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HOW MUCH EFFECTIVE ARE INTRAVENOUS ESMOLOL AND LIDOCAINE IN PREVENTING HAEMODYNAMIC STRESS RESPONSE TO LARYNGOSCOPY AND INTUBATION AS PREMEDICANT? - A COMPARATIVE CLINICAL EVALUATION

Anesthesiology

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ABSTRACT

Background and Aim: The present study was designed to evaluate and study the efficiency of intravenous esmolol and lidocaine (preservative free) in the attenuation of cardiovascular response to laryngoscopy and intubation in normotensive individuals.

Methods: 90 Patients were randomized in three groups, Group E (esmolol) 1.5mg/kg I.V, Group L (lidocaine) 1.5mg/kg I.V and Group C (active-control; midazolam) 0.02mg/kg I.V with 30 patients in each group. Hemodynamic parameters were recorded before the study drug and at specified intervals.

Results and observations: Both esmolol and lignocaine group showed less rise in HR and BP during laryngoscopy and intubation than control group. All haemodynamic parameters were also significantly less in Group-E and Group-L at 1,2, 3,4, 5 and 10 minutes after intubation.

Conclusion: Esmolol is a better agent than Lidocaine in preventing the cardiovascular stress response to laryngoscopy and intubation.

KEYWORDS

Esmolol, hemodynamic stress response, Intubation, Laryngoscopy, Lidocaine

INTRODUCTION

In modern day anaesthesia practice rigid laryngoscopy and tracheal intubation still remains the gold standard in airway management. The influence of airway manipulation on heart rate and blood pressure was recognized more than 50 years ago and the magnitudes of the changes were observed to depend on the depth of anaesthesia [1]. It is now well established that laryngoscopy and endotracheal intubation violate patient's protective airway reflexes and invariably cause hemodynamic changes associated with increased heart rate, increased blood pressure and occasional disturbances in cardiac rhythm.[2,3] These hemodynamic changes arise as a form of sympathoadrenal reflex and due to release of norepinephrine and, to a lesser extent, of epinephrine.[4]

In normotensive subjects these haemodynamic changes are short lived.[5] and probably of little significance. However, these haemodynamic alterations are hazardous to the patients with hypertension, myocardial insufficiency or cerebrovascular disease.[6] In patients with coronary artery disease it may lead to myocardial ischaemia and dysrhythmia because, increase in heart rate and blood pressure associated with laryngoscopy and endotracheal intubation may result in an increase in myocardial oxygen demand and also demand for increased coronary flow. In hypertensive patients these exaggerated responses may lead to left ventricular failure, pulmonary edema and congestive cardiac failure. In patients with intracranial aneurysm or dissecting aneurysm of the aorta, the increase in systemic blood pressure may cause rupture of vessels with life threatening consequences. The magnitude of response is greater with increasing force and duration of laryngoscopy. Elevation of blood pressure and heart rate typically starts within 5 seconds of laryngoscopy, peaks in 1 to 2 minutes and return to baseline level within 5-10 minutes.[7]

So, effective attenuation of the sympathoadrenal stress response is an important goal in anesthesiology. Various pharmacologic and nonpharmacologic method have been tried to limit the pressor response following the insertion of endotracheal tube [8]. The success rate is variable with different methods because each method has its own merits and demerits.

In several trials drugs like opioids, lidocaine, nitrates, calcium channel blockers, alpha-2 adrenergic agonists, beta blockers or magnesium have been used orally or parenterally to obtund these sympathoadrenal responses.

