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Original Article

Comparison between Esmolol and Intravenous Lignocaine for Attenuating Hemodynamic Response to Laryngoscopy and Endotracheal Intubation

Dinesh Suryanarayana Rao[1], Ali Omer Abdelaziz Osman[2]

[1]Senior Resident, Department of Anaesthesiology, Sri Ramachandra Institute of Medical Sciences and Research, Chennai, Tamil Nadu, India, [2]AHS Student, Department of Allied Health Sciences, Sri Ramachandra Institute of Medical Sciences and Research, Chennai, Tamil Nadu, India

Abstract

Introduction: Laryngoscopy and tracheal intubation are usually associated initially with bradycardia followed by sympathetic stimulation characterized by increase in heart rate and blood pressure. The cardiovascular changes appear within a few seconds and can last for 5–10 min. In this study, we are comparing the effectiveness of esmolol with lignocaine in blunting the cardiovascular response to intubation.

Materials and Methods: After obtaining the Institutional Ethics Committee, the patients were divided into three groups with 20 patients in each group. Patients in Group A were given esmolol 0.5 mg/kg 30 s before intubation, while Group B patients received lignocaine 1.5 mg/kg approximately 90 s before intubation. Group C patients did not receive any drug. The heart rate and blood pressure were noted down at each minute after intubation for 5 min in all the groups and tabulated.

Results: From our observations, we found that the heart rate variability was minimal in both Groups A and B, with no statistical significance. However, after laryngoscopy, the 1st min and 2nd min observations showed a significant difference when compared between the two groups. The blood pressure control was better in the esmolol group, compared to the lignocaine group with $P < 0.05$.

Conclusion: The present study concluded that esmolol definitely achieves better control of the hemodynamic response, compared to lignocaine, especially in the first few minutes after intubation.

Key words: Blood pressure, Esmolol, Heart rate, Lignocaine

INTRODUCTION

Laryngoscopy and intubation are essential skills to be mastered by the anesthetist. This intervention is usually associated initially with bradycardia followed by sympathetic stimulation characterized by increase in heart rate and blood pressure. The cardiovascular changes appear within a few seconds and can last for 5–10 min.[1,2] Unlike in healthy patients, they can cause adverse outcomes in patients with comorbidities such as cardiac disease, hypertension, and kidney disease. Various agents have been tried in the past to prevent sympathetic stimulation including opioids, B-blockers, local anesthetic infiltration of airway, intravenous lignocaine, calcium channel blockers, and magnesium.[3] Lignocaine is a commonly used drug for attenuating the intubation response. Esmolol is a short-acting β-adrenergic blocker which has been found to be very effective to control the heart rate and blood pressure increase after intubation.[4] In this study, we are comparing the effectiveness of esmolol with lignocaine in blunting the cardiovascular response to intubation.

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MATERIALS AND METHODS

After obtaining Institutional Ethics Committee, the patients were divided into three groups with 20 patients in each group (in accordance with the power of the study). All patients belonged to ASA I or II and were between the

Corresponding Author: Dr. Dinesh Suryanarayana Rao, 10 Venkatraman Street, Perambur, Chennai - 600 011, Tamil Nadu, India.

