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Efficacy of a Bolus Dose of Esmolol and Bolus Dose of Lignocaine for Attenuating the Pressor Response to Laryngoscopy and Endotracheal Intubation in General Anesthesia: A Comparative Study

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Abstract

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INTRODUCTION

Laryngoscopy and endotracheal intubation are associated with mechanical stimulation of the respiratory tract,[1] leading to sympathoadrenal stimulation. It manifests as an increase in heart rate, blood pressure, and cardiac complications.[2] Attenuation of these responses by intravenous lignocaine found some studies effective[3,4] and in some studies less reliable.[5,6] Use of esmolol is effective in blunting pressor response and provides hemodynamic stability in risk patients.[7,8,9,10,11]

The pressor response during laryngoscopy and intubation was first explained by Reid and Brace in 1940. Yu *et al.*[12] reported that the tachycardia and hypertension are the main cause of morbidity and mortality in perioperative myocardial infarction. Devereaux *et al.*[13] showed that the incidence of mortality and morbidity rates are 12%–40% after perioperative myocardial infarction.

Perioperative beta-blockers use in cardiac surgery by Blessberger *et al.*[14] showed beneficial effects, as they reduce rhythm disturbances, but effect on heart attacks, heart failure, and low blood pressure remains unclear. Esmolol cardioselective β-adrenergic antagonist permits for intraoperative use. It blocks the β-adrenergic receptors and also

reduces the force of contraction and heart rate. Varying doses of esmolol 0.5–2 mg/kg have been used in the past. Parvez *et al.*[15] study supported that attenuation of heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure (MAP), and rate pressure product (RPP) using esmolol 1.5 mg/kg. Singh *et al.*[16] reported that esmolol 2 mg/kg is effective in suppressing the pressor response without any deleterious effects. However, in the literature, no agreement was made in the use of an accurate dose of esmolol for attenuation of hemodynamic response.

Hence, this study was performed to compare the efficacy of an accurate bolus dose of esmolol and bolus dose of lignocaine in attenuating the pressor response to laryngoscopy and endotracheal intubation in general anesthesia with respect to pulse rate (PR), SBP, diastolic blood pressure, MAP, and RPP.

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Materials and Methods

This randomized prospective control study was carried out after obtaining Hospital Ethical Committee clearance (No/58/2015 on 20/11/15) and informed consent from all the patients. Group I was esmolol (n = 30) and Group II was lignocaine (n = 30). Normotensive patients scheduled for surgery under general anesthesia in the age group of 20–50 years of both sexes and the American Society of Anesthesiologists physical Status I and II were included in the study. Patients suffering from comorbidities predicted difficult intubation, prolonged laryngoscopy and intubation, and head-and-neck surgery were excluded from the study.

With anticipated mean and standard deviation (SD) of PR after 5 min in the lignocaine group of 90.27 and 14.83, respectively, and that in the esmolol group of 78.16 and 13.04, respectively, in the reference study, the minimum sample size per group is 27 patients with 90% power and 5% level of significance. It was computed using the following formula:

$$N = 2\left(\frac{[Z_{1-\alpha/2} + z_{1-\beta}] \times S}{d}\right)^2$$

$Z_{1-\alpha/2}$ Level of significance = 95%,

$Z_{1\text{-}b}$ - power of the study = 80%, d = clinically significant difference between two parameters

SD = Common SD. Sample size taken in each group was 30.

Procedure

Patients in the esmolol and lignocaine groups on arrival to operation theater recorded basal parameters and premedicated with injection glycopyrrolate 0.01 mg/kg and injection midazolam 0.02 mg/kg 5 min before induction. After preoxygenation, Group I patients received esmolol 1.5 mg/kg and Group II patients received lignocaine 1.5 mg/kg IV 2 min before intubation and diluted with normal saline 10 ml. Patients were induced with injection thiopentone 5 mg/kg, followed by succinylcholine 1.5 mg/kg to facilitate intubation and ventilation. Laryngoscopy and endotracheal intubation were done with an appropriate-size

endotracheal tube within 15–30 s in the first attempt and anesthesia was continued with O_2, N_2O halothane, and muscle relaxation.

