Attenuation of Cardiovascular Responses to Laryngoscopy and Intubation with Lignocaine and Esmolol in Patients Undergoing Elective Surgery

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ABSTRACT

BACKGROUND

Direct laryngoscopy and endotracheal intubation frequently induce a cardiovascular stress response characterized by hypertension and tachycardia due to reflex sympathetic discharge due to laryngotracheal stimulation which leads to increased plasma nor epinephrine concentration. The response is transient occurring 30 seconds after intubation and lasting for less than 10 minutes. Laryngoscopy and endotracheal intubation are associated with undesirable haemodynamic response which is of little significance in healthy patients but may be detrimental in patients with systemic diseases like hypertension, ischemic heart disease, stroke, perforated eye injury, increased intracranial tension etc. There is a need to attenuate these haemodynamic changes to decrease the mortality and morbidity. This study is designed to evaluate the attenuation of the haemodynamic response to laryngoscopy and endotracheal intubation with available cost effective drugs (esmolol and lignocaine) which are routinely used.

METHODS

This was an observational study conducted in the department of anaesthesiology, Travancore Medical College, Kollam among 140 patients in the age group 18 to 65 years posted for elective surgery from October 2017 to September 2018. Patients who received lignocaine or esmolol as intravenous agent prior to the induction of anaesthesia were recommended to group 'L' and 'E' respectively. Blood pressure and heart rate was recorded prior to laryngoscopy as well as 1 minute, 3 minutes, 5 minutes and 10 minutes after laryngoscopy and intubation. Collected data was tabulated and analyzed using appropriate statistical software (SPSS20).

RESULTS

The rise in heart rate, systolic BP, diastolic BP, and mean arterial pressure were better controlled by esmolol than lignocaine.

CONCLUSIONS

Intravenous esmolol 1.5mg/kg is found to be more effective in the attenuation of hemodynamic responses to laryngoscopy and endotracheal intubation than intravenous lignocaine 1.5mg/kg.

KEY WORDS

Laryngoscopy, Intubation, Attenuation, Cardiovascular Response, Lignocaine, Esmolol

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BACKGROUND

The circulatory response to laryngoscopy and intubation was described first by King in 1951.^{1,2} 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.³ The principle mechanism for hypertension and tachycardia is the sympathetic response which may be due to the increase in catecholamine activity. Direct laryngoscopy and endotracheal intubation frequently induces a cardiovascular stress response characterized by hypertension and tachycardia due to reflex sympathetic stimulation.

The response is transient occurring 30 sec after intubation and lasting for less than 10 min.⁴ It may be well tolerated in healthy people, but may be hazardous in patients with hypertension, tachycardia, myocardial infarction, and other complications. Various pharmacological approaches have been used to attenuate the pressure responses to laryngoscopy and tracheal intubation.⁵ It may predispose these patients for development of pulmonary edema, myocardial insufficiency, cerebrovascular accidents, acute left ventricular failure (LVF) and dysrhythmias. Pressor response is exaggerated in hypertensive patients even though rendered normotensive pre-operatively by antihypertensive medications.^{3,5}

There is a need to attenuate these hemodynamic changes to decrease the mortality and morbidity. This study is designed to evaluate the attenuation of the hemodynamic response to laryngoscopy and endotracheal intubation with available cost-effective drugs which are esmolol and lignocaine.

Esmolol is a striking option among the various beta-blockers, because of its cardio-selectivity and ultra-short duration of action (9 minutes), but it can only be administered intravenously. Lignocaine is commonly used agent for attenuating the circulatory responses associated with tracheal intubation.³ Studies have been conducted with various drugs like lignocaine, volatile anesthetics, calcium channel blockers, beta-blockers, clonidine and vasodilators, but none of them proved to be ideal to attenuate the responses.⁵