Esmolol is a cardioselective β1 adrenergic receptor blocker with rapid onset and a short duration of action. Esmolol has also been used in various studies [9,10] for attenuation of these reflexes. However, no consensus has been reached regarding optimum dose, the mode or timing of its delivery.[11] Lidocaine is a local anaesthetic drug rapidly cleared from bloodstream. Various studies have been shown that intravenous lidocaine administration prior to induction of anesthesia is effective in attenuating the arterial hypertension and tachycardia in response to endotracheal intubation [12-13]

Both lidocaine and esmolol have been used for the attenuation of adrenergic response to laryngoscopy and tracheal intubation.[14-15] In this active-controlled, randomized, prospective study an attempt has been made to observe, assess and compare the efficacy of a lesser dose of esmolol (0.5mg/kg body weight of intravenous bolus) and lidocaine (1.5mg / kg body weight of intravenous bolus) in attenuating the haemodynamic response following laryngoscopy and endotracheal intubation in three groups of adult patients of either sex undergoing upper abdominal surgeries under general anaesthesia. Midazolam (0.02mg/kg), a sedative, anxiolytic with rapid and short duration of action, has been used as active control here.

Methods:

This prospective, randomized, comparative study was conducted at a tertiary care hospital in Eastern India over a period of one year (January 2015-June 2016). We included 90 patients belonging to American Society of Anaesthesiologists (ASA) physical status (PS) I and II, of either sex, aged 20–40 years, weighing between 40 and 80 kg, scheduled for various elective upper abdominal surgeries under general anaesthesia with endotracheal intubation. Patients were explained about the procedure during the pre-anaesthetic visit. 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. Exclusion criteria for the study were patient's refusal, h/o allergy to either esmolol or lidocaine, patients with known cardiac disorders, other systemic disorders of lungs and liver, pregnant patients, patients for emergency procedures, BMI>30kg/m2, ASA grades III-IV, patients on antihypertensive drugs and patients with anticipated difficult airway. After obtaining Institutional ethical committee approval, eligible patients were randomly allocated using computer generated -randomized test to one of three equal (30 in each group) groups: the two study groups (E and L) and the active-control group (C).

Group-E: Patients received intravenous (i.v) Esmolol (0.5mg/kg) diluted in 10 ml normal saline 2 minutes before laryngoscopy and intubation.

Group-L: Patients received preservative free intravenous (i.v) Lidocaine (1.5mg/ kg of 2% solution) diluted in 10 ml normal saline 2 minutes before laryngoscopy and intubation.

Group-C: Patients received calculated dose of intravenous (i.v) Midazolam (0.02mg/kg) diluted in 10 ml normal saline 2minutes before laryngoscopy and intubation.

Anaesthetic technique

All the patients were subjected to pre-anaesthetic assessment including medical history and physical examination with relevant investigations. X-ray chest and ECG was also reviewed. On the day before surgery, all patients included in the study were reexamined and advised for 8 hours fasting state preoperatively. All patients were given tablet alprazolam 0.25mg orally at bedtime on the previous night of surgery.

On the day of surgery after confirmation of NPO status, patients were shifted to the operating room & connected to multiparameter monitor. Basal systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), heart rate (HR), ECG, oxygen saturation (SpO_2) were recorded. Continuous monitoring of the vital parameters were done. An intravenous line was secured with an appropriate sized cannula. Preloading with 500ml of ringer lactate over 30mins was done to maintain adequate hydration.

Inj. Glycopyrrolate 0.2mg i.v, Inj. ondansetron (0.1mg/kg),.and Inj. Fentanyl 2 mcg/kg body weight i.v. were given as premedication before surgery and patients were preoxygenated for 3 minutes with 100% O_2. Anaesthesia was induced with propofol 2mg/kg, followed by inj. vecuronium bromide 0.1mg/kg, to facilitate the direct laryngoscopy and intubation. . All the study drugs were prepared at the room temperature and the anaesthesiologist was unaware of its content. 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 anaesthesiologist. 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 and followed by intermittent dose of Inj. vecuronium, Inj. propofol, analgesics and intravenous fluids based on requirements. Intermittent positive pressure ventilation (IPPV) was continued by mechanical ventilator with tidal volume and respiratory rate adjusted to maintain an EtCO2 between 35-40 mm of Hg. Haemodynamic parameters (HR, SBP, DBP and MAP) were recorded by a blinded observer in the following specific time intervals-before the study drug injection (baseline value) and 1,2,3,4,5, and 10 minutes after intubation. No surgical stimulus was allowed during the study period and haemodynamic changes beyond the study period were not taken into account. At the end of surgery, the patients were adequately reversed with Inj.neostigmine 0.05 mg/ kg and Inj.glycopyrrolate 0.01 mg /kg intravenously as required. After oxygenation for about 5 minutes postoperatively patients were sent to the recovery room. The patients were monitored in the postoperative period for any complications and appropriately treated if required.

