*Original Research Article*

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# A Comparative Study of Dexmedetomidine HCL and Esmolol HCL for Attenuating Pressor Response to Laryngoscopy and Oral Endotracheal Intubation

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**Abstract**

*Introduction:* Laryngoscopy and intubation increases sympathetic activity resulting in tachycardia and hypertension which may cause myocardial ischemia, cardiac arrhythmias and cerebrovascular hemorrhage. We compared the efficacy of intravenous esmolol and dexmedetomidine to attenuate the pressor response to laryngoscopy and intubation. *Design:* Randomised controlled trial. *Method:* Seventy-five patients of ASA I and ASA II undergoing general anesthesia with oral intubation for elective surgery were allocated into three groups. *Group C:* Control group. *Group E:* intravenous esmolol 2mg/kg three minutes before laryngoscopy. *Group D:* Intravenous dexmedetomidine 1µg/kg in 100ml infusion over ten minutes before laryngoscopy. Heart rate, systolic, diastolic and mean arterial pressures were recorded before drug administration, after drug administration, after induction of standard anesthesia, immediately after intubation, every 2 minutes till 10 minutes. Incidence of bradycardia and hypotension were noted. *Statistical Analysis used:* IBM SPSS version. *Results:* Mean HR immediately after intubation for group C was (104.57±8.2) whereas it was (83±4.53, 92.6±4.70) for group D and group E with p value of (0.003,0.002) when compared to control group. While comparing group D and E, p value was 0.0001. MAP immediately after intubation for group C was (109±5.9) whereas it was (98±3.4, 100.2±5.2) for group D and E with p value (0.0001, 0.002) when compared to control group. While comparing group D and E, p value was 0.006. *Conclusion:* Dexmedetomidine and esmolol both attenuated the pressor response to laryngoscopy and intubation but it was better controlled with dexmedetomidine.

**Keywords:** Pressor Response; Laryngoscopy; Esmolol; Dexmedetomidine.

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## Introduction

Laryngoscopy and tracheal intubation leads to increase in arterial blood pressure and heart rate, magnitude of which depends on various factors like depth of anaesthesia, use of any measures prior to airway manipulation, the anaesthetic agent used and the duration of laryngoscopy and intubation. The principle mechanism behind this is the sympathetic response resulting from increased catecholamine activity which is usually transitory, variable, unpredictable and hazardous to patients with hypertension, myocardial insufficiency or cerebrovascular diseases and predisposes to development of pulmonary edema, myocardial insufficiency and cerebrovascular accident [1,2].

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Intravenous anesthetic induction agents do not adequately or predictably suppress these responses. So reseachers have used different pharmacological measures like use of volatile anesthetics, topical and intravenous lidocaine, opioids, clonidine, nitroglycerine, calcium channel blockers and β-blockers prior to initiating laryngoscopy to blunt this response [3-9]. An ideal pharmacological agent would be one which minimizes these responses, prevent impairment of cerebral and coronary blood flow, avoid awareness and does not interfere recovery from anesthesia. Its administration and onset of action should not be time consuming also it should minimally affect the duration or modality of the ensuing anesthesia. Here we had chosen alpha 2 agonist and short acting beta blocker for blunting pressor response and compared their efficacy. α2 agonists are being used for attenuating the pressor response and amongst them dexmedetomidine appears to fulfill all the above criteria and is highly specific and selective α2 adrenoceptor agonist with α2: α1 binding selectivity ratio of 1620:1 compared to 220:1 for clonidine [10]. Its advantages include sedation, analgesia, anxiolysis and improved haemodynamic stability. It produces hyperpolarization of noradrenergic neurons and suppression of neuronal firing in the locus cerelous which leads to decreased systemic noradrenaline release resulting in attenuation of sympatho-adrenal responses and hemodynamic stability during laryngoscopy and tracheal intubation [10].

Esmolol decreases the force of contraction and heart rate by blocking action of catecholamines on beta-adrenergic- 1 receptors of the sympathetic nervous system, mainly found in the heart thus attenuates the tachycardia and hypertensive responses to laryngoscopy and endotracheal intubation [11].