The present study focused on events from the time of injection of study drugs up to 5 min after intubation. Adjuvants and analgesics were administered after the study period. At the following times on arrival to opertation theatre, during laryngoscopy and intubation, and after 1, 2, 3, 4, and 5 min of intubation, PR, SBP, and DBP readings were noted and MAP and RPP were calculated. Patients were monitored for conduction abnormalities, ST-segment changes with electrocardiography monitoring, hypotension, bradycardia, bronchospasm, and pain on injection. At the end of the surgery, the patients reversed with injection neostigmine 0.05 mg/kg and injection glycopyrrolate 0.01 mg/kg. Patients were followed up postoperatively for complications.

Statistical analysis

The collected data were analyzed using proper statistical tests such as Student's *t*-test, and data were represented by mean SD and graphs. Data were analyzed using statistical software SPSS version 17 (17.0 software, IBM Corp, Armonk NY, USA). $P \leq 0.05$ was considered as statistically significant.

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RESULTS

In the study, the groups were collectively matched for their demographic data [Table 1]. There was a significant increase in mean PR in lignocaine group, which did not come back to baseline level even after 5 min. In the esmolol group, there was a significant attenuation of mean PR during and following intubation [Table 2 and Figure 1]. Mean of MAP in lignocaine group showing a significant rise from baseline values, especially during and 1 and 2 min after laryngoscopy and endotracheal intubation. Mean of MAP in the esmolol group, there was a significant fall of the hypertensive response to laryngoscopy and endotracheal intubation [Table 3 and Figure 2]. In this study, mean of RPP in lignocaine group, shows significant rise during and after intubation which did not reach the baseline value even after 5 min. There was a fall in mean RPP in esmolol group during intubation, which persisted even after 5 min [Table 4 and Figure 3].

Table 1

Distribution of study population

Variables	Mean±SD	Unpaired *t*-test

	Esmolol	Lignocaine	
Age	35.7±7.72	35.33±7.06	*P*=0.8471 (NS)
Weight	55.33±7.95	55.67±8.54	*P*=0.8738 (NS)
Height	161.93±6.48	162.86±6.37	*P*=0.5773 (NS)

SD=Standard deviation, NS=Nothing significant

Table 2

Changes in mean pulse rate

Variables	Mean±SD				
	Baseline	Intubation	After intubation		
			1 min	2 min	3 min	5 min

Variables	Mean±SD					
	Baseline	Intubation	After intubation			
			1 min	2 min	3 min	5 min
Esmolol	83.6±6.38	78±6.21	75.1±5.7	73.4±5.97	73.47±5.53	76.3±4.94
Lignocaine	82.3±7.87	95.13±8.14	107.43±9.57	116.1±8.8	111.97±8.28	105.67±8
t	0.7	8.85	15.89	21.98	21.18	17.11
P	>0.05	<0.01	<0.001	<0.001	<0.001	<0.001

