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Original Research Article

Clinical efficacy of Esmolol, Lignocaine and Diltiazem as premedicant for attenuation of hemodynamic responses of laryngoscopy and endotracheal intubation- a comparative evaluation

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ABSTRACT

Background: Direct laryngoscopy and endotracheal intubation after induction of anesthesia is almost always associated with hemodynamic stress response. The aim of this study was to compare esmolol, lignocaine and diltiazem for suppression of laryngoscopy and intubation response.
Methods: This randomized prospective double-blind control study was performed on 120 patients of either sex, aged between 18 and 58 years of ASA physical status I and II with Mallampatti grade I and II, undergoing elective surgeries under general anesthesia with endotracheal intubation. Patients were randomized in four groups, Group N (normal saline), Group E (esmolol) 1.5mg/kg I.V, Group D (diltiazem) 0.2mg/kg I.V, and Group L (lignocaine) 1.5mg/kg I.V with 30 patients in each group. Hemodynamic parameters were recorded during the basal period, preinduction, during intubation and at specified intervals.
Results: There was significant increase in systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate in the control group (Group N) in association with tracheal intubation. The heart rate was significantly lower in Group E (Esmolol group), followed by Group D (Diltiazem group) and Group L (Lignocaine group). Rate pressure product was significantly lower in group E as compared to other groups, followed by group D and group L. Maximum increase in rate pressure product (RPP) just after laryngoscopy and intubation was ±74.29% in group N, ±16.11% in group E, 25.38% in group D and 38.77%in group L.
Conclusions: Esmolol was better than diltiazem and they both were better than lignocaine for preventing the hemodynamic response after laryngoscopy and intubation.

Keywords: Diltiazem, Esmolol, Hemodynamic stress response, Intubation, Laryngoscopy, Lignocaine

INTRODUCTION

The extent of hemodynamic response to direct laryngoscopy by Macintosh laryngoscope and endotracheal intubation depends upon factors like distortion of airway or type and duration of physical stimulus to oropharyngeal structures. The principle mechanism for hypertension and tachycardia is the sympathetic response which may be due to the increase in catecholamine activity.[1-3]

Transitory hypertension and tachycardia are probably of no significance in healthy individuals, but these may be hazardous in patients with hypertension, myocardial insufficiency or cerebrovascular diseases.[3] It may predispose these patients for development of pulmonary edema, myocardial insufficiency, cerebrovascular

accidents, acute left ventricular failure (LVF) and dysrhythmias.[4-7] Pressor response is exaggerated in hypertensive patients even though rendered normotensive pre-operatively by antihypertensive medications.[8]

Pharmacological measures using volatile anesthetics- to deepen the anaesthesia, topical or intravenous lignocaine opioid analgesics like fentanyl, magnesium sulphate, calcium channel blockers beta-blockers clonidine and vasodilators- sodium-nitroprusside, nitro-glycerine have been studied, but none of the above approaches have proved to be ideal due to their limitations and side effects.[9-14] Hence the search of an ideal agent to attenuate the hemodynamic responses of laryngoscopy and intubation is still continuing.

Out of the various beta- blockers, esmolol is a striking option because of its cardio-selectivity and ultra-short duration of action, but it can only be administered intravenously.[14] Lignocaine is commonly used agent for attenuating the circulatory responses associated with tracheal intubation. Intravenous lignocaine depresses the central nervous system and its antiarrhythmic effect was found to be suitable alternative method to minimize this hemodynamic pressor response.

Diltiazem a calcium channel blocker can blunt hemodynamic responses because of its directly acting vasodilating effects.[11-13] It reduces the pressor effect of circulating noradrenaline on resistance vessels, resulting from inhibition of the calcium influx that accompanies stimulation of α2 receptors, thus leading to attenuation of increase in arterial pressure after elevated plasma concentrations of noradrenaline. Calcium channel blockers are also preferred because myocardial depression produced by them is minimized by reduction in afterload so that cardiac output remains unchanged, but they show no effect on increase in heart rate. The present study was aimed to study and compare esmolol, lignocaine and diltiazem given intravenously for suppression of laryngoscopy and intubation response.

