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# Attenuation of Hemodynamic Responses to Laryngoscopy and Endotracheal Intubation: Comparison of Esmolol and Lignocaine for Elective Surgeries Under General Anaesthesia

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**Abstract**
We compared the effects of intravenous esmolo land lignocaine as premedicantin attenuation of hemodynamic response to laryngoscopy and intubation in normotensive patients undergoing elective surgery. A total of 60 patients undergoing general anesthesia were enrolled in the study and were randomly allocated into two groups of 30 each. Group 1 patients received intravenous esmolol 1mg/kg 3 minutes before intubation and Group 2 patients received intravenous lignocaine 1.5 mg/kg 3 minutes before intubation. Both groups were matched for age, sex, BMI and ASA grade. The highest HR recorded at the time of intubation was 90.2±12.4(3.2% above baseline) and 106.2±15.6(26.5% above baseline) in group1 and 2 respectively. This was statistically significant (p value-0.010). With respect to SBP, DBP and MAP there was no statistically significant difference between the two groups. Intravenous esmolol 1mg/kg given 3 minutes before intubation was found to be more effective in controlling heart rate to laryngoscopy and intubation compared to intravenous lignocaine(1.5mg/kg) given 3 minutes before intubation and with respect to systolic, diastolic and mean arterial pressures both the drugs were comparable.

## INTRODUCTION

Laryngoscopy and intubation is a basic skill all anaesthesiologists' possess. Laryngoscopy and endotracheal intubation are potent stimuli that can induce increased sympathetic activity leading to tachycardia, hypertension and dysrhythmias. It may have deleterious respiratory, neurological and cardiovascular effects[1,2]. In attenuating these responses and maintaining stable haemodynamics lies the fine skill of the anaesthesiologist.

## MATERIALS AND METHODS

Institutional ethical committee approval was taken for this prospective, randomised, double blind study. Written informed consent was obtained from the patients. The study was conducted in 60 subjects aged between 15-65 years, ASA I and II patients undergoing elective surgeries under General Anaesthesia. Patients with Diabetes, Hypertension, Cardiovascular, respiratory, or neurological disorders and patients on beta-blockers or calcium channel blockers were excluded.

All the patients included in the study was given Tab. Alprazolam 0.5 mg and Tab. Pantoprazole 40 mg orally, on the night before surgery and was kept nil per oral 6 hours for solids and 2 hours for clear fluids before induction. On arrival to the operation theatre (O.T), intravenous(IV) access was taken using 18G intravenous cannula on the non-dominant hand and an infusion of normal saline was started. The patient was connected with the multi-parameter monitor, which records heart rate (HR), non- invasive measurements of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial presuure (MAP), continuous ECG monitoring and oxygen saturation ($SpO_2$). The baseline systolic blood pressure, diastolic blood pressure, mean arterial pressure, heart rate and oxygen saturation were recorded. All the patient were pre-oxygenated with 100% oxygen for 3 minutes and pre-medicated with Inj. Midazolam (0.02 mg/kg body weight), Inj. Glycolpyrrolate (0.01 mg/kg bodyweight), Inj. Fentanyl (1.5 mcg/kg body weight), Inj. Ondansetron (0.1mg/kg body weight) three minutes before induction. Group 1 was given intravenous Esmolol (1 milligram per kilogram) three minutes before intubation. Group 2 was given intravenous Lignocaine (1.5 milligram per kilogram) three minutes before intubation. Induction of general anaesthesia for all patients was done with Injection propofol 2mg/kg. This will be followed by an intubating dose of injection vecuronium bromide 0.1mg/kg. Patients then were bag and mask ventilated for three minutes. Endotracheal intubation was done using appropriate endotracheal tube cuffed. After confirming bilateral equal air entry, cuff inflated, tube fixed, connected to circuit and positive pressure ventilation will be continued. Recordings of HR, SBP, DBP, MAP and $SPO_2$ were taken at basal (before premedication), after induction, at laryngoscopy and intubation and at 1 min, 5min and 10 min post intubation. Anaesthesia was maintained with Oxygen (33%)+Nitrous oxide (66%) + volatile anesthetic (sevoflurane or isoflurane) and further neuromuscular blockade was maintained by vecuronium bromide at dose of 0.01mg/kg and IPPV. Adequacy of ventilation was monitored clinically and $SpO_2$ monitoring.

## RESULTS AND DISCUSSIONS

Table 1 shows demographic data-age, BMI, gender and ASA grade which were comparable between Group 1 and Group 2.