The hemodynamic response to the stress of laryngoscopy and endotracheal intubation does not present a problem for most patients. However, patients with cardiovascular or cerebral disease may be at increased risk of morbidity and mortality from the tachycardia and hypertension resulting from this stress.⁶ In patients ranked ASA PS 1, laryngoscopy and intubation lead to an average increase in blood pressure of 40% to 50%, and a 20% increase in heart rate. These changes, which are greatest one minute after intubation, last for 5 to 10 min. They are due to sympathetic and adrenal stimulation, which may also result in some arrhythmias. About half the patient with coronary artery disease experience episodes of myocardial ischemia during intubation when no specific prevention is undertaken.⁷ Perioperative myocardial infarction is a leading cause of postoperative morbidity and mortality due to hypertension and tachycardia.⁸

Abou-Madiet al⁹ found that the optimal dose of lignocaine to attenuate the response as 1.5 mg/kg given 3 minutes

before laryngoscopy. Miller et al¹⁰ found no benefit of intravenous lignocaine (1.5 mg/kg) given at 1, 2, or 3 minutes prior to laryngoscopy.

This study was aimed to compare the effects of lignocaine and esmolol to attenuate the cardiovascular stress response during laryngoscopy and intubation. And To assess the hemodynamic variations to laryngoscopy and intubation.

METHODS

After receiving approval from the Ethics Committee of the institution, the study was undertaken. This was an observational study conducted in the department of anaesthesiology, Travancore Medical College, Kollam among 140 patients in the age group 18 to 65 years posted for elective surgery from October 2017 to September 2018.

Inclusion Criteria

The study included patients belonging to:

- ASA-PS I or II
- Age between 18 and 65 years
- Mallampatti class I and II
- Only elective non-cardiac surgeries performed under general anesthesia.

Exclusion Criteria

Patients who were:

- At extremes of age
- Undergoing emergency surgery
- History of drug allergy
- Morbidly obese
- With difficult airway
- With cardiovascular diseases
- Heart rate less than 60/minute
- Systolic blood pressure less than 100mmHg
- Total duration of laryngoscopy and intubation more than 15 seconds were excluded from the study.

Preanesthetic Preparation

All patients underwent a pre-anesthetic evaluation with proper history-taking and physical examination of all the systems and airway. Pre-operative routine investigations such as hemoglobin, platelet count, hematocrit, total and differential count, serum electrolytes, RFT, RBS, blood grouping and Rh typing, chest radiography and electrocardiogram were done. Patients were advised to fast for 6 hours before surgery. Tablet Alprazolam 0.25mg was given the night before surgery to allay anxiety.

Anesthetic Technique

Patient identification was done following which a short preoperative history was taken. Then, clinical examinations and routine investigations were rechecked in all patients. Study objective and procedure were explained and informed written consent taken from each patient.

Intravenous access was secured and Ringer's lactate solution infusion started. The patients were pre-medicated with 0.004 mg/kg glycopyrrolate bromide intravenously 30 minutes prior to surgery. Then, the patient was shifted to the operating room after which routine non-invasive monitor was applied and vital signs monitored. Patients were preoxygenated for 3 minutes with 100% oxygen and then induced with 2 mg/kg propofol intravenously in incremental doses, 0.1 mg/kg vecuronium, followed by administration of the study drug (lignocaine or esmolol). Patient was ventilated with oxygen using IPPV with a fresh gas flow of 6 l/minute until intubation. After 3 minutes of administering the study drug, laryngoscopy was done using Macintosh laryngoscope blade and trachea was intubated with an appropriate size cuffed endotracheal tube. After confirmation of the endotracheal tube placement, anesthesia was maintained with oxygen:air (50:50) and sevoflurane 2%.

Heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure changes were recorded before induction(baseline), after induction and after tracheal intubation at 1, 3, 5 and 10 minutes for the study. No painting nor draping the patient was allowed till 10 minutes after the study drug administration. Fentanyl 2mcg/kg intravenous was given before surgery.

Statistical Analysis

Statistical analysis was done by using chi-square test and students t test and the Statistical Package for the Social Sciences (SPSS 20) software was used for the data analysis. $P < 0.05$ was considered as the level of significance.

RESULTS

In the present study, all the patients were between the age of 18 to 65 years. The mean age for group L was around 38.471 ± 8.8017 years and for group E around 40.771 ± 8.0076 years of age in which there was no statistically significant difference in terms of age. It was observed that both groups were comparable ($p = 0.108$, non significant) with respect to mean age of patient. It was observed that out of the total patient in group L, 41% were males and 29% were female, in group E, 38% were males and 32% were females. Statistically, both the groups were similar with respect to the gender ($p = 0.367$, non significant).