Statistical Analysis:

Sample size was decided in consultation with statisticians and based on previous studies which indicated that approximately 23-25 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 three groups, a total of 90 patients were incorporated for the present study. The results of the observations were tabulated, compiled and statistically analyzed using Statistica version 6 [Tulsa, Oklahoma: Stat Soft Inc., 2001] and SPSS Statistics version 17 [Illinois, Chicago: SPSS Inc., 2008]. Qualitative data (sex, ASA grade, postoperative complications) were compared between groups with Chi-Square ($\chi 2$) test. Quantitative data (age, body weight, height, HR, SBP, DBP, MAP) were compared between groups with ANOVA. A p value < 0.05 was considered as statistically significant and < 0.01 was considered as highly significant.

RESULTS:

In total 125 patients were screened for the study and 90 patients meeting the inclusion criteria and not having any exclusion criteria were randomized into three groups. The flow of the patients enrolled was represented in the CONSORT- flow diagram [Figure 1].

Demographic data

All the three groups were statistically comparable with respect to sex, age, body weight, height and ASA grading. No significant differences were observed between the groups (p value > 0.05) [Table 1].

Comparison of Heart rate (beats/min) between groups

When the preoperative baseline HR was compared between three groups, no statistically significant difference was found (p value 0.951). 1minute after intubation, significant reduction in HR is noted in Group-E and Group-L compared to Group-C(p value <0.01).HR was also significantly lower in Group-E and Group-L compared to Group-C(p value < 0.01) 2 min after intubation. The increases in HR at 1,2,3,4,5 and 10minutes after intubation were highly significant in Group C compared to Group-E and Group-L (p value < 0.01) [Table 2].

Comparison of SBP (mmHg) between groups

When the preoperative baseline SBP was compared between three groups, no statistically significant difference was found (p value 0.842). 1minute after intubation, significant reduction in SBP is noted in Group-E and Group-L compared to Group-C (p value <0.01). SBP was also significantly lower in Group-E and Group-L compared to Group-C (p value < 0.01) 2 min after intubation. The increases in SBP at 1,2, 3,4, 5 and 10minutes after intubation were highly significant in Group C compared to Group-E and Group-L (p value < 0.01) [Table 3].

Comparison of DBP (mm Hg) between groups

When the preoperative baseline DBP was compared between three groups, no statistically significant difference was found (p value 0.808). 1minute after intubation, significant reduction in DBP is noted in Group-E and Group-L compared to Group-C (p value <0.01). DBP was also significantly lower in Group-E and Group-L compared to Group-C (p value < 0.01) 2 min after intubation. The increases in DBP at 1,2, 3,4, 5 and 10minutes after intubation were highly significant in Group C compared to Group-E and Group-L (p value < 0.01) [Table 4].

