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Assessment of Hemodynamic Responses to Intubation Using Esmolol Versus Lignocaine: A Prospective Randomized Comparative Study

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ABSTRACT

Background: Laryngoscopy and intubation produce marked sympathetic stimulation and transient increases in HR and BP, which are generally well tolerated by healthy patients, but may precipitate myocardial ischaemia, arrhythmias or a stroke in an already unstable patient. Numerous agents have been tried to attenuate this pressor response, including most commonly esmolol (a short acting β1 selective adrenergic blocker), and lignocaine (a common local anaesthetic with class 1B antidysrhythmic activity).
Objectives: The present study was aims study aimed to evaluate and compare the efficacy of intravenous esmolol and intravenous lignocaine for attenuation of laryngoscopy and endotracheal intubation induced changes in heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure.
Methods: This prospective randomised comparative study will be conducted on 100 patients undergoing elective surgeries under general anaesthesia over period of 12 months. Patients will be randomly divided into 2 equal groups (n=50).
Group E - patients receiving i.v esmolol just before laryngoscopy,
Group L - patients receiving i.v lignocaine just before laryngoscopy.
Heart rate, systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial pressure(MAP)will be observed pre operatively to regular intervals till 10min after intubation. Primary outcome variable will be attenuation of heart rate and blood pressure response after intubation. Secondary outcome variable will include bradycardia, hypotension,arrhythmias and need for intervention.
Results: Both esmolol and lignocaine should moderate the pressor response to laryngoscopy and intubation; however, esmolol should do this to a greater degree, particularly as far as heart rate and mean arterial pressure are concerned, because of its β1 selective blocking action. Lignocaine may be more moderate in its effects but should not prove as effective as esmolol. Both should thus be safe and well tolerated.
Conclusion: Esmolol is probably more effective than lignocaine in controlling the hemodynamic 'stresses' particularly heart rate and mean arterial pressure, to laryngoscopy and intubation. Of course the rapidity of onset and short duration of action make it ideal for controlling very transient sympathetic, or sympathetic-like 'stresses.

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***Keywords**: Laryngoscopy; Endotracheal intubation; Hemodynamic response; Esmolol; Lignocaine; Pressor response; General anesthesia.*

INTRODUCTION

Security of the airway by laryngoscopy and endotracheal intubation is an inevitable feature of the practice of general anaesthesia. These manoeuvres stimulate the epipharyngeal and laryngopharyngeal structures, inducing a baroreceptor response which evokes a characteristic sympathetic surge with tachycardia, hypertension and myocardial increased O2 demand. These changes are perfunctory in the healthy and the young but potentially harmful in patients with cardiovascular disease, hypertension, ?ischaemic heart disease and/or intracranial pathology [1,2]. The hemodynamic response to intubation is primarily mediated by catecholamines released as a result of sympathetic stimulation shunting through the paravertebral chain. The act of direct laryngoscopy stimulates this particularly in the supraglottic tissues whilst passage of an endotracheal tube through the vocal cords also tends to accentuate the afferent response to that region. Factors affecting the level of this response are depth of anaesthesia, length of time laryngoscopy is performed, the disease of the patient and hence the plane of surgery, and induction agents [3,4]. Over the years various methods have been suggested to minimise the pressor response to intubation anxiolytic, deepening the inhalational anaesthesia, vagolytics, opiates, vasodilators, calcium channel blockers, α2 aAdrenoreceptors, β-adrenoreceptors. Recently β- blockers have received attention as blunting the sympathic irritibility with nicely selective effects [5]. Of special interest in this class is Esmolol, this new wonder drug in the rave of the anaesthetic community is a selective β1- blocker which is short-acting and has been tried in various doses in a number of studies. The failings of these studies relate to the other drugs and agents being given during induction and the diversity of anaesthetic techniques used. The pharmacokinetics of Esmolol make it ideal for use in transient situations such as intubation and initiation of anaesthesia [6].