In the present study it was observed that out of the total patients, in group L, 46 (65%) were ASA grade 1 and 24 (34.2%) were ASA grade 2, and in group E, 42 (60%) were ASA grade 1 and 28 (40%) were ASA grade 2. Statistically there was no significant difference ($p = 0.30$) in the two groups in terms of ASA grading. Series 1 - ASA 1, Series 2 - ASA 2

In the present study it was observed that out of the total patients, in group L, 49 (70%) were Mallampatti class 1 and 21 (30%) were Mallampatti class 2, and in group E, 40 (57.1%) were Mallampatti class 1 and 30 (42.8%) were Mallampatti class 2. Statistically there was no significant difference ($p = 0.08$) in the two groups in terms of Mallampatti classification. Series 1 - Mallampatti class I, Series 2 - Mallampatti class II

Hemodynamic Variables

Hemodynamic parameters were recorded at: Baseline value (Baseline readings taken before intravenous esmolol 1.5mg/kg and lignocaine 1.5g/kg was given). At induction, then 1 minute, 3 minutes, 5 minutes and 10 minutes after laryngoscopy and intubation.

Heart Rate	Group	N	Mean	Std. Deviation	P value (Students t test)
Baseline	Lignocaine	70	79.200	4.3091	0.057
	Esmolol	70	80.543	3.9515	
At induction	Lignocaine	70	73.471	4.7081	0.0001
	Esmolol	70	77.786	5.0930	
1 min after intubation	Lignocaine	70	91.329	4.1867	0.0001
	Esmolol	70	84.800	3.6578	
3 min after intubation	Lignocaine	70	87.543	4.1448	0.0001
	Esmolol	70	81.814	3.0564	
5 min after intubation	Lignocaine	70	84.529	4.2723	0.0001
	Esmolol	70	79.243	3.1689	
10 min after intubation	Lignocaine	70	82.086	3.8476	0.0001
	Esmolol	70	76.843	3.8735	

Table 1. Comparison of changes in Mean heart rate at different interval in two groups.

Table 1: Displays the changes in the mean heart rate at different intervals:- Mean heart rate at baseline in lignocaine group was 79.200 ± 4.3091 beats per minute (bpm) and in esmolol group was 80.543 ± 3.9515 beats per minute (p value > 0.05 , non-significant). At induction, comparison of heart rate in lignocaine group was 73.471 ± 4.7081 and in esmolol group was 77.786 ± 5.0930 (p value > 0.05 , non-significant).

After laryngoscopy and intubation, at 1st, 3rd, 5th and 10th minutes, comparison of heart rate in lignocaine group was 91.329 ± 4.1867 , 87.543 ± 4.1448 , 84.529 ± 4.2723 , 82.086 ± 3.8476 respectively and in esmolol group at 1st, 3rd, 5th and 10th minutes were 84.800 ± 3.6578 , 81.814 ± 3.0564 , 79.243 ± 3.1689 , 76.843 ± 3.8735 respectively (p value < 0.05) which was statistically significant.

Mean heart rate at baseline in lignocaine group was 79.200 ± 4.3091 beats per minute and in esmolol group was 80.543 ± 3.9515 beats per minute (p value > 0.05 non-significant).

At induction, the comparison of heart rate in lignocaine group was 73.471 ± 4.7081 beats per minute and in esmolol group was 77.786 ± 5.0930 beats per minute (p value > 0.05 , non-significant).

After laryngoscopy and intubation at 1st, 3rd, 5th and 10th minutes, comparison of heart rate in lignocaine group was 91.329 ± 4.1867 , 87.543 ± 4.1448 , 84.529 ± 4.2723 and 82.086 ± 3.8476 respectively and in esmolol group at 1st, 3rd, 5th and 10th minutes were 84.800 ± 3.6578 , 81.814 ± 3.0564 , 79.243 ± 3.1689 and 76.843 ± 3.8735 respectively (p value < 0.05) which were statistically significant. None of the patient in any of the study group developed bradycardia by the end of 10th minutes of intubation and pulse rate was not less than 60 beats per minute in any of the readings.