Comparison of MAP (mmHg) between groups

When the preoperative baseline MAP was compared between three groups, no statistically significant difference was found (p value 0.891). 1minute after intubation, significant reduction in MAP is noted in Group-E and Group-L compared to Group-C (p value <0.01). MAP was also significantly lower in Group-E and Group-L compared to Group-C (p value < 0.01) 2 minute after intubation. The increases in MAP at 1,2, 3,4, 5 and 10minutes after intubation were highly significant in Group C compared to Group-E and Group-L (p value < 0.01) [Table 5]

Post operative complications

The HR in the postoperative period less than 60 bpm was considered as bradycardia. Two patients in Group-E and three patients in Group-L had HR below 60 bpm in the early postoperative period. Postoperative SBP less than 90mmHg, or DBP less than 60 mmHg, or both were considered as hypotension. It was seen in two patients in Group-E, three patients in Group-L and two patients in Group-C. Hypoxaemia was adjudged by any drop in SpO_2 below 90% and it was seen in six patients (two in each group) in the early postoperative period. Two patients in each group complained of shivering in the recovery room .Postoperative nausea was complained by two patients in Group-E, two patients in Group-L and four patients in Group-C.When these complications were compared between three groups with Chi-Square ($\chi 2$) test, no statistically significant difference was found (p value 0.6688) [Table 6].

DISCUSSION

Laryngoscopy and endotracheal intubation are associated with rise in heart rate, blood pressure and occasional disturbance in cardiac rhythm.[2,3] Although in normotensive subjects these responses of blood pressure and heart rate are transient and short lived,[5] they may prove to be detrimental in high risk patients especially in those with cardiovascular diseases, increased intracranial pressure and anomalies of the cerebral blood vessels.[6] Orotracheal intubation consists of two phases: Direct laryngoscopy and passing of endotracheal tube through the vocal cords and trachea.[16] It has been seen in various studies that increase in HR occurs during endotracheal intubation whereas the greatest increase in BP occurs during laryngoscopy.[17] Both sympathetic and parasympathetic element has been found as a mechanism to this intubation response. The sympathetic response is a polysynaptic pathway due to glossopharyngeal and vagus nerve forming the afferent arc to the sympathetic nervous system through the brain stem and spinal cord causing increased firing of the cardio-accelerator fibers and release of adrenergic mediators including norepinephrine, epinephrine, and vasopressin.[15] On the other hand, the parasympathetic reflex is monosynaptic, more common in children but can occur in some adults. The reflex is mediated by the increased

vagal tone at the SA node.[18] So, effective attenuation of haemodynamic response to laryngoscopy and tracheal intubation is of great importance in prevention of perioperative morbidity and mortality.

This randomized, active controlled study was undertaken to compare the usefulness of two drugs, esmolol, potent short acting β_1-adrenergic receptor blocker and lidocaine, a local anaesthetic in attenuation of the haemodynamic response following laryngoscopy and endotracheal intubation.

The factors influencing the cardiovascular changes associated with laryngoscopy and intubation are age, drugs, type and duration of procedures, depth of anaesthesia, hypoxia and hypercarbia.

Variations in heart rate to stressful events decrease with increasing age. Young patients show more extreme changes.[19] Marked fluctuations in hemodynamic response have been also reported in geriatric patients.[20,21] Therefore, patients with an optimal age range of 20 to 40 years were selected for this study. Difficult intubation takes longer time and is invariably associated with marked haemodynamic changes even in well premedicated patients. So, patients with higher Mallampati class (III and IV) were excluded from this study.

Patients on antihypertensive drugs were also excluded as they might exhibit a decrease in pressor response to laryngoscopy and intubation. Lidocaine extensively metabolized in liver and excreted in urine. Therefore, patients with altered liver functions and renal functions were not included in this study.

Patients with cardiovascular diseases are also excluded as these drugs can cause adverse cardiovascular complications. Patients on beta blocker therapy are also excluded because they might exhibit a decrease in pressor response to laryngoscopy and intubation.

The most significant factor influencing cardiovascular responses is the duration of laryngoscopy.[22] The force applied during laryngoscopy has only minor effect.[23] In this study the durations of laryngoscopy and intubation were limited to less than 15 seconds. During laryngoscopy adequate depth of anaesthesia was maintained avoiding hypoxia and hypercarbia.