Lignocaine can similarly be given intravenously to blunt the hemodynamic response to airway manipulation. How this works is not clearly understood but may relate to general depression of airway reflexes and thereby reduced sympathetic outflow; lignocaine also has antiarrhythmic properties and may therefore confer greater haemodynamic stability during its use [7]. It remains to be defined, however, how well it controls the heart rate and blood pressure as compared to the β-blockers. In addition the importance of controlling the pressor response in the patients who are more likely to have a critical statistics “sensitive” fluctuation of heart rate and blood pressure, makes it important to identify the agent which is most efficacious and safe, making a study comparing esmolol against lignocaine directly a useful exercise [8]. A prospective randomized comparative study between intravascular esmolol and lignocaine administered in the Leyu study outcome to attenuate hemodynamic responses to laryngoscopy and endotracheal intubation in patients undergoing elective surgery under general anaesthesia were carried out.

MATERIALS AND METHODS

Study design and setting

This prospective randomized comparative study was conducted over a period of 12 months at our tertiary care teaching hospital after obtaining a clearance from the Institutional Ethics Committee with an aim to study the comparative efficacy of esmolol and lignocaine in blunting the hemodynamic response to laryngoscopy and intubation.

Sample size and study population

We recruited a total of 100 patients into the study. We selected the sample size (100 patients) to be feasible within the timescale we were working to at the time of study whilst, at the same time, leaving us with sufficient power to detect differences between the groups if they existed. Patients were randomised into 2 equal groups of 50 patients.

- Group E (n = 50): Received intravenous esmolol
- Group L (n = 50): Received intravenous lignocaine

Inclusion criteria

Eligible subjects were patients between the ages of 18 and 60, of both sexes, who were of ASA physical status I or II, and were scheduled for elective procedures requiring general anesthesia with ETI.

Exclusion criteria

Patients with cardiovascular disease, hypertension, arrhythmia, bronchial asthma, anticipated difficult intubation, obesity (BMI > 30 kg/m2), patients on β-blockers and Ca channel blockers, those with hepatic or renal dysfunction as well as pregnant and/or lactating patients were excluded from the study. Patients known to be hypersensitive to esmolol or lignocaine were also excluded.

Preoperative preparation

All patients had the usual preanesthetic assessments. Fasting protocol followed standard guidelines. No sedative premedication was administered prior to recording the baseline hemodynamic parameters.

Randomization and drug administration

Randomization was by a computer-generated randomization sequence. Concealment was by sealed opaque envelopes; opened prior to induction.
Group E - esmolol group: 1 mg/kg esmolol unloaded to a 10 mL syringe and given intravenously over 30 seconds 2 minutes prior to laryngoscopy.
Group L - lignocaine group: lignocaine 1.5 mg/kg administered intravenously 2 minutes prior to laryngoscopy.

Anesthetic technique
Standard intra-operative monitoring was instituted for all patients (electrocardiography, non-invasive blood pressure, and pulse oximetry). Baseline HR, SBP, DBP and MAP were noted. Patients were then anaesthetized using standard doses of intravenous induction agents and muscle relaxant in order to facilitate intubation. Laryngoscopy was performed by an experienced anaesthesiologist with a standard Macintosh blade. Laryngoscopy was less than 20s in all patients.

Hemodynamic measurements
Hemodynamic parameters (HR, SBP, DBP, MAP) were recorded at the following time intervals:

- T0: Baseline (before study drug administration)
- T1: After study drug administration (before induction)
- T2: Immediately after intubation
- T3: 1 minute after intubation
- T4: 3 minutes after intubation
- T5: 5 minutes after intubation
- T6: 10 minutes after intubation

Outcome measures
Primary outcome:

- Attenuation of heart rate and mean arterial pressure following intubation.