Thus, attenuation of pressor response (rise in mean heartrate) is better in esmolol group than in lignocaine group. The mean heart rate comes near to baseline in Esmolol group at 5 minutes, while it is higher in lignocaine group at all intervals. Esmolol at dose of 1.5mg/kg provided a reliable and consistent attenuation against the increase in heart rate.

Systolic BP	Group	N	Mean	Std. Deviation	P value (Students t test)
Baseline	Lignocaine	70	123.071	9.3224	0.457
	Esmolol	70	124.200	8.5527	
induction	Lignocaine	70	114.186	11.4212	0.006
	Esmolol	70	119.014	8.7865	
1 min after intubation	Lignocaine	70	143.243	7.1311	0.0001
	Esmolol	70	129.514	7.6533	
3 min after intubation	Lignocaine	70	139.943	6.6461	0.0001
	Esmolol	70	125.971	7.4794	
5 min after intubation	Lignocaine	70	136.186	6.5282	0.0001
	Esmolol	70	121.971	7.4093	
10 min after intubation	Lignocaine	70	130.514	6.2780	0.0001
	Esmolol	70	117.143	7.5801	

Table 2. Comparison of changes in Mean SBP (mmHg) at different time interval in two groups.

Table 2:- Displays the changes in mean SBP at different time intervals in the two groups. It was seen that the baseline mean SBP in Lignocaine group was 123.071 +/- 9.3224 mmHg and Esmolol group was 124.200 +/- 8.5527 mmHg (p value >0.05 nonsignificant). At induction, values for mean SBP in Lignocaine and Esmolol group were 114.186 +/- 11.4212 mmHg and 119.014 +/- 8.7865 mmHg respectively. After intubation at 1st, 3rd, 5th and 10th minute comparison of mean SBP in Lignocaine group were 143.243 +/- 7.1311, 139.943 +/- 6.6461, 136.186 +/- 6.5282 and 130.514 +/- 6.2780 mmHg respectively and in Esmolol group were 129.514 +/- 7.6533, 125.971 +/- 7.4794 (p value <0.05, significant). Maximum attenuation in mean SBP is achieved by Esmolol group (1.5 mg/kg IV bolus) as compared to Lignocaine group (1.5 mcg/kg IV bolus) was at first minute only. Esmolol gives consistent and reliable fall in mean SBP than Lignocaine groups at all intervals.

Diastolic BP	Group	N	Mean	Std. Deviation	P value (Students t test)
Baseline	Lignocaine	70	77.61	6.789	0.084
	Esmolol	70	79.39	5.123	
At induction	Lignocaine	70	73.40	6.931	0.052
	Esmolol	70	75.41	5.069	
1 min after intubation	Lignocaine	70	90.23	6.179	0.0001
	Esmolol	70	83.26	4.306	
3 min after intubation	Lignocaine	70	88.16	6.142	0.0001
	Esmolol	70	80.81	4.084	
5 min after intubation	Lignocaine	70	85.46	5.750	0.0001
	Esmolol	70	77.19	4.278	
10 min after intubation	Lignocaine	70	81.143	5.9595	0.0001
	Esmolol	70	73.100	4.0366	

Table 3. Comparison of changes in Mean DBP (mmHg) at different time interval in two groups

Table 3: displays the changes in mean DBP at different time interval compared to the baseline in the two groups. It was seen that the baseline mean DBP in Lignocaine group was 77.61 +/- 6.789 mmHg and Esmolol group was 79.39 +/- 5.123 mmHg (p value >0.05 non-significant). At induction, mean DBP in Lignocaine and Esmolol group were 73.40 +/- 6.931 mmHg and 75.41 +/- 5.069 mmHg respectively (p value > 0.05 non-significant). After intubation at 1st minute, 3rd minute, 5th and 10th minute, the mean DBP in Lignocaine group were 90.23 +/- 6.179 mmHg, 88.16 +/- 6.142 mmHg, 85.46 +/- 5.750 mmHg and 81.143 +/- 5.9595 mmHg respectively and in Esmolol group, the mean DBP were 83.26 +/- 4.306 mmHg, 80.81 +/- 4.084 mmHg, 77.19 +/- 4.278 mmHg and 73.100 +/- 4.0366 mmHg respectively (p value < 0.05) which was statistically significant.