1 minute after intubation, significant reduction in HR was noted in Group-L and Group-E compared to Group-C (p value 0.019) but there was no significant difference between Group-E and Group-L (p value > 0.05). HR remained significantly lower in Group-E and Group-L compared to Group-C (p value < 0.01). Esmolol had a better effect than lignocaine in controlling HR at rest of all points during the study (p value < 0.01).

Similar findings were observed by Oxorn D et al [24] who showed significant attenuation of increase in heart rate by single bolus dose of esmolol on laryngoscopy and endotracheal intubation, though effect on systolic BP, diastolic BP and mean BP was less significant. Incidence of ventricular arrhythmia was less with esmolol. Dyson et al [25] used esmolol in doses of 1,1.5 and 2mg/kg in controlling haemodynamic responses to laryngoscopy and intubation and observed that increase in heart rate was attenuated by all doses of esmolol.

Significant fall in NIBP (SBP, DBP and MAP) (p value < 0.05) were noted in Group E compared to Group-C after study drug administration and the values became highly significant (p value < 0.01) at rest of all time intervals throughout the study period. SBP and MAP values were significantly low(p value < 0.01) in Group-L compared to Group-C at 1,2,3,4,5 and 10 minutes postintubation. DBP values were also significantly lower(p value < 0.01) in Group-L compared to Group-C at 1,2,3,4 and 5minutes post intubation but not significant (p value 0.146) at 10 minutes post intubation. NIBP (SBP, DBP and MAP) were better maintained in Group-E than Group-L throughout the study period and values were significant (p value < 0.05) at 1,2,3,4,5 and 10 minutes post intubation time period. This findings corroborate with the result of the study by Koju RB et al[26] who showed that esmolol 1.4mg/kg I.V was significantly more effective in controlling the haemodynamic response following laryngoscopy and intubation in comparison to lidocaine 1.5 mg/kg. In Another study by Jain and Vats[15] concluded that intravenous lidocaine (2 mg/kg) and esmolol (2 mg/kg) are effective in attenuating the hemodynamic response to laryngoscopy and intubation for about 5 min without any deleterious effect**.** The important difference of the present study from other studies[9,10,14,15] was that a lower dose of esmolol (0.5mg/kg) i.v was used in current study, but beneficial effects were found to be similar.

In all groups peak haemodynamic surges were observed at 1 minute after intubation. HR increased from the baseline value by 2.4% and 12 % in Group E and Group L respectively. In control group a rise of 29.3% in HR was noted and this was statistically highly significant (p value < 0.01). Similarly, BP increased by 7.2% in Group L and 21.63% in Group C (p value < 0.01) from baseline value but decreased in Group-E by 2%.This one minute post intubation peak corroborate with the result of the study by Derbyshire DR et al [27] and Shribman AJ et al[7] who concluded that plasma catecholamine concentration increased to their maximum at 1 minute after laryngoscopy.

These altered pressor responses were normalized after 5 minutes in Group L and 10 minutes in Group C. This finding is again in agreement with the study by Shribman AJ et al[7] which showed that plasma catecholamine concentration comes down by 5 to 10 minutes after laryngoscopy. Haemodynamic parameters in Group E remained around the baseline value in first 1 to 3 minutes after intubation followed by a gradual fall in the next few minutes.

A few patients in all groups showed certain dysrhythmias during laryngoscopy and intubation on continuous ECG monitoring. These included mostly sinus tachycardia and ventricular premature contractions. However, none of these dysrhythmias reached alarming levels during the study to require treatment and converted to normal sinus rhythms spontaneously. It has been shown by various authors like Bedford RF [28] and Shribman AJ et al[7] that reflex autonomic responses provoked by laryngoscopy and endotracheal intubation could cause various types of dysrhythmias.

