



# International Journal of Medical Anesthesiology

E-ISSN: 2664-3774  
P-ISSN: 2664-3766  
[www.anesthesiologypaper.com](http://www.anesthesiologypaper.com)  
IJMA 2022; 5(1): 17-20  
Received: 09-10-2021  
Accepted: 14-11-2021

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## Comparison of effect of IV esmolol and IV labetalol for attenuation of hemodynamic response to laryngoscopy and intubation

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DOI: <https://doi.org/10.33545/26643766.2022.v5.i1a.336>

### Abstract

Most patients undergoing general anesthesia, laryngoscopy and endotracheal intubation are required that can cause a large haemodynamic stress response, caused by sympathetic adreno-medullary response due to cortisol, norepinephrine, and epinephrine production. These can lead to tachycardia, hypertension, occasional dysrhythmias, angina, myocardial infarction or stroke. Considering the clinical significance of these changes stress attenuation is needed to blunt these responses. This is done by a variety of medications like lidocaine, deep inhalation anesthetic, ganglion blocking drugs, calcium channel blockers, vasodilators, opioids, and adrenergic blockers. An observational comparative study was designed to compare the effectiveness and safety of intravenous esmolol 1 mg/kg with labetalol 0.4 mg/kg for suppression of haemodynamic response to laryngoscopy and endotracheal intubation.

**Keywords:** Hemodynamic response, esmolol, labetalol, endotracheal intubation

### Introduction

Despite the emergence of new airway devices in recent years, rigid laryngoscopy and endotracheal intubation still remains the gold standard in airway management. Direct laryngoscopy exerting pressure over the base of tongue by the laryngoscopy blade which stimulates proprioceptors, resulting in a significant proportionate increase in catecholamine that alters haemodynamic parameters. Passage of the tube through the trachea further exaggerates this response by somato-visceral reflex. Intubation of the trachea alters respiratory and cardiovascular physiology by both reflex response and by the physical presence of the endotracheal tube.

The response is transient occurring 30 seconds after intubation and lasting for less than 10 minutes. Prevention of this sympathetic responses is essential for smooth induction in general anaesthesia

Reid and Brace in 1940 and King *et al.* [1] in 1951 described circulatory response to tracheal intubation as reflex sympathoadrenal stimulation and showed that sympathetic reflex is provoked by stimulation of epipharynx and larynx. Tachycardia and hypertensive response may be hazardous in patients with hypertension, ischaemic heart disease and cerebrovascular disease where circulation is already jeopardized. To stop these pressor response, many pharmacological drug trials were done.

### Cardiovascular Response

Laryngoscopy and endotracheal intubation causes intense reflex increase in heart rate and blood pressure which are mediated by the sympathetic nervous system. Laryngoscopy alone causes increase in both systolic and diastolic blood pressures from supraglottic pressure stimulus. The hypertension is due to increased cardiac output rather than increase in SVR & is associated with transient rise in central venous pressure. Endotracheal intubation causes further cardiovascular response due to increase in catecholamine from infraglottic receptor stimulus which significantly increases HR.

### Materials and methods

After an institutional approval by the ethical committee of the institution, the comparative observational study was carried out.

Sixty healthy normotensive patients of American Society of Anesthesiologists (ASA) class 1 or 2, aged between 20-50 years of either sex and weighted between 40-70 kg, undergoing elective surgery under general anaesthesia with orotracheal intubation were included in this study. Patients with significant comorbidities, taking antihypertensive drugs, expected difficult intubation, having known allergy to study drug or requiring nasal intubation were excluded.

The sixty patients were randomly and equally allocated in the following two groups:

Group E: 30 patients were given intravenous esmolol (1 mg/kg) diluted up to 10ml with 0.9% normal saline, 2 minutes prior to induction.

Group L: 30 patients were given intravenous labetalol (0.4 mg/kg) diluted up to 10 ml with 0.9% normal saline, 5 minutes prior to induction.