Esmolol has a better control of the diastolic BP from the first minute after intubation and also, there is a better control of the diastolic blood pressure at different intervals as compared to lignocaine (p value < 0.05).

MAP	Group	N	Mean	Std. Deviation	P value (Students t test)
Baseline	Lignocaine	70	92.69	5.747	0.051
	Esmolol	70	94.39	4.358	
At induction	Lignocaine	70	86.83	6.195	0.001
	Esmolol	70	89.90	4.354	
1 min after intubation	Lignocaine	70	107.74	4.409	0.0001
	Esmolol	70	98.66	3.926	
3 min after intubation	Lignocaine	70	105.33	4.252	0.0001
	Esmolol	70	95.89	3.775	
5 min after intubation	Lignocaine	70	102.24	4.080	0.0001
	Esmolol	70	92.03	3.938	
10 min after intubation	Lignocaine	70	97.50	4.207	0.0001
	Esmolol	70	87.81	3.785	

Table 4. Comparison of changes in Mean MAP (mmHg) at different time interval in two groups

Table 4: displays the changes in MAP at different time interval compared to the baseline in the two groups. It was seen that the baseline mean MAP in Lignocaine group was 92.69 +/- 5.747 mmHg and in Esmolol group was 94.39 +/- 4.358 mmHg (p value >0.05 non-significant). At induction, MAP in Lignocaine group was 86.83 +/- 6.195 mmHg and in Esmolol group was 89.90 +/- 4.354 mmHg respectively. After intubation at 1st, 3rd, 5th and 10th minutes comparison of MAP in Lignocaine group was 107.74 +/- 4.409 mmHg, 105.33 +/- 4.252 mmHg, 102.24 +/- 4.080 mmHg and 97.50 +/- 4.207 mmHg respectively. After intubation at 1st, 3rd, 5th and 10th minutes comparison of MAP in Esmolol group was 98.66 +/- 3.926 mmHg, 95.89 +/- 3.775 mmHg, 92.03 +/- 3.938 mmHg and 87.81 +/- 3.785 mmHg respectively (p value <0.05, significant).

Esmolol shows a better attenuation of the mean arterial pressure than lignocaine at different intervals (p < 0.05, significant). On observation, it is seen that esmolol produces a better attenuation of the hemodynamic variables than lignocaine. There is a better control of the HR and the BP by esmolol following laryngoscopy and intubation, which is done after 3 minutes of administration of esmolol. It shows that we get a better control of the stress response with 1.5 mg/kg of esmolol given 3 minutes prior to laryngoscopy and intubation than 1.5 mg/kg of lignocaine given 3 minutes before laryngoscopy and intubation.

DISCUSSION

Orotracheal intubation consists of two phases: Direct laryngoscopy and passing of endotracheal tube through the vocal cords and trachea. It has been seen in various studies that increase in HR occurs during endotracheal intubation whereas the greatest increase in BP occurs during laryngoscopy. 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. The net effect of this autonomic surge is an increased BP, HR, pulmonary artery wedge pressure, and decreased ejection fraction. 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.^{6,11}

In 1981, Russell et al monitored the changes in arterial pressure and arterial concentrations of noradrenaline, adrenaline and dopamine were monitored in 16 patients undergoing endotracheal intubation. Significant increases in mean arterial pressure and plasma noradrenaline were noted. The increases in arterial pressure were associated with increases in noradrenaline concentrations. Adrenaline and dopamine concentrations did not change significantly following intubation. The results suggest a predominantly sympathetic response during intubation and the need for prophylaxis in patients at risk¹² In 1983, Derbyshire et al measured the plasma adrenaline and noradrenaline concentrations in 24 patients during the induction of anaesthesia and the subsequent tracheal intubation. The patients received either suxamethonium 1 mg kg⁻¹ or pancuronium 0.1 mg kg⁻¹ to facilitate tracheal intubation. Mean arterial pressure (MAP) increased in both groups following laryngoscopy and tracheal intubation and there were concomitant increases in the plasma catecholamine concentrations, the changes being more marked in the suxamethonium group. There was a significant correlation between MAP and plasma catecholamine concentrations in the suxamethonium group. Measurement of plasma catecholamine concentrations in samples obtained simultaneously from central venous, peripheral venous and arterial sites were in broad agreement; the greatest changes occurred in central venous samples.¹³