METHODS

This randomized prospective double-blind control study was performed on 120 patients of either sex, aged between 18 and 58 years of ASA physical status I and II with Mallampati grade I and II, undergoing elective surgeries under general anaesthesia with endotracheal intubation after approval from institutional ethical committee. Each patient received a written and verbal description of the research protocol and written informed consent was taken from all the patients in their language for inclusion in the study.

Patients with anticipated difficult airway, who required more than one attempt for intubation, uncontrolled hypertension or chronic respiratory disease, hepatic disease or renal disease, neurological disorder or endocrinal disease, were excluded from the study. Other exclusion criteria were pregnant and lactating women, obese patients, patients with known hypersensitivity or drug allergies, taking any antihypertensive or antidepressant drugs and patients who refused to participate in the study.

Preliminary sample size was decided in consultation with statisticians and based on previous studies which indicated that approximately 25-27 patients should be included in each group in order to ensure power of 80% and α-error of 0.05 for detecting clinically meaningful reduction by 20% in heart rate and mean arterial blood pressure during laryngoscopy and endotracheal intubation. Assuming a 5% drop out rate and for equal distribution of patients in all the four groups, a total of 120 patients were incorporated for the present study.

Patients were randomized in four groups of 30 patients each by card method. A total of 120 cards were prepared by another person who was blinded about the study protocol. After recruitment, every patient was asked to draw one card and grouped accordingly:

- Group N- received 10 ml of normal saline intravenously 2 minutes prior to laryngoscopy and intubation
- Group E- received intravenous esmolol 1.5mg/kg diluted in 10 ml normal saline, 2 minutes before laryngoscopy and intubation
- Group D- received intravenous diltiazem 0.2mg/kg diluted in 10 ml normal saline, 2 minutes prior to laryngoscopy and intubation
- Group L- received intravenous lignocaine 1.5mg/kg diluted in 10 ml normal saline, 2 minutes before laryngoscopy and intubation.

Anesthetic technique

All the patients were subjected to pre-anesthetic assessment including medical history and physical examination with relevant investigations. X-ray chest and ECG was also reviewed. All patients were premeditated orally with tab. alprazolam 0.5mg and tab ranitidine 150 mg at bed time previous night. They all were kept fasting for 6 hours prior to surgery.

On arrival to operation theatre, an 18-gauge cannula was placed and lactated Ringer solution was started in all patients. Monitoring was done using a multipara monitor to record heart rate, noninvasive measurements of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), end tidal carbon dioxide ($EtCO_2$) and continuous electrocardiographic (ECG) monitoring with peripheral capillary oxygen saturation (SPO_2) by finger pulse oximeter.

All patients received intravenous inj. midazolam 0.02 mg/kg, inj. glycopyrrolate 0.005mg/kg, inj. ondansetron 4mg and inj. morphine 0.1mg/kg. After preoxygenation for 3 minutes with 100% oxygen by face mask,

anaesthesia was induced with propofol 2mg/kg, followed by inj. vecuronium bromide 0.1mg/kg, to facilitate the direct laryngoscopy and intubation. The patient's lungs were manually ventilated by face mask with 100% O_2. All patients received study drugs according to allocated group one minute after induction. Direct laryngoscopy and endotracheal intubation was done two minutes after administering the study drug. All intubations were done by an experienced anesthesiologist. Any patient requiring more than 20 seconds or more than 1 attempt for intubation was excluded from the study. Anaesthesia was maintained by isoflurane, nitrous oxide 60% in oxygen. Intermittent positive pressure ventilation (IPPV) was continued by mechanical ventilator with tidal volume and respiratory rate adjusted to maintain an $EtCO_2$ between 35-40 mm of Hg.

Further neuromuscular blockade was maintained by inj. vecuronium bromide at a dose of 0.02 mg/kg. At the end of surgery, residual neuromuscular blockade was reversed by inj. neostigmine 0.05mg/kg and glycopyrrolate 0.01mg/kg. Extubation was performed when respiration was adequate and patient could follow simple commands. Patients were transferred to post anaesthetic care unit and monitored for any adverse events and complications of hypotension, hypertension, arrhythmias, hypoxemia and bronchospasm.

Intra-operative monitoring

The hemodynamic parameters of heart rate, systolic, diastolic and mean blood pressure and ECG changes, were noted by anesthesiologist who was blinded about the study drug. Parameters were recorded at baseline, after induction, after giving study drug and just after tracheal intubation, then at regular intervals of 1 minute, 2 minutes, 3 minutes, 4 minutes, 5 minutes, 7 minutes and 10 minutes after tracheal intubation.