### Parameters to be studied

Heart rate (HR), systolic (SBP), diastolic (DBP) and mean arterial blood pressure (MAP), rate pressure product (RPP), spo<sub>2</sub>, procedure related complications.

In all cases premedication, induction and maintenance of anaesthesia were similar in both groups. Haemodynamic parameters like HR, SBP, DBP, MAP, RPP and SPO<sub>2</sub> were monitored at baseline, after premedication, before induction, immediately after intubation (considered as '0' min) and 2, 3, 5, 10 minutes after intubation.

Patients were premedicated (10 minutes before induction) with

- Inj. Glycopyrrolate 0.004 mg/kg i.v
- Inj. Midazolam 0.02 mg/kg i.v
- Inj. Ondansetron 0.1 mg/kg i.v

After premedication, all vital parameters were recorded.

Patients were pre-oxygenated with 100% oxygen for three minutes. The Study drug was given as mentioned above. Then vital parameters were recorded before induction.

The patient was induced with 2.5% thiopentone sodium 5 mg/kg intravenously till the eyelash reflex was lost. Then injection succinylcholine 2 mg/kg iv was given. All intubations were completed within 15 seconds.

### Maintenance of Anaesthesia

Anaesthesia was maintained with N<sub>2</sub>O/O<sub>2</sub> mixture, sevoflurane and an intermittent vecuronium as per need of surgery.

### Reversal of Anaesthesia

After completion of surgery, residual neuromuscular blockade was reversed with inj. neostigmine 0.05 mg/kg i.v & inj. glycopyrrolate 0.01 mg/kg i.v. & patients were extubated.

### Study method

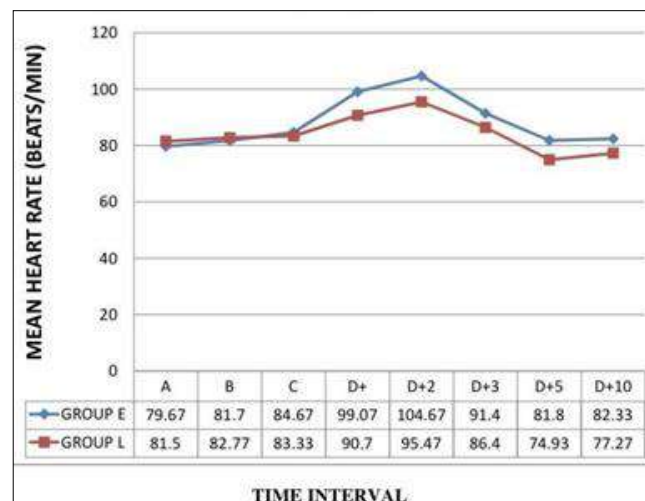
Results were tabulated as mean  $\pm$  SD. Paired t-test was used for statistical analysis. P-value of less than 0.05 was considered as statistically significant and less than 0.001 was considered as highly significant.

### Observation

#### Heart Rate

In our study, parameters of baseline, after premedication and before induction, there was no statistically significant difference in HR among both groups ( $P>0.05$ ). At 2

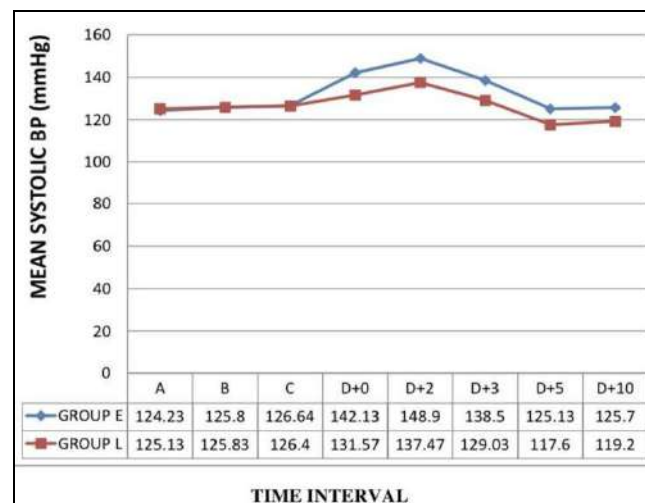
minutes after intubation, there was maximum increase in HR from baseline of 25 beats/min in group E and 13.97 beats/min in group L. Heart rate returned to near baseline in group E and below baseline in group L at 5 minutes post intubation. Group L showed better attenuation of HR than group E which was statistically highly significant ( $p<0.001$ ). Naveen N *et al.* [7] and Suruchi A *et al.* [4] studied effective attenuation of heart rate with IV esmolol 0.5 mg/kg and IV labetalol 0.25 mg/kg after intubation till 10 min.