Both HR and BP are determinants of oxygen delivery and demand. An increase in HR deleteriously affects both supply and demand of oxygen. BP is related to cardiac output (CO) and systemic vascular resistance (SVR). A change in either CO or SVR will result in a compensatory change in the other. Hypertension can, therefore, also affect both supply and demand.¹⁴ The strict control of hemodynamic variables has reduced myocardial ischemia, while hemodynamic aberrations, such as tachycardia, systolic hypertension and hypotension, elevated RPP and MBP/HR ratio of less than one may cause ischemia, RPP is correlated with myocardial oxygen demand and a threshold value of RPP had been correlated with the onset of angina. During anaesthesia, myocardial ischemia is poorly correlated with RPP.

With the development and usage of ICP monitoring devices such as the subarachnoid pressure screw and the intraventricular catheter, the intracranial effects of laryngoscopy and intubation have been studied. Burney and Winn first demonstrated that laryngoscopy and intubation could cause an increase in ICP in patients undergoing a craniotomy.¹⁵ These patients had a subarachnoid pressure screw placed for ICP monitoring. They found a rise in ICP in every patient and that the peak rise in ICP occurred within 1 minute for all but one patient (93%). The increase in ICP from pre-induction measurements ranged from 8 to 40 cm H₂O.

Suctioning, similar to laryngoscopy and intubation, seems to induce a comparable hemodynamic response. Endotracheal suctioning has also been shown to stimulate the cough reflex.¹⁶ It is hypothesized that coughing can cause an increase in intrathoracic pressure that is transmitted into the cerebral venous system and ultimately can lead to a rise in cerebral venous pressure, intracranial volume, and ICP.¹⁷ Hamill et al. compared intravenous with laryngotracheal lidocaine in 22 patients undergoing elective craniotomy. Using a subarachnoid pressure screw to measure ICP, they

found that 1.5 mg/kg of intravenous lidocaine attenuated the increases in ICP, while laryngotracheal lidocaine did not.¹⁸

Esmolol appears to be an appropriate choice of agent for attenuating the hemodynamic response to laryngoscopy and tracheal intubation, due to its cardioselective property, rapid onset of action and short elimination half-life (9 min) along with no significant drug interaction with commonly used anesthetics. Esmolol decreases the force of contraction and HR by blocking beta-adrenergic receptors of the sympathetic nervous system which are found in the heart, blood vessels, and other organs of the body. Esmolol prevents the action of two naturally occurring neurotransmitters epinephrine and norepinephrine, thereby attenuates the tachycardia and hypertensive responses to laryngoscopy and tracheal intubation.¹⁹ Esmolol (betaadrenergic receptor antagonist + ultra-short-acting) provides hemodynamic stability during laryngoscopy and tracheal intubation without side effects.²⁰ Kindler et al. found that esmolol administration before laryngoscopy was sufficient to control HR after intubation, but it did not affect SAP.

Ebert et. al., found, in 68 patients undergoing general anesthesia for elective surgery, that esmolol (loading dose of 500 mg/kg/min for 4 minutes, followed by a maintenance infusion of 300 mg/kg/min for 11 minutes) significantly attenuated the maximum increases in HR and BP when compared with placebo.²¹ Korpinen et al in 1998,²² on the basis of the study concluded that propofol-alfentanil anesthesia combined with esmolol is a satisfactory method to meet specific demands of laryngomicroscopy in young and middle-aged ASA-PS I-II patients. However, a combination of propofol and esmolol showed a tendency to decrease both the heart rate and arterial pressure and a caution is necessary when the combination were used in elderly patients.