SD=Standard deviation

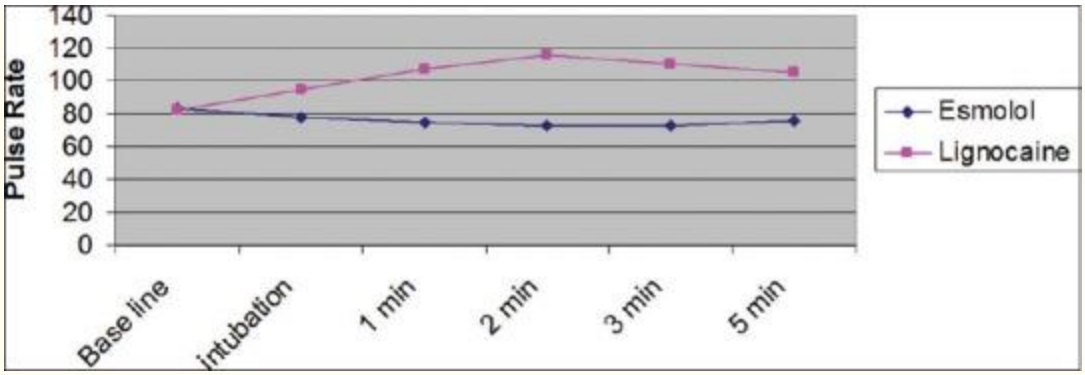

Figure 1

Comparison of mean pulse rate between esmolol and lignocaine

Table 3

Changes in mean arterial pressure

Variables	Mean±SD					
	Baseline	Intubation	After intubation			
			1 min	2 min	3 min	5 min
Esmolol	92.93±6.27	90.5±4.9	89.07±4.6	89.1±4.9	88.8±5.2	88.5±5.3

Variables	Mean±SD					
	Baseline	Intubation	After intubation			
			1 min	2 min	3 min	5 min
Lignocaine	95.03±5.7	101.1±5.1	107.5±5.6	110.87±6.1	107±5.5	102.57±5.9
t	1.43	2.61	4.07	4.03	3.01	2.45
P	>0.05	<0.01	<0.001	<0.001	<0.01	<0.01

SD=Standard deviation

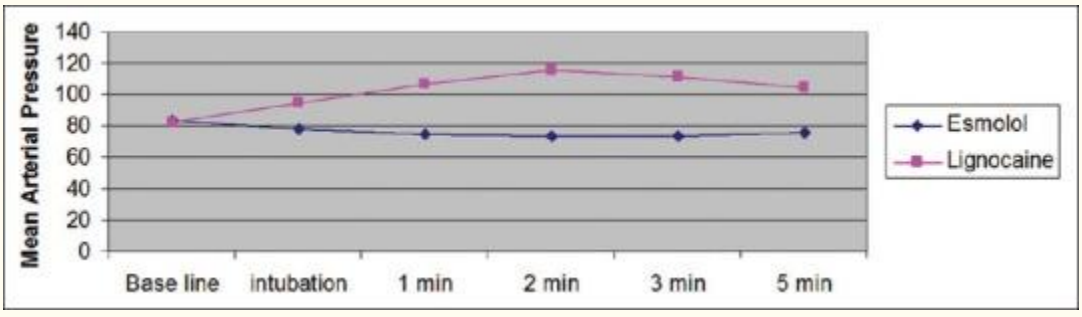

Figure 2

Comparison of mean arterial pressure between esmolol and lignocaine

Table 4

Changes in rate pressure product

Variables	Mean±SD					
	Baseline	Intubation	After intubation			
			1 min	2 min	3 min	5 min
Esmolol	10,161±13	10,064±13	9904±11	9900±12	9761±10	9852±96
Lignocaine	10,344±12	12,801±13	15,002±16	16,581±17	15,613±15	14,103±14
t	1.43	2.61	4.07	4.03	3.01	2.45
P	>0.05	<0.01	<0.001	<0.001	<0.01	<0.01

SD=Standard deviation

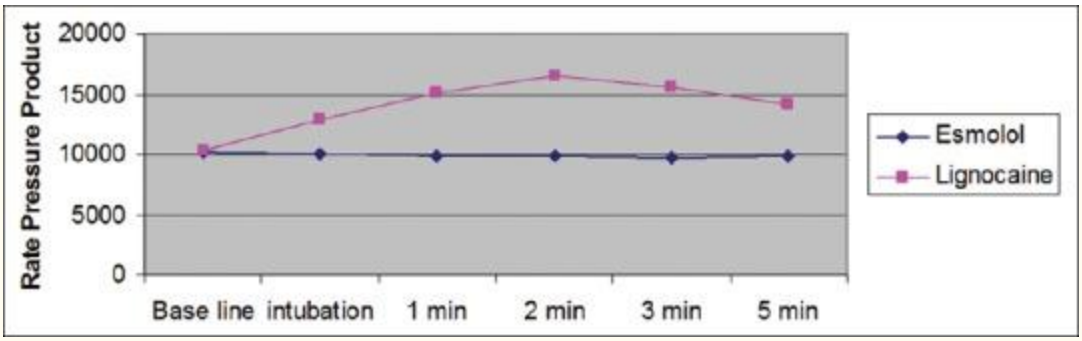

Figure 3

Comparison of rate pressure product between esmolol and lignocaine

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DISCUSSION

The pressor response to the procedure of laryngoscopy and endotracheal intubation is a reflex response.[2] Deep pressure on the base of the tongue is also responsible for such response. These stimuli activate suprasegmental and hypothalamic sympathetic centers to cause a peripheral sympathoadrenal response which releases catecholamines.[5]