Graph 1: Mean heart rate at different time intervals

### Systolic Blood Pressure

In our study there was no significant difference in SBP in both groups at baseline, after premedication and before induction ( $P>0.05$ ). There was maximum rise of SBP from baseline of 24.67 mmHg in group E and 12.34 mmHg in group L at 2 minutes post intubation. At 5 minutes after intubation mean SBP returned to near baseline in group E and below baseline in group L. SBP is better controlled with labetalol than esmolol which was statistically significant ( $p<0.001$ ). In a study by B Sawbhagya laxmi *et al.* [3] SBP was significantly less in patients receiving labetalol (0.5 mg/kg) compared to esmolol (1 mg/kg).



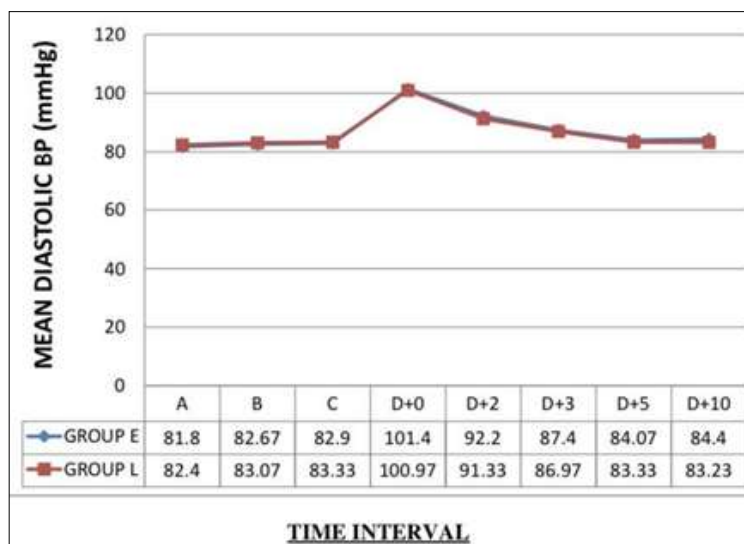
Graph 2: Mean systolic BP at different time intervals

### Diastolic blood pressure

In our study, there was no significant difference in DBP from baseline up to 10 minutes post intubation ( $P>0.05$ ).

among both groups. Maximum rise of DBP from baseline was 19.6 mmHg in group E and 18.57 mmHg in group L immediately after intubation. DBP returned to near baseline at 5 minutes after intubation in both groups. We concluded

that both the drugs didn't have much effect on diastolic blood pressure. Supportive to our findings, study by Singh SP *et al.* [5] and Mimiksha Giri *et al.* [6] (both used esmolol 0.5 mg/kg and labetalol 0.25 mg/kg) had similar results.