Among the various drugs used, lidocaine has received the most attention. While several studies have demonstrated its ability to decrease the cough reflex, HR and BP increases,²³ and ICP increases occurring with laryngoscopy, endotracheal intubation, and suctioning, other studies have suggested the lack of efficacy of lidocaine.²⁴ It has been used as a lidocaine gargle for oropharyngeal anesthesia, as a lidocaine aerosol for intratracheal anesthesia,²⁵ or as an intravenous bolus for systemic anesthesia.²⁶ Intravenous lidocaine, in particular, has been found to suppress the cough reflex,²⁷ to prevent increases in intracranial pressure,²⁸ to attenuate circulatory responses and to possess antiarrhythmic properties.²⁹

In our study, there is a better control of HR, SBP, DBP, and MAP with esmolol than lignocaine. The heart rate comes to around baseline after 5 minutes of tracheal intubation. In the case of SBP, DBP and MAP, there is a significant difference in the attenuation of cardiovascular responses by both the drugs ($p < 0.0001$), a better control provided by esmolol than lignocaine. This study is comparable to the study conducted by M Begum, P Akter demonstrated highly significant reduction in HR, DBP, RPP and MAP in both groups ($p < 0.01$), 2 and 4 minutes after induction. But the SBP reduction was only statistically significant ($p < 0.05$). In group-E patients, these reductions were more than that of in group-L patients. Four minutes after intubation, HR, SBP, DBP, RPP and MAP returned to almost baseline values in esmolol group. Singh H, Vichitvejpaisal P, et al³⁰ compared the effects of the lidocaine, esmolol, and nitroglycerin and showed lidocaine 1.5 mg/kg i.v. and nitroglycerin 2 micrograms/kg i.v. were ineffective in

controlling the acute hemodynamic response following laryngoscopy and intubation. Esmolol 1.4 mg/kg i.v. was significantly more effective than either lidocaine or nitroglycerin in controlling the HR response to laryngoscopy and intubation ($p < 0.05$). Following laryngoscopy and intubation, MAP increased in all the group, lignocaine (55% $\pm$ 26%) heart rate was significantly lower (and esmolol (25% $\pm$ 11%) compared with preinduction baseline values. In esmolol pretreated patients, the increase in heart rate was significantly lower (20% $\pm$ 3%) compared with lignocaine (52% $\pm$ 8%).

Feng C K, Chan K H in their study^[28] showed that only esmolol could reliably offer protection against the increase in both HR and SBP and 2 mg/kg lidocaine had no effect to blunt adverse hemodynamic response during laryngoscopy and tracheal intubation which is in agreement with our study. It is also comparative with another study conducted by Ajay Gupta, Renu Wakhloo, showing a better control of HR, DBP and SBP obtained with esmolol compared to lignocaine. Christopher H. Kindler, Schneider³¹ studied the effects of intravenous lidocaine and/or esmolol on hemodynamic responses to laryngoscopy and intubation and found that esmolol 1 to 2 mg/kg is reliably effective in attenuating HR in tracheal intubation.

Limitations

- Varying degree of resting sympathetic tone of patients can cause interference with the readings.
- ASA grade III and IV patients especially with IHD, MI, HTN were not included in study.
- As our sample size is only of 140 patients, so this study cannot be generalized to all ASA I and II patients and further studies with larger sample size is needed.
- Infusion of study drugs after bolus might have yielded better results than single bolus dose of study drugs. Which require more studies in future.
- Influence of premedication with glycopyrrolate, which cause tachycardia might have interfered with the readings.

CONCLUSIONS

Thus, from our study we conclude that in patients with ASA-PS grade I and II, intravenous bolus dose of Lignocaine (1.5mg/kg) and Esmolol(1.5mg/kg) given 3 minute prior to laryngoscopy and intubation is safe and effective prophylactic method for attenuating hemodynamic response to laryngoscopy and intubation. Esmolol provides reliable and consistent protection against rise in pulse rate and blood pressure. Esmolol provides better cardio-protection in patients against hyperadrenergic responses to laryngoscopy and endotracheal intubation. Esmolol appears to be the drug of choice in maintaining hemodynamic stability during laryngoscopy and intubation.

Data sharing statement provided by the authors is available with the full text of this article at jemds.com.

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