*Aims and Objective*

In this study we compared the efficacy and safety of dexmedetomidine and esmolol in blunting pressor response to laryngoscopy and endotracheal intubation.

## Materials and Methods

After approval from the Institutional Ethical Committee, 75 patients of ASA I and II, from age group of 18 - 60 years undergoing surgery under general anesthesia requiring oral intubation were selected and allocated into three groups (25 patients in each group). Written informed consent was obtained from each patient. Patients with ASA III and IV, anticipated difficult intubation, history of drug reaction with study drugs and patients on beta blockers or á agonists were excluded from the study.

After taking patient in the operation theatre, baseline vital parameters including heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), oxygen saturation ($SpO_2$) and electrocardiogram (ECG) were recorded. Intravenous access was secured. All the patients were premediated with IV Inj. Ondansetron 0.08 mg/kg, Inj. Ranitidine 1 mg/kg and Inj. glycopyrrolate 0.004 mg/kg. Study drugs were given as following:

*Group C:* Control group – 100ml normal saline infusion over 10 minutes followed by 10 ml normal saline 3 minutes before laryngoscopy.

*Group E:* 100 ml normal saline infusion over 10 minutes followed by intravenous esmolol 2mg/kg diluted in 10 ml normal saline 3 minutes before laryngoscopy.

*Group D:* Intravenous dexmedetomidine 1ìg/kg in 100 ml normal saline infusion over 10 minutes followed by 10 ml normal saline 3 minutes before laryngoscopy

HR, SBP, DBP and MAP were recorded before drug administration, after drug administration, after induction of anesthesia, immediately after intubation and every 2 minutes till ten minutes and then at every 5 minutes interval. The patients were blinded to the treatment group and all data was collected by same anesthesiologist blinded to the group allocation. Induction of anesthesia was performed with Inj. thiopentone 5 mg/kg and Inj. Succinylcholine 2mg/kg intravenously. Then patients were ventilated manually with 100% oxygen.

Laryngoscopy and intubation was done by same experienced anesthesiologist with appropriate size-cuffed disposable portex endotracheal tracheal tube (No. 8 for males and 7 for females) within 15-20 seconds in all patients. Failure to intubate within this period was excluded from this study. Anesthesia was maintained with 50:50 Oxygen and Nitrogen dioxide mixture, 1% sevoflurane and Inj. Vecuronium 0.08 mg/kg/hour. Patients were ventilated with tidal volume of 6-8 ml/kg at frequency of 12 to 14/ minute. No surgical intervention was allowed till the study period of 10 min. Incidence of bradycardia and hypotension were noted. Decrease in MAP greater than 20% below the baseline value or SBP less than

90 mm of Hg was considered as hypotension and was treated by increasing the IV fluid infusion rate and then reducing sevoflurane concentration or incremental dose of Inj. Phenylephrine 0.1 mg bolus IV if necessary. Decrease in HR (<50 beats/min) was treated with atropine 0.6 mg IV.

*Statistics:* Groups were compared for demographic data (age, weight) and hemodynamic parameters by one-way analysis of variance. Paired t-test was used for comparison among the groups, while for comparison within the groups unpaired t-test was used. Statistical analysis was done by IBM SPSS version. Probability was considered to be significant if less than 0.05. Data are represented as mean± SD.

*Observations*

All the 75 patients completed the study. The demographic profile of the patients in terms of age, body weight, male:female ratio, ASA status, Mallampati Class were comparable and there were no significant differences among the three groups ($p > 0.05$) as shown in Table 1.

## Results

Following laryngoscopy and tracheal intubation, HR and blood pressure increased immediately after intubation from the baseline in Group C which is statistically significant ( $p<0.001$). But increase in Group E and D was not statistically significant.

*Inter group variation*

The rise in HR was 15% in Group C, 5 % in Group E and 2% in group D. This rise was significantly lower in Groups E and D compared to Group C at all-time intervals ($p<0.01$).

There was significant difference in rise of heart rate between Groups E and D (p - 0.01).

Blood pressure (SBP, DBP and MBP) increased immediately after intubation from the baseline in group C which was statistically significant ($p<0.001$).