Regarding other complications, three patients in Group L and two patients in Group E had bradycardia in the early postoperative period. Intravenous atropine 0.5 mg was prescribed them to normalize the HR. Postoperative hypotension was seen in three patients in Group L, two patients in Group E and two patients in Group C. They were treated with intravenous colloid infusion. Hypoxaemia was seen in six patients (two in each group) in the early postoperative period, which was corrected by the administration of oxygen by face mask alone. None of them had to be intubated or required artificial ventilation postoperatively. Two patients in each group complained of shivering in the recovery room, which was resolved with warm blanket covering and oxygen supplementation. Postoperative nausea was complained by two patients in Group E, two patients in Group L and four patients in Group C. These patients were treated with intravenous ondansetron.

Limitations of the study

- Serum catecholamines are the most important markers to assess the sympathoadrenal stress response to any stimulus. But, in this study we could not measure its level in every patient due to scarcity of the resources. This was the major limitation of our 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 literatures, it was observed that there is still no consensus on optimal dose and timing of administration of esmolol and lidocaine for attenuation of laryngoscopic pressor responses. More studies are required to determine this.

CONCLUSION

From the observations and analysis of the present study, it can be inferred that both esmolol (0.5mg / kg body weight of intravenous administration 2 minutes prior to induction) and lidocaine (1.5mg/kg body weight of intravenous administration 2 min prior to induction) effectively prevent the haemodynamic response of laryngoscopy and intubation by limiting the extent of rises in heart rate and blood pressure. Esmolol, even in a lower dosage has been found to provide better haemodynamic stability than lidocaine. Both the study drugs are devoid of any serious adverse effect and found safe in this study.

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Conflicts of interest
There are no conflicts of interest.

Figure - 1 : CONSORT-FLOW diagram showing division of patients at every stage of trial

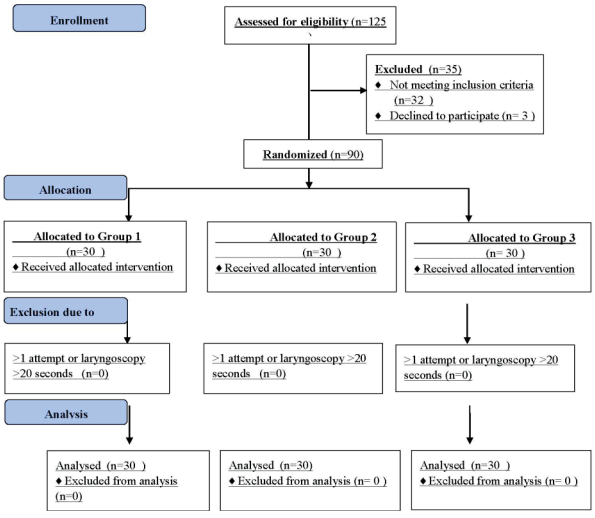

Table 1. Comparison of demographic variables between three study groups

Demographic Variables	**Group-E** n=30 (Mean±SD)	**Group-L** n=30 (Mean±SD)	**Group-C** N=30 (Mean±SD)	*p value*
Sex (M:F)	11:19	12:18	10:20	0.866
Age (YEARS)	39.07 ± 8.461	38.03 ± 7.902	39.20 ± 8.290	0.835
Body Weight (KG)	56.90 ± 9.935	56.07 ± 9.684	55.37 ± 9.197	0.826
Height (CM)	160.03± 10.361	161.63± 9.419	159.6 ± 9.568	0.705
ASA Grade (I:II)	24:6	24:6	23:7	0.936

SD : standard deviation

Table-2. Comparison of heart rates between the study groups at different points of time

TIME INTERVAL	**Group E** (n =30) (Mean ± SD)	**Group L** (n =30) (Mean ± SD)	**Group C** (n =30) (Mean ± SD)	p value
Before study drug injection (baseline) (T1)	84.00±9.124	83.47±9.035	83.27±9.490	0.951
1 minute after intubation (T2)	86.03±7.320	93.53±7.167	107.67±6.381	<0.01
2 minute after intubation(T3)	83.5±6.575	92.3±6.753	104.2±6.374	<0.01
3 minute after intubation (T4)	81.90±6.661	91.43±7.094	103.13±6.756	<0.01
4 minutes after intubation (T5)	80.2±6.789	87.9±7.292	97.67±7.685	<0.01
5 minutes after intubation (T6)	78.00±6.953	84.50±7.286	94.70±8.412	<0.01
10 minutes after intubation (T7)	73.93±6.933	80.10±6.588	84.37±8.286	<0.01