Statistical analysis

The data obtained in the study are presented in tabulated manner and variables are expressed as Mean±SD. Categorical data was compared using Chi-square test. Numerical data was compared using one-way ANOVA for multiple comparison. For intragroup comparison paired 't' test was used and two group comparisons were done using Student 't' test. P-value < 0.05 was considered as significant and P-value <0.001 was taken as highly significant.

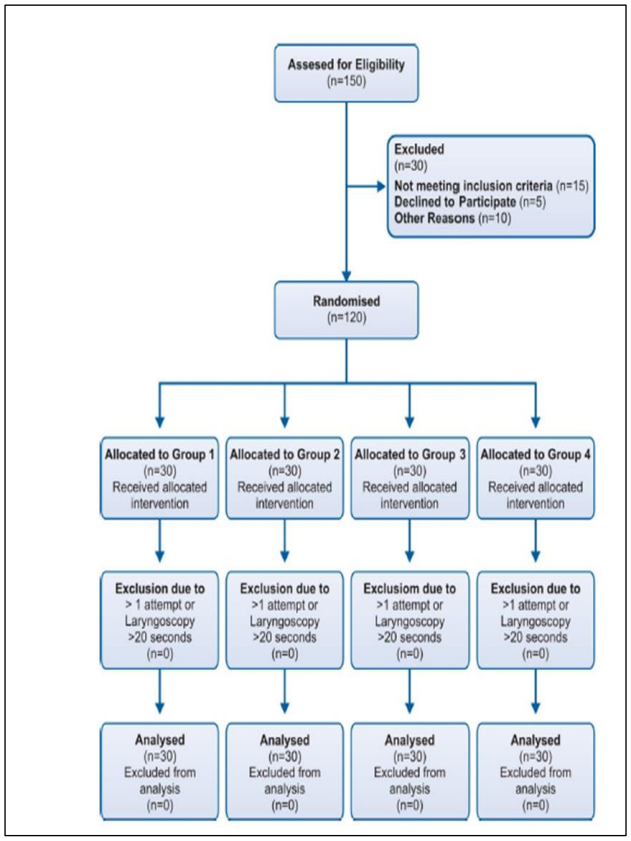

Figure 1: Consort flow.

RESULTS

Demographic profile for age weight height M/F ratio and ASA grading were comparable in all the four groups. (Table 1).

Table 1: Demographic profile.

	Group N	Group E	Group D	Group L	P-value
Age (years)	35.73±10.61	33.97±12.22	34.23±10.05	35.85±11.86	0.79
Wt (Kg)	57.73±5.86	57.17±5.59	58.43±5.61	59.23±5.876	0.69
Gender (M/F)	16/14	13/17	14/16	12/18	0.78
ASA (I/II)	21/9	22/8	20/10	20/10	0.85
Height (Cms)	161.3±6.87	162.6±7.12	161.9±6.36	162.3±7.06	0.51

Data are presented in Mean + SD or absolute numbers. P value <0.05 is statistically significant.

Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and heart rate (HR) in the control group (Group N) increased significantly in association with tracheal intubation. The heart rate was

significantly lower in Group E (Esmolol group), followed by Group D (Diltiazem group) and Group L (Lignocaine group). Maximum increase in HR occurred just after laryngoscopy. It was observed that the increase in heart rate for group N was +40.10%, group E was +4.74%, group D was +9.3% and for group L was +17.51% from the baseline, just after laryngoscopy and intubation (Table 2).

Table 2: Changes in heart rate (beats/min) among the groups.