The rise in MAP was 11% in Group C, 6 % in Group E and 2 % in Group D. A significant rise in MAP was observed up to 10 minutes post-intubation in Group C. In between Groups C and D, between Group C and E and between Groups E and D, the differences were significant at all-time intervals ($p<0.01$).

*Intra Group Variation*

There was significant rise in heart rate and blood pressure between baseline and immediately after intubation in Group C (p Value- 0.0003 for heart rate, 0.0004 for SBP, 0.002 for DBP and 0.001 for MAP), whereas in group E and D there was no significant rise between baseline and immediately after intubation (p – 0.2 for HR, 0.3 for SBP, 0.1 for DBP and 0.2 for MAP ) in Group E and (p- 0.1 for HR, 0.2 for SBP, 0.4 for DBP and 0.3 for MAP ) in Group D. Hypotension was seen in 1 case (4 %) in Group D and 2 cases (8%) in Group E. Bradycardia

**Table 1:** Patient's characteristics

| | Group E | Group C | Group D | P Value (E/C) | P Value (D/C) | P Value (D/E) |
|---|---|---|---|---|---|---|
| Age | 48.44±13.78 | 45.7±12.76 | 43.76±14.21 | 0.19 | 0.21 | 0.24 |
| Sex (M:F) | 15/10 | 16/9 | 15/10 | | | |
| Weight | 63.64±9.50 | 68±8.56 | 58.8±11.49 | 0.17 | 0.19 | 0.11 |

Abbreviations: M: Male, F: Female

**Table 2:** Changes in Heart Rate

| HR (BPM) | Group D (Mean± SD) | Group E (Mean± SD) | Group C (Mean± SD) | P Value | P Value D/E | P Value D/C | P Value E/C |
|---|---|---|---|---|---|---|---|
| Baseline | 88.6± 8 | 88.5± 5.31 | 88.5 ± 7.99 | 0.99(NS) | >0.05(NS) | >0.05(NS) | >0.05(NS) |
| Immediately after study drug injection | 75.7± 7.05 | 82.8±5.45 | 88± 7.3 | <0.0001 | <0.001 | <0.001 | >0.05(NS) |
| After induction | 70.9±5.2 | 78.2±5.20 | 87.3 ± 6.6 | <0.0001 | <0.001 | <0.001 | <0.001 |
| After intubation immediately | 83± 4.53 | 92.6 ±4.70 | 104.57 ± 8.2 | <0.0001 | 0.0001 | 0.003 | 0.002 |
| 3RD Minute | 85.5±5.58 | 99 ± 4.04 | 119.8 ± 8.06 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 5TH Minute | 80 ±5.4 | 92.7 ±3.10 | 113.52 ± 6.16 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 7TH Minute | 74.36± 4.5 | 88.12 ± 3.8 | 107.16 ± 6.16 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 10TH Minute | 73 ±3.7 | 86.3±4.04 | 98 ± 5.5 | <0.0001 | <0.001 | <0.001 | <0.001 |

**Table 3:** Changes in Systolic Blood pressure

| SBP (mmHg) | Group D (Mean±SD) | Group E (Mean±SD) | Group C (Mean±SD) | P Value | P Value D/E | P Value D/C | P Value E/C |
|---|---|---|---|---|---|---|---|
| Baseline | 130± 10 | 130.4±5.8 | 130.92±9.9 | 0.06 | >0.05(NS) | >0.05(NS) | >0.05(NS) |
| Immediately after study drug injection | 113.2± 8.2 | 121.3± 4.93 | 132± 9.05 | <0.0001 | <0.01 | <0.001 | >0.05(NS) |
| After induction | 108.16±10.4 | 115.3±5.2 | 126.72± 7.8 | <0.0001 | <0.01 | <0.001 | >0.05(NS) |
| After intubation immediately | 118 ± 3.42 | 135.1 ±4.08 | 150 ± 6.13 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 3RD Minute | 115± 6.31 | 137.36± 3.8 | 144 ± 5.45 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 5TH Minute | 111±7.6 | 132.3± 3.83 | 140.1 ± 5.43 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 7TH Minute | 106.5± 7.2 | 128.3±4.2 | 135.5± 5.7 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 10TH Minute | 104± 9.2 | 122.9± 3.71 | 130.7± 5.02 | <0.0001 | <0.001 | <0.001 | <0.001 |

