Table 3: Comparison of MAP and HR, before and after 3 minutes of intubation between groups

Heart Rate (bpm)	Group I (n=30) Mean ± SD	Group II (n=30) Mean ± SD	p-value
Before intubation	108.43 ± 6.26	102.10 ± 10.20	0.322
After 3 minutes	76.20 ± 4.16	85.97 ± 3.26	0.0001
Mean arterial pressure (MAP)	Group I (n=30) Mean ± SD	Group II (n=30) Mean ± SD	p-value
Before intubation	112.20 ± 8.88	109.07 ± 8.35	0.266
After 3 minutes	88.20 ± 3.93	97.90 ± 5.74	0.001

Discussion

A highly selective alpha-2 adrenergic agonist, dexmedetomidine has sedative, anxiolytic, and analgesic effects that are dose dependant. Dexmedetomidine's effect is well established for lowering sympathetic tone and maintaining hemodynamic stability, if given pre-operatively.¹¹ The efficacy of dexmedetomidine on hemodynamic response during laryngoscopy and tracheal intubation have been studied in various studies. Due to the sympathetic adrenergic output triggered by laryngeal tissue stimulation, laryngoscopy and tracheal intubation, are risky procedures that may result in tachycardia, hypertension, or arrhythmia. Furthermore, elderly patients with concomitant diseases are more likely to experience perioperative problems due to this hemodynamic instability, which can have disastrous consequences including

myocardial ischemia, cardiac arrhythmia, or a cerebrovascular accident.¹² As a result, anesthesiologists have employed a variety of drugs to lessen the adrenergic responses. However, many of the research on this subject had been limited to normotensive patients due to lack of hemodynamic stability in geriatric population.¹³

The current study was conducted to compare changes in mean heart rate and mean arterial pressure (MAP) in patients undergoing endotracheal intubation receiving 0.5 µg/kg dexmedetomidine versus 1.00 µg/kg dexmedetomidine and found that the mean HR and MAP before intubation in group I was 108.43 ± 6.26 bpm, 112.20 ± 8.88 mmHg and after 3 minutes of intubation was 76.20 ± 4.16 bpm, 88.20 ± 3.93 respectively while in group II, the mean HR and MAP before intubation 102.10 ± 10.20 bpm, 109.07 ± 8.35 mmHg and after 3 minutes of intubation was 85.97 ± 3.26 bpm, 97.90 ± 5.74 mmHg respectively.

A study conducted by Jarineshin et al. reported that 0.5 µg/kg dexmedetomidine has similar effects on hemodynamics as that of 1 µg/kg of dexmedetomidine. In their study mean arterial pressure (MAP) after 3 minutes of intubation was 82.0 ± 5.2 mmHg in 0.5 µg/kg versus 79.5 ± 5.5 mmHg in patients receiving 1.0 µg/kg dexmedetomidine group. Mean heart rate in their study was 70.9 ± 9.2 beats/min in 0.5 µg/kg dexmedetomidine versus

66.1 ± 7.1 beats/min in 1.0 µg/kg dexmedetomidine group.¹⁴ While Smitha et al. concluded that 1.0 µg/kg dexmedetomidine is more effective than 0.5 µg/kg dexmedetomidine. In their study, MAP after 3 minutes of intubation was 101±9.1 mmHg in 0.5 µg/kg dexmedetomidine versus 86±4.4 mmHg in 1.0 µg/kg dexmedetomidine group, and mean heart rate was 85±9.1 beats/min in 0.5 µg/kg dexmedetomidine versus 71±6.0 beats/min in 1.0 µg/kg dexmedetomidine group.¹⁵ The mean age of patients, may be an important difference between our study and Smitha's research. The mean age in the first study was 29 years old, but the mean age in the subsequent study ranged between 39 and 42 years old.

Dexmedetomidine dosages of 0.25 to 2 µg/kg have been employed by researchers in earlier studies. Although dexmedetomidine's effects are known to be dose-dependent, side effects like bradycardia and hypotension are also more likely to occur at higher doses. As a result, it's crucial to modify the dose in the best way possible based on the characteristics of the patient.¹⁶ The hemodynamic response to intubation and extubation was lessened by a single dose of dexmedetomidine before anaesthesia induction, according to Kumari K et al.¹⁷ However, they used 2 µg/kg of the drug, and hypotension and bradycardia were found to occur more frequently in the dexmedetomidine group than in the control group. The studies listed above, however, involved young, normotensive people. Age-related pharmacodynamic changes necessitate dose decrease in the elderly.

In a meta-analysis of Piao and Wu, on evaluation of dexmedetomidine as an anaesthetic agent, it was discovered that the incidence of negative effects like hypotension and bradycardia with dexmedetomidine was significantly higher as compared to controls. In their comparative study of dexmedetomidine doses of 1.0 µg/kg and 0.5 µg/kg, they reported a higher incidence of hypotension and bradycardia with the higher dose.¹⁸

In contrast to the other two teams, Khan AA et al. demonstrated that 1 µg/kg of dexmedetomidine dramatically lowered all hemodynamic parameters, including SBP, DBP, and MAP. The group getting 1 µg/kg of dexmedetomidine showed a significant difference from the other two teams. Two dosages of

dexmedetomidine (0.5 µg/kg and 1 µg/kg) were used in this investigation, and they reduced SBP, DBP, and MAP in comparison to the control group.¹⁹

Srivastava et al. compared 20 ml of 0.9% normal saline (Group C), 1 mcg/kg of dexmedetomidine (Group D), and 1.5 mg/kg of esmolol (Group E). Their research found that dexmedetomidine 1mcg/kg was more effective at reducing hemodynamic stress response than esmolol 1.5mg/kg. In a study by Reddy et al., the attenuation of the pressor response to laryngoscopy and tracheal intubation was examined in relation to intravenous dexmedetomidine (1mcg/kg) infusion, esmolol (2mg/kg) intravenous infusions, and placebo. Dexmedetomidine 1.0 mcg/kg was found to be dominant when compared to esmolol 2 mg/kg in the study's conclusion for delivering a dependable and consistent blunting of sympathoadrenal response.²⁰

Conclusion

Higher dose of dexmedetomidine (1.0 µg/kg) significantly attenuates hemodynamic response during endotracheal intubation as compare to lower dose (0.5 µg/kg).

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