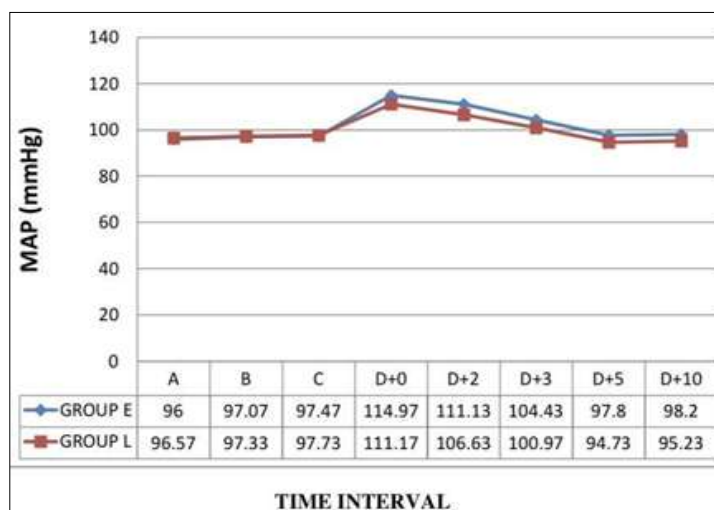
**Graph 3:** Mean diastolic BP at different time intervals

### Mean Arterial Pressure

Mean arterial pressure (MAP) is the average pressure throughout each heartbeat cycle. The MAP is defined as the diastolic blood pressure plus one-third of the difference between systolic and diastolic blood pressure.

MAP in both groups at baseline, after premedication and before induction was not significant ( $P > 0.05$ ). Immediately after intubation, there is a maximum increase in MAP of 18.97 mmHg in group E and 14.6 mmHg in group L from

baseline. Rise in MAP returned to near baseline in group E and below baseline in group L at 5 minutes post intubation. We can conclude that labetalol provide better control of MAP than esmolol which was highly significant ( $p < 0.001$ ). Atit kumar *et al.* [1] studied esmolol (1 mg/kg) and labetalol (0.4 mg/kg) and there is significant difference in MAP among both groups immediately after intubation and 2, 8 minutes post intubation.



**Graph 4:** Mean arterial pressure at different time intervals

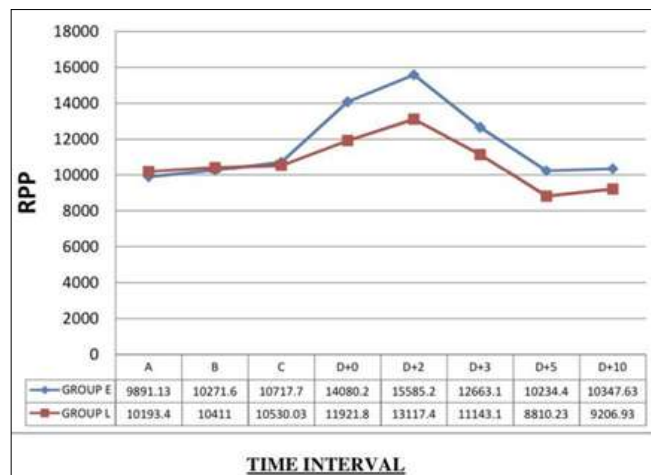
### Rate Pressure Product

Rate pressure product is a measure of the stress put on the cardiac muscle. RPP is HR multiplied by SBP.

In our study, values of RPP had not crossed the critical limit of 15,000. There was a maximum rise of 5694.07 in group E and 2924 in group L at 2 minutes post intubation from baseline. At 5th minute of intubation RPP returned to near

baseline in esmolol and fell below baseline in labetalol. RPP is better controlled with labetalol than esmolol which was statistically highly significant ( $p < 0.001$ )

In studies by Singh SP *et al.* [5] and Naveen N *et al.* [7] there was significant difference in RPP during and after intubation up to 10 minutes with esmolol (0.5 mg/kg) and labetalol (0.25 mg/kg), findings were same as in our study.



**Graph 4:** RPP at different time intervals

### Spo2

There was no significant difference among both groups throughout our study

### Complications

There was no incidence of hypotension, bradycardia, arrhythmia, bronchospasm in our study in both groups

### Conclusion:

In conclusion, labetalol in dose of 0.4 mg/kg is more effective when compared to esmolol in dose of 1 mg/kg for attenuation of haemodynamic responses of laryngoscopy and endotracheal intubation without any significant side effects.

### Funding

There are no organizations that funded this paper.

### Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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