Table 3. Comparison of systolic blood pressures between the study groups at different points of time

TIME INTERVAL	**Group E** (n =30) (Mean ± SD)	**Group L** (n =30) (Mean ± SD)	**Group C** (n =30) (Mean ± SD)	**p value**
Before study drug injection (baseline) (T1)	122.47±12.219	121.60±11.764	120.63±12.366	0.842
1 minute after intubation (T2)	118.77±8.152	130.73±8.064	148.00±7.602	<0.01
2 minute after intubation(T3)	116.33±8.786	128.03±8.244	144.43±6.881	<0.01
3 minute after intubation (T4)	113.73±8.505	126.23±8.195	142.47±7.519	<0.01
4 minutes after intubation (T5)	111.97±8.248	121.13±8.685	134.97±7.863	<0.01
5 minutes after intubation (T6)	110.30±8.663	117.73±9.534	130.23±7.973	<0.01
10 minutes after intubation (T7)	106.60±8.427	112.53±9.504	119.97±7.867	<0.01

Table 4. Comparison of diastolic blood pressures between the study groups at different points of time

TIME INTERVAL	**Group E** (n =30) (Mean ± SD)	**Group L** (n =30) (Mean ± SD)	**Group C** (n =30) (Mean ± SD)	***p* value**
Before study drug injection (baseline) (T1)	79.73±9.472	80.83±9.436	79.27±9.667	0.808
1 minute after intubation (T2)	78.37±7.416	86.37±9.208	95.87±7.205	<0.01
2 minute after intubation(T3)	77.13±7.487	85.4±8.127	94.2±6.82	<0.01
3 minute after intubation (T4)	75.93±7.584	85.00±8.882	92.33±6.789	<0.01
4 minutes after intubation (T5)	73.9±7.522	81.6±8.908	87.43±6.862	<0.01
5 minutes after intubation (T6)	73.13±7.408	78.67±9.301	83.47±7.215	<0.01
10 minutes after intubation (T7)	69.57±7.496	75.03±9.050	78.83±6.518	<0.01

Table 5. Comparison of mean arterial pressures between three study groups at different points of time

TIME INTERVAL	**Group E** (n =30) (Mean ± SD)	**Group L** (n =30) (Mean ± SD)	**Group C** (n =30) (Mean ± SD)	***p* value**
Before study drug injection (baseline) (T1)	93.90±10.330	94.33±10.186	93.07±10.570	0.891
1 minute after intubation (T2)	91.83±7.488	101.13±8.513	113.20±7.184	<0.01
2 minute after intubation(T3)	90.17±7.593	99.6±7.972	110.8±6.8	<0.01
3 minute after intubation (T4)	88.57±7.610	99.23±8.625	109.07±6.807	<0.01
4 minutes after intubation (T5)	86.5±7.436	94.8±8.596	103.2±6.875	<0.01
5 minutes after intubation (T6)	85.50±7.510	91.73±9.255	99.00±7.334	<0.01
10 minutes after intubation (T7)	81.93±7.451	87.43±9.054	92.50±6.801	<0.01

Table 6. Comparison of post-operative complications between three study groups

Post-operative complications	**Group E (n =30)**	**Group L (n =30)**	**Group C (n =30)**	**Statistical Analysis**
Bradycardia	2	3	0	Chi-Square (χ^2) value 5.8070 p value 0.6688
Hypotension	2	3	2	
Hypoxaemia	2	2	2	
Shivering	2	2	2	
PONV	2	2	4	

PONV : postoperative nausea and vomiting

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