**Table 4:** Changes in Diastolic Blood Pressure

| DBP (mmHg) | Group D (Mean±SD) | Group E (Mean±SD) | Group C (Mean±SD) | P Value | P Value (D/E) | P Value (D/C) | P Value (E/C) |
|---|---|---|---|---|---|---|---|
| Baseline | 79.64± 7.8 | 78.16± 7.9 | 79.6± 7.8 | 0.70(NS) | >0.05(NS) | >0.05(NS) | >0.05(NS) |
| Immediately after study drug injection | 68.6± 5.9 | 74.12± 2.43 | 79.5±8.70 | <0.0001 | <0.01 | <0.001 | <0.05 |
| After induction | 65.5± 5.3 | 71.92± 3.25 | 76.36± 7.15 | <0.0001 | <0.001 | <0.001 | <0.05 |
| After intubation immediately | 75.24± 4.89 | 90.2± 4.02 | 102.96±5.08 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 3RD minute | 73± 5 | 91.9± 2.67 | 104.9±3.96 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 5TH minute | 69.04± 4.92 | 87 ± 3.3 | 96.56± 3.24 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 7TH minute | 67.24± 6.3 | 82.4± 3.02 | 92.6±2.81 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 10TH minute | 68.6± 6.23 | 79.6± 2.3 | 89.32± 3.73 | <0.0001 | <0.001 | <0.001 | <0.001 |

**Table 5:** Changes in Mean Arterial Blood Pressure

| MBP (mmHg) | Group D (Mean±SD) | Group E (Mean±SD) | Group C (Mean±SD) | P Value | P Value (D/E) | P Value (D/C) | P Value (E/C) |
|---|---|---|---|---|---|---|---|
| Baseline | 96.6± 7.31 | 95.48± 6.01 | 96.64± 7.31 | 0.75(NS) | >0.05(NS) | >0.05(NS) | >0.05(NS) |
| Immediately after study drug injection | 83.64± 5.6 | 89.92± 2.58 | 96.96±7.31 | <0.0001 | <0.01 | <0.001 | <0.05 |
| After induction | 79.96±5.70 | 86.36 ± 2.99 | 91.2± 5.74 | <0.0001 | <0.001 | <0.001 | <0.05 |
| After intubation immediately | 98± 3.4 | 100.2±5.2 | 109±5.9 | <0.0001 | 0.006 | 0.0001 | 0.002 |
| 3rd minute | 83.2± 4.14 | 107.04±2.55 | 117.5± 3.72 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 5th minute | 80.2± 5.02 | 100.32±2.70 | 110.52±2.56 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 7th minute | 81.16± 5.89 | 95.28± 2.7 | 106.96±2.89 | <0.0001 | <0.001 | <0.001 | <0.001 |
| 10th minute | 80.52± 6.65 | 91.68±2.51 | 104.6±3.08 | <0.0001 | <0.001 | <0.001 | <0.001 |

*Abbreviations:*
D – Dexmedetomidine
E – Esmolol
C – Control
SD – Standard Deviation

was seen in 2 cases (8%) and 3 cases (12%) in Group D and E respectively. All were treated appropriately.

## Discussion

Laryngoscopy and endotracheal intubation are considered as most crucial procedures during induction of general anesthesia because they lead to a transient yet marked sympatho-adrenal response [1]. Healthy individuals may tolerate this response but patients with cardiovascular compromise, cerebrovascular disease and intracranial aneurysm may not tolerate these transient changes in hemodynamics which may result in ventricular failure, pulmonary edema, myocardial ischemia, arrhythmias and intracranial bleeds. This response usually lasts for 7- 10 minutes [2], this observation was concomitant with our finding of increase in hemodynamics till approximately 10 minutes in control group.

The hemodynamic response to this stimulus is due to the intense sympathetic discharge caused by stimulation of oro-laryngopharynx. Also placing the endotracheal tube through vocal cords and inflating the cuff in infra-glottic region contributes to sympatho-adrenal response.