Secondary outcomes:

- Changes in systolic and diastolic blood pressure.
- Incidence of tachycardia (HR > 100 bpm).
- Incidence of hypertension (increase in MAP > 20% from baseline).
- Occurrence of bradycardia (HR < 60 bpm).
- Hypotension (MAP decrease > 20% from baseline).
- Any arrhythmias or adverse effects requiring intervention.

Statistical analysis
Continuous variables were expressed as mean ± SD. Categorical variables were expressed as number and percentage. The variables between the two groups were compared using scale variables. Continuous variables were compared using appropriate tests and a p value of <0.05 was considered as significant.

RESULTS
A hundred patients were included and completed the study out of the hundred patients. The two groups were comparable related to age, sex allocation, body weight, and ASA physical status. The hemodynamic profile including baseline heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure were similar statistically between both groups before hydrating with study drugs. Post-hydration with either study drugs there is a statistically significant fall of heart rate seen with esmolol over lignocaine. Immediately after intubation heart rate and blood pressure rises, although lesser in the esmolol group. The peak response was noticed at 1 minute post intubation in both groups. The hemodynamic parameters returned to baseline values by about 5-10 minutes in both the groups but was quicker with esmolol. The anyway incidence of tachycardia and hypertension was noted to be more in the lignocaine group. The bradycardia was slightly more with esmolol, though not clinically significant. Overall esmolol is better than lignocaine in ameliorating pressor response to laryngoscopy and intubation.

Table 1: Demographic profile of patients

Table 1 shows that both groups were comparable with respect to age, sex, and body weight.

Parameter	Group E (n = 50)	Group L (n = 50)
Age (years, mean ± SD)	39.8 ± 10.6	40.5 ± 11.1
Male/Female	28 / 22	30 / 20
Weight (kg, mean ± SD)	66.2 ± 8.9	65.7 ± 9.3

Table 2: ASA physical status distribution

Table 2 shows similar ASA distribution in both groups.

ASA status	Group E (n = 50)	Group L (n = 50)

ASA I	32 (64%)	34 (68%)
ASA II	18 (36%)	16 (32%)

Table 3: Baseline hemodynamic parameters (T0)

Table 3 shows comparable baseline hemodynamic values between groups.

Parameter	Group E (mean ± SD)	Group L (mean ± SD)
Heart Rate (bpm)	82 ± 9	84 ± 10
SBP (mmHg)	124 ± 12	126 ± 11
DBP (mmHg)	78 ± 8	80 ± 7
MAP (mmHg)	93 ± 9	95 ± 8

Table 4: Heart rate changes at different time intervals

Table 4 shows heart rate response following intubation in both groups.

Time Interval	Group E (mean ± SD)	Group L (mean ± SD)
T1	78 ± 8	82 ± 9
T2	88 ± 10	102 ± 12
T3	92 ± 9	106 ± 11
T4	86 ± 8	98 ± 10
T5	82 ± 7	90 ± 9
T6	80 ± 6	86 ± 8

Table 5: Systolic blood pressure changes

Table 5 shows systolic blood pressure variations at different intervals.

Time Interval	Group E (mean ± SD)	Group L (mean ± SD)
T1	120 ± 10	124 ± 11
T2	134 ± 12	152 ± 14
T3	138 ± 13	156 ± 15
T4	128 ± 11	144 ± 13
T5	122 ± 10	132 ± 11
T6	118 ± 9	126 ± 10

Table 6: Diastolic blood pressure changes

Table 6 shows diastolic blood pressure trends in both groups.

Time Interval	Group E (mean ± SD)	Group L (mean ± SD)
T1	76 ± 7	78 ± 8
T2	86 ± 9	96 ± 10
T3	88 ± 8	98 ± 9
T4	82 ± 7	90 ± 8
T5	78 ± 6	84 ± 7
T6	76 ± 5	82 ± 6

Table 7: Mean arterial pressure changes

Table 7 shows MAP changes across time intervals.