Esmolol short-acting cardioselective β-adrenergic antagonist permits rapid titration to a desired level of β-blockade on administration; hence, it a suitable agent for the perioperative period. At therapeutic doses, it does not have intrinsic sympathetic activity or membrane-stabilizing activity.[7,8,9,10,11] In the literature, different doses of esmolol have been reviewed, but the selection of an effective dose of esmolol is essential to balance between desired effects and adverse effects.

Prajwal Patel *et al.*[17] study received esmolol 1.5 mg/kg and labetalol 0.25 mg/kg 2 min before extubation and concluded that esmolol was more efficient than labetalol at extubation and immediately postextubation. Shailaja and Srikantu[18] study in hypertensive patients received normal saline, esmolol 1.5 mg/kg, and esmolol 1.5 mg/kg with fentanyl 2 μg/kg during laryngoscopy and endotracheal intubation and concluded that esmolol 1.5 mg/kg is effective in attenuating hemodynamic response to laryngoscopy and intubation, but the combination of the drug causes hypotension following intubation. Talwar *et al.*[19] study reported that esmolol (1.5 mg/kg) and esmolol with diltiazem were both effective in attenuating heart rate, SBP, DBP, and MAP after laryngoscopy and endotracheal intubation. Sharma *et al.*[20] showed that esmolol 1.5 mg/kg and dexmedetomidine both suppress the hemodynamic response to intubation when compared to the control group. In accordance with the above studies, we preferred esmolol 1.5 mg/kg in our study and found significant results compared to lignocaine 1.5 mg/kg.

Helfman *et al.*[21] reported that significant attenuation of heart rate, SBP in bolus doses of esmolol 200 mg. Singh *et al.*[22] showed that esmolol 1.4 mg/kg was significantly more effective than lignocaine 1.5 mg/kg in minimizing the increase in MAP.[22] We also found a

significant reduction in MAP and PR in our study. Some study showed that patients with RPP greater than 12,000 developed ischemic electrocardiographic changes.[23] Esmolol reduces RPP in van den Berg *et al.*[24] study compared to lignocaine.

Lignocaine in attenuating the hemodynamic response during endotracheal intubation is due to direct cardiac depression and peripheral vasodilatation. It has antiarrhythmic and analgesic properties. In Marulasiddappa and Nethra[25] study, the use of lignocaine 1.5 mg/kg is inadequate in comparison with clonidine to control heart rate and blood pressure in neurosurgical cases.

Oxorn *et al.*,[26] Sheppard *et al.*,[10] and Vucevic *et al.*[27] reported that the only side effect of esmolol was phlebitis, which can be avoided by suitable dilution. There were no adverse effects seen in our study.

In our results, we found a difference in mean PR, MAP, and mean RPP between esmolol group and lignocaine group during laryngoscopy and endotracheal intubation as in similar study. However, we have not observed any complications in the study.

There were certain limitations to our study. We were unable to assess the plasma level of catecholamines during the study. We had taken only the changes in normotensive patients, not comorbid patients. Hence, further studies required to know the effective, accurate plasma concentration of the drug to prevent pressor response during laryngoscopy and intubation in risk patients.

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CONCLUSIONS

Esmolol, an ultra-short-acting cardioselective beta-adrenergic antagonist in the bolus dose of 1.5 mg/kg intravenously, is effective in blunting the pressor response to laryngoscopy and endotracheal intubation when compared with intravenous lignocaine 1.5 mg/kg in general anesthesia without causing undue bradycardia and hypotension.

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Conflicts of interest

There are no conflicts of interest.

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