	Group N	Group E	Group D	Group L	p-value
Baseline	88.20±4.23	86.69±3.71	86.80±3.15	86.82±4.43	0.24
After induction	87.23±3.46	87.09±5.10	88.26±2.26	87.69±4.73	0.06
After study drug	88.88±4.22	78.63±6.23	85.15±3.38	85.26±3.87	<0.001**
Just after laryngoscopy and intubation	123.60±3.82	90.60±3.22	98.88±2.92	102.11±3.16	<0.001**
1 minute	116.1±2.30	88.80±2.86	92.88±2.36	101.63±2.68	<0.001**
2 minutes	114.6±2.20	88.26±2.87	92.31±3.58	99.91±3.44	<0.001**
3 minutes	109.5±1.16	87.31±2.26	89.90±3.08	98.91±3.50	<0.001**
4 minutes	104.23±2.23	86.63±2.87	89.86±2.46	97.26±3.19	<0.001**
5 minutes	98.13±1.47	85.66±3.61	87.70±2.41	93.97±3.52	<0.001**
7 minutes	94.23±2.23	84.77±3.32	86.60±3.33	85.23±3.64	<0.001**
10 minutes	91.26±3.46	84.17±3.22	85.20±5.28	85.34±4.57	<0.001**

Data are presented in Mean + SD. P value <0.05 is statistically significant and P value < 0.001 is highly significant statistically.

Systolic blood pressure was significantly lower in group D (Diltiazem group) until 3 minutes after intubation after which it was lower in group E (Esmolol group) as compared to other groups. Diastolic blood pressure was significantly lower in group D (Diltiazem group) until 1 minute after intubation. After 2 minutes, diastolic blood pressure was comparable in both group D and group E. Mean arterial pressure: there was no significant difference in mean arterial pressure (MAP) between group E and group D up to 7 minutes after intubation. After 10 minutes, the mean arterial pressure (MAP) of group E was significantly lower than group D.

Maximum increase in mean arterial pressure (MAP) was just after laryngoscopy and intubation which was 22.12% in group1, 10.89% in group 2, 9.25% in group 3 and 17.47% in group L (Table 3).

Table 3: Comparison of mean arterial pressure (mmhg) among the groups.

	Group N	Group E	Group D	Group L	p- value
Baseline	97.43±2.79	97.60±4.36	96.92±3.53	96.84±4.38	0.734
After Induction	93.07±1.38	92.86±5.68	91.58±3.26	93.90±2.63	0.351
After Study drug	94.98±2.36	85.34±4.36	84.22±2.86	93.15±4.23	<.0001**
Just after laryngoscopy and intubation	118.99±2.68	108.23±5.93	105.89±3.83	113.76±4.09	<.0001**
1 minute	117.28±1.43	106.23±2.68	104.48±4.25	112.96±3.06	<.0001**
2 minutes	114.53±2.67	103.89±5.63	102.80±3.23	107.93±4.30	<.0001**
3 minutes	111.18±2.18	102.00±2.35	101.14±2.08	106.25±4.63	<.0001**
4 minutes	106.67±2.86	99.75±4.36	98.86±2.80	103.68±2.08	<.0001***
5 minutes	101.93±2.18	97.28±5.62	96.40±1.68	101.75±4.98	<.0001**
7 minutes	99.40±1.18	95.77±3.23	96.07±2.02	98.96±5.20	<.0001**
10 minutes	98.80±2.26	91.67±2.36	93.74±1.63	96.58±5.02	0.003*

Data are presented in Mean±SD. *P value <0.05 is statistically significant and ** P value < 0.001 is highly significant statistically.

Rate pressure product was significantly lower in group E as compared to other groups, followed by group D and group L. Maximum mean difference was just after laryngoscopy and intubation. Maximum increase in rate pressure product (RPP) just after laryngoscopy and

intubation was +74.29% in group N, +16.11% in group E, 25.38% in group D and 38.77%in group L (Table 4).

Intraoperative complications during study

- One patient in esmolol Group developed bradycardia (heart rate= 55 beats/minute). It occurred just after giving the study drug but increased after laryngoscopy and intubation, so no intervention was required
- One patient in diltiazem group developed hypotension (mean arterial pressure = 64 mm Hg). It occurred just after giving the study the drug but improved over a period of time.

Post-operative complications

No postoperative complications of hypotension/hypertension, bradycardia/tachycardia, sedation, respiratory depression, nausea and vomiting occurred.

Table 4: Changes in rate pressure product among the groups.