The highest HR recorded at the time of intubation in group 1 was 90.2±12.4(3.2% above baseline) and 106.2±15.6(26.5% above baseline) in group 2. This was statistically significant with p value-0.010. Thus showing that esmolol controlled the heart rate better than lignocaine at intubation. The differences later on were comparable between the 2 groups.

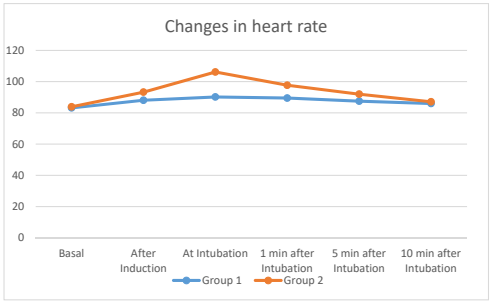


Fig. 1: Changes in heart rate

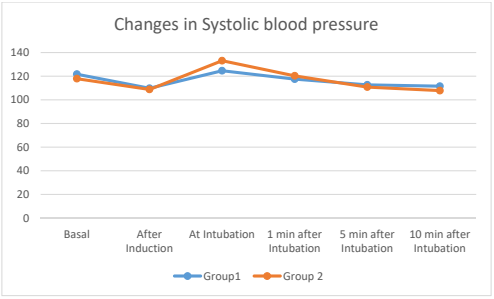


Fig. 2: Changes in systolic blood pressure

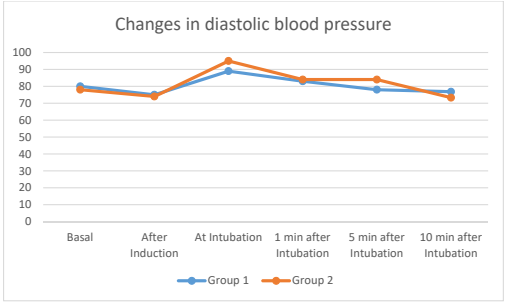


FIG. 3: Changes in diastolic blood pressure

**Table 1: Demography of the patients**

| Parameters | Group 1 | Group 2 | p-value |
|---|---|---|---|
| No. of patients | 30 | 30 | - |
| Age (years) | 22.87± 5.204 | 22.87± 5.204 | 1.00 |
| BMI(Kg/m2) | 24.67± 1.373 | 24.00± 1.554 | 0.236 |
| Sex (M/F) | 15/15 | 15/15 | - |
| ASA Grade I | 23 | 24 | 0.935 |
| ASA Grade II | 7 | 6 | |

**Table 2: Comparison of heart rate**

| | Group 1 | | | Group 2 | | | p-value |
|---|---|---|---|---|---|---|---|
| | Mean | SD | % Diff | Mean | SD | % Diff | |
| Basal | 83.2 | 15.6 | -- | 83.9 | 14.8 | - | 0.089 |
| After Induction | 88.1 | 13.2 | 2.2 | 93.3 | 15.9 | 11.2 | 0.046 |
| At Intubation | 90.2 | 12.4 | 3.2 | 106.2 | 15.6 | 26.5 | 0.010 |
| 1 min after Intubation | 89.5 | 11.0 | 2.8 | 97.7 | 15.3 | 16.4 | 0.056 |
| 5 min after Intubation | 87.5 | 12.3 | 2.1 | 92.0 | 13.5 | 9.6 | 0.511 |
| 10 min after Intubation | 86.0 | 12.2 | 1.06 | 87.1 | 13.9 | 3.8 | 1.00 |

**Table 3: Comparison of SBP**

| | Group 1 | | | Group 2 | | | p-value |
|---|---|---|---|---|---|---|---|
| | Mean | SD | % Diff | Mean | SD | % Diff | |
| Basal | 121.7 | 10.9 | -- | 117.9 | 9.6 | - | 0.529 |
| After Induction | 109.8 | 12.4 | -9.8 | 108.8 | 10.5 | -7.7 | 1.00 |
| At Intubation | 124.6 | 13.8 | 2.4 | 133.1 | 16.2 | 12.9 | 0.130 |
| 1 min after Intubation | 117.5 | 12.5 | -3.5 | 120.3 | 16.0 | 2.0 | 1.00 |
| 5 min after Intubation | 112.7 | 10.0 | -7.4 | 110.8 | 13.9 | -6.0 | 1.00 |
| 10 min after Intubation | 111.5 | 9.3 | -8.4 | 107.8 | 12.6 | -8.6 | 0.637 |