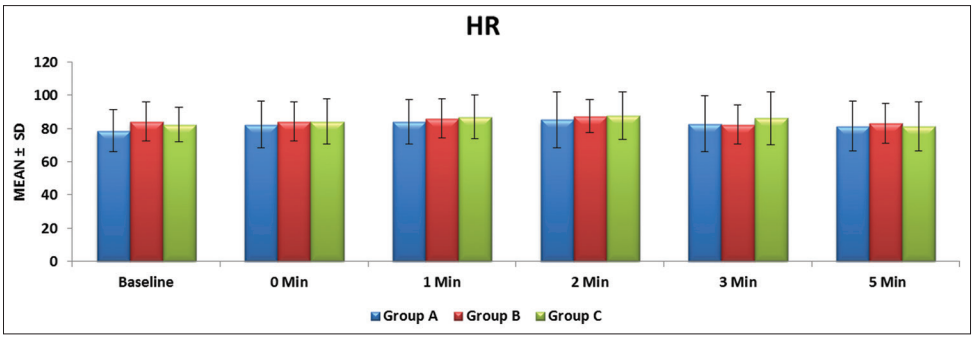

Figure 1: Changes in heart rate

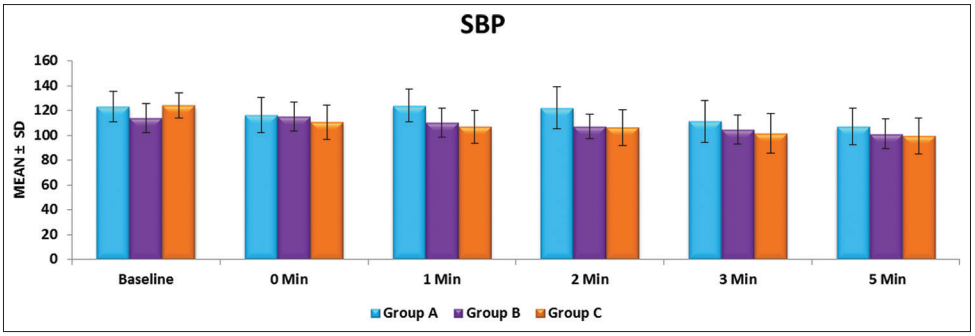

Figure 2: Changes in systolic blood pressure

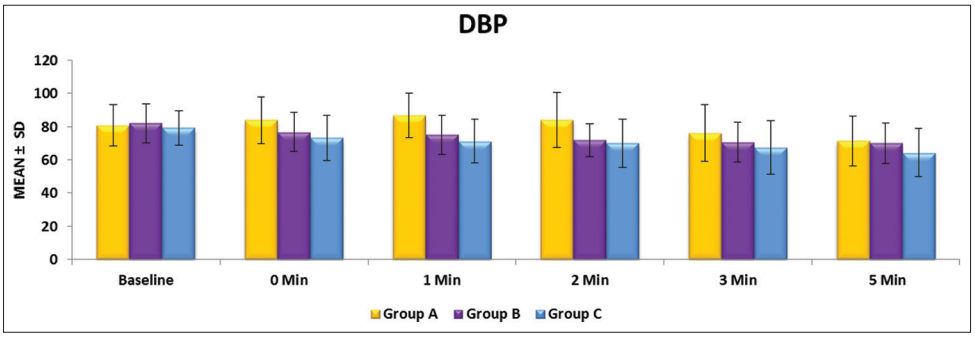

Figure 3: Changes in diastolic blood pressure

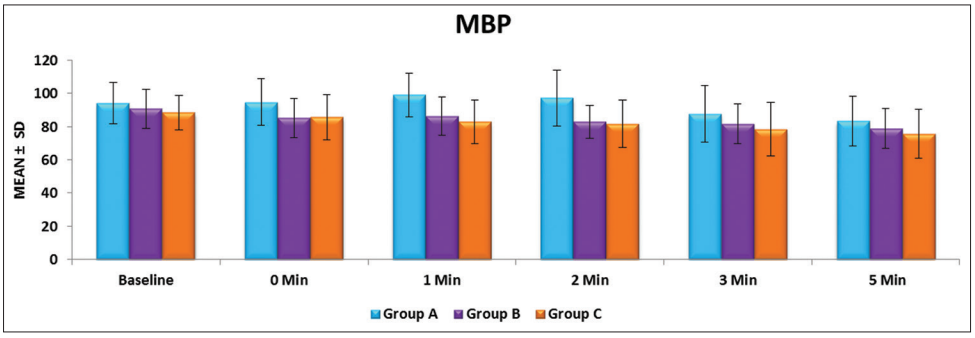

Figure 4: Changes in mean arterial pressure

ages of 20 and 45 years. The baseline heart rate and blood pressure were obtained before induction.