Dexmedetomidine acts on α adrenoreceptors that are involved in regulating the autonomic nervous system and cardiovascular system. α2 receptors are located on blood vessels and central sympathetic presynaptic terminals. Stimulation of these receptors leads to reduction in central sympathetic outflow, augmentation of vagal activity and sedation which results in decrease in HR and cardiac output and in turn decreases blood pressure [12].

Dexmedetomidine has been used for attenuating pressor response by many authors at doses in range of 0.5 to 1 micrograms/kg [12-21]. But higher dose may be associated with hypotension and bradycardia. Rapid and bolus administration can cause tachycardia, bradycardia and hypertension followed by hypotension [15]. We used 1microgram/kg dexmedetomidine over 10 min infusion, yet encountered two cases of bradycardia.

A biphasic cardiovascular response of transient increase in blood pressure and reflex bradycardia is seen in healthy patients due to α2 receptor stimulation of vascular smooth vessels. This can be decreased by slow infusion. Apart from this effect, dexmedetomidine also seems to decrease adverse cardiac events like myocardial infarction by decreasing α receptor stimulation thus modulating coronary blood flow [13,15]. In our study we did not encounter any cardiac events except from bradycardia in 2 patients.

Siddareddigari Velayudha Reddy et al. [16] did similar study between dexmedetomidne 1 microgram/kg and esmolol 2 mg/kg for their efficacy in blunting pressor response and found that mean increase in HR was minimal in Group D than Group E and control group immediately after intubation and mean HR was not significantly increased in Group D at any time interval. While comparing the effect on MAP, Group D had lower MAP immediately after intubation till end of surgery whereas esmolol has less effect on control of MAP. They also showed decrease in requirements of anesthetic agents in both groups, more in dexmedetomidine group .

Esmolol has various properties like cardio-selectiveness, ultrashort acting, less drug interactions with commonly used anesthetics which makes it a valuable agent [11]. Many studies [3,4,5,9,15,16,18,22] have proved that esmolol has solely or predominantly negative chronotropic effect and less effect on blood pressure. In accordance to this our study also reflected that esmolol at 2 mg/kg was more effective in blunting the rise of HR than MAP.

Arti rathore et al. [22] used esmolol at different doses of 50 mg, 100mg, 150mg and demonstrated prevention of rise in heart rate in dose response manner. Decrease in MAP was significant only at higher dose with single incidence of treatable bradycardia. We used esmolol at 2 mg/kg which caused fewer side effects of bradycardia and hypotension.

Ajay Gupta et al. [5] compared esmolol 1.5 mg/kg with lignocaine 1.5 mg/kg and found that esmolol significantly attenuated HR for a maximum duration of 2 minutes after intubation. In contrast to this, our study suggested mild increase of 5% from baseline after intubation and later maintenance of heart rate at around baseline HR till 10 minutes.

Researchers have used invasive arterial blood pressure for monitoring exact fluctuations in the blood pressure. Usual non-invasive blood pressure monitoring requires an average of 40 seconds to measure blood pressure by oscillatory method, but the pressor response is a continuous process (maximum within 1 minute) which requires continuous monitoring [18]. We did not use invasive BP monitoring which may be the limitation of our study. This study was done on ASA I and II so effects in high risk patients could not be seen.

## Conclusion

Evaluation of baseline and immediately after intubation value of hemodynamic parameters revealed a greater percentage variation in MAP in the esmolol and control groups as compared to the dexmedetomidine group. While considering variations in mean heart rate, attenuation was seen in both the study groups D and E. This suggests that both esmolol and dexmedetomidine blunts rise in heart rate but rise in blood pressure is better controlled with dexmedetomodine. Within the constraints of this study we demonstrated that administration of a single dose of dexmedetomidine before general anesthesia induction was an effective method for attenuating the hemodynamic response to endotracheal intubation.

## Acknowledgement

*Conflict of Interest* No

*Key Messages*

Pressor response attenuation during laryngoscopy and oral intubation is very important aspect specially in compromised patients with cardiac and neurological diseases. Appropriate pharmacological agent is being sought for and dexmedetomidine seems to be promising.

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