	Group N	Group E	Group D	Group L	P- value
Baseline	11016.5±189.2	10879.5±200.2	10980.2±202.68	10921.95±193.64	0.48
After induction	10354.3±202.6	10400.6±162.3	10458.8±182.63	10583.3±160.83	0.21
After study drug	10605.3±193.6	8235.9±186.2	8960.43±126.33	10250.8±175.96	<.0001**
Just after laryngoscopy and intubation	19310.4±316.6	12632.3±293.2	13767.6±293.2	15156.7±340.23	<.0001**
1 minute	17770.9±304.5	12175.7±302.6	12780.2±304.1	15110.6±304.68	<.0001**
2 minutes	16990.1±280.8	12064.5±212.6	12474.7±270.6	13819.5±310.96	<.0001**
3 minutes	15877.5±267.3	11839.2±189.36	11967.4±230.6	13681.2±301.68	<.0001**
4 minutes	14415+383.0	11351.1±170.40	11754.7±192.3	12951.9±270.36	<.0001**
5 minutes	12910.4±248.8	10833.9±180.35	11245.1±180.68	12425.7±250.53	<.0001**
7 minutes	12127.4±168.3	10603.45±130.83	10887.4±173.38	11122.9±180.68	<.0001**
10 minutes	11571±182.6	9968.25±145.68	10283.64±163.34	10952.7±192.16	0.04*

Data are presented in Mean±SD. *P value <0.05 is statistically significant and ** P value < 0.001 is highly significant statistically.

DISCUSSION

Laryngoscopy and intubation causes marked increase in arterial pressure and heart rate due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation, which can be harmful to certain subset of patients.[9] Although these hemodynamic changes are short lived, lasting for less than 10 mins, but they may be undesirable in patients with pre-existing myocardial or cerebral insufficiency. There is increasing evidence that control of the heart rate and blood pressure response to endotracheal intubation is essential to prevent adverse cardiovascular outcomes.[15,16] Out of various approaches for attenuation of hemodynamic response during endotracheal intubation, pharmacological approach is considered better as it reduces heart rate as well as blood pressure.

The precise mechanism that leads to the hemodynamic response to laryngoscopy and intubation probably involves intense sympathetic discharge and release of catecholamines, as evident in the control group. The markedly high cardiovascular changes occurred within few seconds following laryngoscopy and intubation. In the present study, there was increase in heart rate just after intubation in patients of all groups which can be attributed to an increase in the central sympathetic outflow. There is 31.6%, 17.61%, 7.00% and 2.20 % rise in heart rate in control, lignocaine, diltiazem and esmolol group respectively at 1 minute from baseline after laryngoscopy and intubation with statistically highly significant difference (p< 0.001).

The rise in systolic blood pressure, diastolic blood pressure and mean arterial pressure was highest in control group after 1 minute of laryngoscopy and intubation. Mean arterial pressure at 1 minute of laryngoscopy and intubation depicted a rise of 20.37%, 16.64%, 7.86% and 8.84% in control, lignocaine, diltiazem, and esmolol group respectively from baseline. The rate pressure product recorded a rise of 61.48%, 38.35% 16.39% and 11.91% in control, lignocaine, diltiazem and esmolol group respectively from baseline at 1 minute after laryngoscopy and intubation. The highest rise was in control group.

Sanjeev Singh et al found that in Control, Lignocaine, Diltiazem and Esmolol groups, the percentage changes in heart rate (HR) from baseline and 1min after intubation were 30.45%, 26.00%, 7.01% and 1.50% respectively.[17]

The Percentage changes in mean arterial pressure from baseline and 1min after intubation were 20.83%, 15.89%, 10.90% and 10.20% in control, Lignocaine, Diltiazem and Esmolol groups respectively. The percentage changes in rate pressure product from baseline and at 1 min after intubation were 61.49%, 40.93%, 17.26% and 11.68% in Control, Lignocaine, Diltiazem and Esmolol groups respectively. Their results are similar to the results of the present study. The minor differences in results could be due to use of larger dose (2mg/kg) of esmolol and use of different induction agent (Thiopentone sodium) by them.

Agrawal P et al concluded that esmolol group when compared with its baseline values, showed significant rise in heart rate only at 1 minute and 2 minutes after intubation. In the diltiazem group, there was significant increase in heart rate after up to 4 minutes after intubation. In the lignocaine group, when compared to baseline values, showed significant increase up to 7 minutes after intubation.[18] In the present study, the rise in mean arterial pressure was significant up to 4 minutes in esmolol group, 4 minutes in diltiazem group but 5 minutes in lignocaine group. Their results are similar to our results with minor differences, probably due to use of lower dose of esmolol (1mg/kg) in their study and difference in timing of administration of the (3 minutes) drugs and smaller sample (n=25) size in their study.