Exclusion criteria included patients with multiple comorbidities, ASA III or more, cardiac surgeries, emergency surgeries, and laryngoscopy time of >30 s. Patients in Group A were given esmolol 0.5 mg/kg 30 s before intubation, while Group B patients received lignocaine 1.5 mg/kg approximately 90 s before intubation. Group C patients did not receive any drug. The heart rate

Table 1: Changes in heart rate

Parameters	Mean±standard deviation			
	Group A	Group B	Group C	*P*-value
Baseline HR	78.80±12.539	84.25±11.787	82.40±10.415	0.327
HR 0 mins	82.30±14.057	84.30±11.721	84.25±13.649	0.861
HR 1 mins	84.15±13.287	86.05±11.741	86.95±13.193	0.779
HR 2 mins	85.35±16.819	87.60±9.848	87.80±14.439	0.829
HR 3 mins	82.95±16.925	82.50±11.905	86.25±16.039	0.694
HR 5 mins	81.40±14.905	83.10±12.109	81.30±14.553	0.900

Table 2: Changes in systolic blood pressure

Parameters	Mean±standard deviation			
	Group A	Group B	Group C	*P*-value
Baseline SBP	123.20±20.172	114.00±23.143	124.05±21.135	0.270
SBP 0 mins	116.45±17.310	115.00±17.137	110.55±25.353	0.634
SBP 1 mins	123.95±23.119	110.00±13.401	106.95±23.175	0.025
SBP 2 mins	122.15±24.071	106.90±14.205	106.15±16.155	0.013
SBP 3 mins	111.40±19.685	104.70±14.412	101.50±13.197	0.146
SBP 5 mins	107.00±19.714	101.15±13.838	98.40±10.096	0.257

Table 3: Changes in diastolic blood pressure

Parameters	Mean±standard deviation			
	Group A	Group B	Group C	*P*-value
Baseline DBP	80.85±14.755	82.15±16.187	79.35±13.096	0.835
DBP 0 min	83.90±14.567	76.80±15.966	73.25±17.597	0.112
DBP 1 min	86.80±18.412	75.05±9.997	71.25±18.669	0.010
DBP 2 min	84.05±17.721	71.85±13.433	70.05±11.776	0.007
DBP 3 min	76.15±15.675	70.60±10.976	67.35±9.298	0.081
DBP 5 min	71.35±17.055	70.10±11.544	64.35±6.596	0.117

DBP: Diastolic blood pressure

Table 4: Changes in mean arterial pressure

Parameters	Mean±standard deviation			
	Group A	Group B	Group C	*P*-value
Baseline MAP	94.25±15.437	90.90±12.371	88.45±11.468	0.384
MAP 0 min	94.75±15.338	85.40±15.024	85.80±19.856	0.150
MAP 1 min	99.15±19.911	86.40±10.620	82.85±20.142	0.012
MAP 2 min	97.35±18.230	83.00±13.872	81.80±13.121	0.003
MAP 3 min	87.80±16.976	81.65±11.156	78.55±10.081	0.084
MAP 5 min	83.35±17.966	79.00±11.494	75.75±6.703	0.184

MAP: Mean arterial pressure

and blood pressure were noted down at each minute after intubation for 5 min in all the groups and tabulated.

RESULTS

The heart rate baseline in Group A had a mean of 78.80 ± 12.539, while in Group B, the mean was 84.25 ± 11.787 and $P = 0.327$. At 0 min, Group A mean was 82.30 ± 14.057, and in Group B, the mean was 84.30 ± 11.721, and $P = 0.861$. At 1 min, Group A had a mean of 84.15 ± 13.287, while in Group B, the mean was 86.05 ± 11.741, and $P = 0.779$. At 2 min, the mean of Group A was 85.35 ± 16.819, and Group B mean was 87.60 ± 9.848, and $P = 0.829$. At 3 min, Group A mean was 82.95 ± 16.925, while in Group B, the mean was 82.50 ± 11.905, and $P = 0.694$. At 5 min, the mean of Group A was 81.40 ± 14.905, and Group B mean was 83.10 ± 12.109, and $P = 0.900$ [Table 1 and Figure 1].

The systolic blood pressure baseline in Group A had a mean of 123.20 ± 20.172, while in Group B, the mean was 114.00 ± 23.143, and $P = 0.270$. At 0 min, Group A mean was 116.45 ± 17.310, and in Group B, the mean was 115.00 ± 17.137, and $P = 0.634$. At 1 min, Group A had a mean of 123.95 ± 23.119, while in Group B, the mean was 110.00 ± 13.401, and $P = 0.025$. At 2 min, the mean of Group A was 122.15 ± 24.071, and Group B mean was 106.90 ± 14.205, and $P = 0.013$. At 3 min, Group A mean was 111.40 ± 19.685, while in Group B, the mean was 104.70 ± 14.412, and $P = 0.146$. At 5 min, the mean of Group A was 107.00 ± 19.714, and Group B mean was 101.15 ± 13.838, and $P = 0.257$ [Table 2 and Figure 2].