**Table 4: Comparison of DBP**

| | Group 1 | | | Group 2 | | | p-value |
|---|---|---|---|---|---|---|---|
| | Mean | SD | % Diff | Mean | SD | % Diff | |
| Basal | 80 | 8 | | 78 | 8 | | 1.00 |
| After Induction | 75 | 10 | -6.3 | 74 | 11 | -5.1 | 1.00 |
| At Intubation | 89 | 11 | 11.3 | 95 | 17 | 21.8 | 0.286 |
| 1 min after Intubation | 83 | 8 | 3.8 | 84 | 13 | 7.7 | 1.00 |
| 5 min after Intubation | 78 | 8 | -2.5 | 84 | 13 | 7.7 | 0.419 |
| 10 min after Intubation | 76.8 | 7.4 | 4 | 73.3 | 13.0 | 6.02 | 0.633 |

**Table 5: Comparison of MAP**

| | Group 1 | | | Group 2 | | | p-value |
|---|---|---|---|---|---|---|---|
| | Mean | SD | % Diff | Mean | SD | % Diff | |
| Basal | 94 | 9 | | 91 | 8 | | 0.928 |
| After Induction | 87 | 11 | -7.4 | 85 | 11 | -6.6 | 1.00 |
| At Intubation | 101 | 12 | 7.4 | 108 | 16 | 18.7 | 0.241 |
| 1 min after Intubation | 94 | 10 | 0.0 | 93 | 14 | 2.1 | 0.865 |
| 5 min after Intubation | 90 | 9 | -4.3 | 86 | 13 | -5.5 | 0.714 |
| 10 min after Intubation | 88 | 8 | -6.4 | 85 | 13 | -6.6 | 0.534 |

The baseline SBP in group 1 was 121.7±10.9 mm Hg, which dropped to 109.8±12.4 mm Hg with induction and later increased to 124.6±12.4 mm Hg at intubation and was the maximum SBP recorded following which there was gradual fall. In group 2, baseline SBP was 117.9±9.6 mm Hg which dropped to 108.8±10.5 mm Hg with induction and increased at intubation to 133.1±16.2 mm Hg which was the maximum SBP recorded and gradually decreased to 107.8±12.6 mm Hg after 10 minutes. There was no statistically significant difference between the two groups.

In group 1, baseline DBP was 80±8 mm Hg which dropped to 75±10 mm Hg with induction and increased at intubation to 89±11 mm Hg which was the maximum SBP recorded and gradually decreased to 76.8±7.4 mm Hg after 10 minutes. The baseline DBP in group 2 was 78±8 mm Hg, which dropped to 74±11 mm Hg with induction and later increased to 95±17 mm Hg at intubation and was the maximum DBP recorded following which there was gradual fall and 73.3±13.0 mm Hg after 10 minutes. Diastolic pressures were comparable in group 1 and group 2.

In group 1, baseline MAP was 94±9 mm Hg and it decreased with induction to 87±11 mm Hg and increased at intubation to 101±12 mm Hg and finally settled down to 88±8 mm Hg after 10 minutes. The baseline MAP in group 2 was 91±10 mm Hg and decreased at induction to 85±11 mm Hg. With intubation it rose to 92±16 mm Hg and gradually decreased and at 10 minutes it was 77±11 mm Hg. MAP in both groups were comparable.

Intravenous lignocaine is one of the most commonly used drugs to attenuate intubation response. Esmolol hydrochloride is a relatively new cardioselective, i.v. beta adrenoceptor antagonist. It has a rapid onset of action, exerts a peak haemodynamic effect within minutes and possesses a short elimination half-life of 9 min[3]. Consequently, it should prove ideal for control of the short-lived haemodynamic sequelae associated with laryngoscopy and intubation[4]. Hence, we chose to compare the two

drugs with respect to intubation response attenuation.

In our study, the heart rate increased by 3.2% in esmolol group as compared to 26.5 % in lignocaine group at intubation with a p value-0.010 which was statistically significant. So, heart rate in patients receiving esmolol was better controlled as compared to patients receiving lignocaine. Several studies, Singh[5], Helfman[5], Singh[7] and Kindler[8] support our results regarding heart rate changes observed in the esmolol group.

Regarding systolic, diastolic and mean arterial pressure, there was no statistically significant difference between the two groups. Similar observations were noted by Kindler[8] in their study. But Helfman[6] noted that esmolol was effective in protecting against increase in systolic blood pressure compared to lignocaine. This could be attributed to the higher dose of esmolol(150 mg) used in their study.

## CONCLUSION

Intravenous esmolol 1mg/kg given 3 minutes before intubation was found to be more effective in controlling heart rate to laryngoscopy and intubation compared to intravenous lignocaine(1.5mg/kg) given 3 minutes before intubation and with respect to systolic, diastolic and mean arterial pressures both the drugs were comparable.

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