Kumar S et al, found that Esmolol group showed no significant rise in heart rate at any time interval.[19] They found that esmolol was better than diltiazem for attenuating the heart rate with statistically significant difference. In the present study, it was also found that esmolol was significantly better than diltiazem for attenuating the heart rate till 5 minutes after laryngoscopy and intubation. The minor difference in result might be due to a use of larger dose of esmolol (2m/kg) and difference in time of administration of diltiazem (60 seconds) in their study.

Sarkar A et al, stated that rise in heart rate was similar in both esmolol and diltiazem group with no statistical significance between them at any time interval.[20] They also found that diltiazem causes greater fall in systolic blood pressure and diastolic blood pressure post-induction than esmolol and blunted the pressor response significantly better than esmolol. In the present study, the rise in systolic and diastolic blood pressure was less in diltiazem group as compared to esmolol group but this difference was insignificant for systolic blood pressure and significant only till 1 minute for diastolic blood pressure. Since, they have not specified the time at which the study drugs were administered before laryngoscopy, the difference in results from present study could be because the peak effect of drug administered did not correspond to timing of laryngoscopy in their study.

Rate pressure product is calculated by multiplying heart rate with systolic blood pressure and is a good estimate of myocardial oxygen requirement. The rate pressure product (RPP) levels close to 20,000 are normally associated with angina and myocardial ischemia ([16]). In the present study, the rate pressure product (RPP) following tracheal intubation was not more than 20,000 in any study group, suggesting that critical increases in rate pressure product (RPP) can be avoided by using esmolol, diltiazem or lignocaine prior to laryngoscopy and intubation. These findings confirmed their cardio-protective effect during laryngoscopy and endotracheal intubation.

Singh S et al, found marked elevation of rate pressure product in control group as compared to groups-Lignocaine, Diltiazem and Esmolol after laryngoscopy and intubation.[17] The percentage changes in rate pressure product from baseline and at 1 min after intubation were 61.49%, 40.93%, 17.26% and 11.68% in Control, Lignocaine, Diltiazem and Esmolol groups respectively. These results are similar to the present study.

Parvez G et al compared the rate pressure product between esmolol and diltiazem group and found that there was significant difference between them at different time intervals. Esmolol group showed lesser values at all time intervals. It was found in the present study that esmolol was significantly better than diltiazem for attenuating the rate pressure product at all time intervals.[21]

Mohan K et al found that in diltiazem(0.2mg/kg) group, rate pressure product was increased by an average of 2302, while in lignocaine (1.5mg/kg) group, it increased by 3933 after 1 minute of laryngoscopy and intubation.[22] The difference in rise of RPP between the two groups was statistically significant. In the present study, it was found that average increase in diltiazem group was 1800 and lignocaine group was 4259 after 1 minute of laryngoscopy and intubation, the difference was statistically significant between these two groups. The results of their study were similar to the present study.

Limitations of the study

- Use of non-invasive blood pressure monitoring-Invasive monitoring gives us time to time variability and exact readings while with Noninvasive blood pressure, there was a time lag present
- After reviewing the literature, it was observed that there is still no consensus on optimal dose of esmolol and diltiazem for attenuation of laryngoscopic pressor responses. More studies are required to determine the optimal dose of esmolol and diltiazem for attenuation of laryngoscopic pressor responses
- After reviewing the literature, it is seen that there is still no consensus on timing of administration of esmolol, lignocaine and diltiazem for attenuation of laryngoscopic pressor responses. More studies are required to determine the timing of administration of esmolol, diltiazem and lignocaine for attenuation of laryngoscopic responses.

CONCLUSION

It can be concluded from the present study that esmolol, being a beta receptor antagonist, has superior clinical efficacy than diltiazem on heart rate and diltiazem is superior to lignocaine for attenuating the heart rate. Both esmolol and diltiazem were equally effective in preventing the rise of mean arterial pressure after laryngoscopy and intubation and this was better than effect of lignocaine. Esmolol was better than diltiazem and they both were better than lignocaine for preventing the rise of rate pressure product after laryngoscopy and intubation.

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