The diastolic blood pressure baseline in Group A had a mean of 80.85 ± 14.755, while in Group B, the mean was 82.15 ± 16.187, and $P = 0.835$. At 0 min, Group A mean was 83.90 ± 14.567, and in Group B, the mean was 76.80 ± 15.966, and $P = 0.112$. At 1 min, Group A had a mean of 86.80 ± 18.412, while in Group B, the mean was 75.05 ± 9.997, and $P = 0.010$. At 2 min the mean of Group A was 84.05 ± 17.721, and Group B mean was 71.85 ± 13.433, and $P = 0.007$. At 3 min, Group A mean was 76.15 ± 15.675, while in Group B, the mean was 70.60 ± 10.976, and $P = 0.081$. At 5 min, the mean of Group A was 71.35 ± 17.055, and Group B mean was 70.10 ± 11.544, and $P = 0.117$ [Table 3 and Figure 3].

The mean arterial pressure baseline in Group A had a mean of 94.25 ± 15.437, while in Group B, the mean was 90.90 ± 12.371, and $P = 0.384$. At 0 min, Group A mean was 94.75 ± 15.338, and in Group B, the mean was 85.40 ± 15.024, and $P = 0.150$. At 1 min, Group A had a mean of 99.15 ± 19.911, while in Group B, the mean was 86.40 ± 10.620, and $P = 0.012$. At 2 min, the mean of Group A was 97.35 ± 18.230, and Group B mean was 83.00 ± 13.872, and $P = 0.003$. At 3 min, Group A mean was 87.80 ± 16.976, while in Group B, the mean was 81.65 ± 11.156, and $P = 0.084$. At 5 min, the mean of Group A was 83.35 ± 17.966, and Group B mean was 79.00 ± 11.494, and $P = 0.184$ [Table 4 and Figure 4].

The ASA classification in Group A was 70% of patients belong to ASA I and 30% belong to ASA II. While in

Group B, 45% of patients belong to ASA I and 55% of patients belong to ASA II.

DISCUSSION

General anesthesia associated with laryngoscopy and intubation is painful, associated with acute increase in hemodynamic responses, which lasts for around 5–10 min. This sympathetic stimulation can be deleterious in patients with comorbidities and high-risk patients (ASA III or more). Various agents have been tried previously such as opioids, alpha-2 agonists, N-Methyl-D-aspartate receptor antagonists, beta-blocker, and lignocaine.[2,3]

Intravenous lignocaine is a conservative free local anesthetic that can be used to suppress the intubation response. Lignocaine has the ability to cause direct myocardial suppression and peripheral vasodilation. It's significance lies in the fact that it can suppress the airway irritation to tracheal mucosa during intubation and hence can blunt the adrenergic response.[5,6]

Esmolol hydrochloride is a relatively cardioselective, beta-antagonist. It has a rapid onset of action, has a peak hemodynamic effect within minutes, and possesses a short elimination half-life of 9 min. Hence, it is believed to be an ideal agent to be used for the intubation response.[7,8]

From our observations, we can find that the heart rate variability was minimal in both Groups A and B, with no statistical significance. This is in accordance with the study done by Singh *et al.* which showed that both lignocaine and esmolol are effective in controlling the heart rate during intubation.[6,9,10]

Blood pressure observations showed no significance in pre-induction baseline values. However, after laryngoscopy, the 1st min and 2nd min observations showed a significant difference when compared between the two groups.[11,12] The blood pressure control was better in the esmolol group, compared to the lignocaine group with $P < 0.05$.[13,14] This observation is supported in the study by Ugur *et al.* which showed that esmolol provided better control of blood pressure compared to lignocaine.[7,15]

CONCLUSION

Comparing the two most common drugs used for intubation response, we find that both esmolol and lignocaine play a role in preventing the sympathetic stimulation. However, esmolol has definitely been shown to achieve better control of the hemodynamic response, especially in the first few minutes after intubation. Its pharmacokinetic profile makes it a suitable agent in this regard.

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