Time Interval	Group E (mean ± SD)	Group L (mean ± SD)
T1	90 ± 8	94 ± 9
T2	102 ± 10	115 ± 12
T3	105 ± 9	118 ± 11
T4	98 ± 8	108 ± 10
T5	94 ± 7	100 ± 8
T6	92 ± 6	96 ± 7

Table 8: Incidence of tachycardia (>100 bpm)

Table 8 shows higher tachycardia incidence in Group L.

Parameter	Group E (n = 50)	Group L (n = 50)
Tachycardia	6 (12%)	18 (36%)

Table 9: Incidence of hypertension (>20% rise in MAP)

Table 9 shows greater hypertension incidence in Group L.

Parameter	Group E (n = 50)	Group L (n = 50)
Hypertension	8 (16%)	20 (40%)

Table 10: Adverse effects

Table 10 shows adverse events in both groups.

Adverse effect	Group E (n = 50)	Group L (n = 50)
Bradycardia	5 (10%)	1 (2%)
Hypotension	3 (6%)	2 (4%)
Arrhythmias	0 (0%)	1 (2%)

Table 1 shows that the mean age in Group E was 39.8 ± 10.6 years compared to 40.5 ± 11.1 years in Group L. Male patients constituted 56% in Group E and 60% in Group L, while females accounted for 44% and 40% respectively. The mean body weight was 66.2 ± 8.9 kg in Group E and 65.7 ± 9.3 kg in Group L. These findings confirm demographic homogeneity between the groups, minimizing baseline confounding factors. **Table 2** demonstrates that ASA I patients comprised 64% of Group E and 68% of Group L, whereas ASA II patients constituted 36% and 32% respectively. This comparable distribution of preoperative physical status indicates that both groups had similar baseline anesthetic risk profiles. **Table 3** shows that baseline heart rate was 82 ± 9 bpm in Group E and 84 ± 10 bpm in Group L. Baseline systolic blood pressure was 124 ± 12 mmHg versus 126 ± 11 mmHg, diastolic blood pressure was 78 ± 8 mmHg versus 80 ± 7 mmHg, and mean arterial pressure was 93 ± 9 mmHg versus 95 ± 8 mmHg. These comparable baseline values confirm that both groups started under similar hemodynamic conditions prior to drug administration. **Table 4** indicates that heart rate increased in both groups after intubation; however, the peak heart rate at 1 minute (T3) was 92 ± 9 bpm in Group E compared to 106 ± 11 bpm in Group L. This represents an approximate rise of 12% from baseline in Group E versus nearly 26% in Group L. Tachycardia (>100 bpm) occurred less frequently in the esmolol group, reflecting superior attenuation of the chronotropic response. **Table 5** shows that peak systolic blood pressure at 1 minute post-intubation reached 138 ± 13 mmHg in Group E compared to 156 ± 15 mmHg in Group L. This corresponds to an approximate 11% increase from baseline in Group E versus a 24% increase in Group L, demonstrating more effective control of the systolic pressor response with esmolol. **Table 6** demonstrates that peak diastolic blood pressure increased to 88 ± 8 mmHg in Group E and 98 ± 9 mmHg in Group L. The percentage rise from baseline was lower in Group E, indicating better modulation of peripheral vascular resistance with β1-adrenergic blockade. **Table 7** confirms that mean arterial pressure peaked at 105 ± 9 mmHg in Group E compared to 118 ± 11 mmHg in Group L. The relative increase in MAP was significantly higher in the lignocaine group, reinforcing the superior efficacy of esmolol in suppressing sympathetic stimulation following intubation. **Table 8** shows that tachycardia (>100 bpm) occurred in 6 patients (12%) in Group E compared to 18 patients (36%) in Group L. This threefold higher incidence in the lignocaine group highlights the stronger chronotropic control achieved with esmolol. **Table 9** indicates that hypertension (MAP rise >20% from baseline) occurred in 8 patients (16%) in Group E compared to 20 patients (40%) in Group L. The markedly higher incidence in Group L demonstrates less effective attenuation of the pressor response with lignocaine. **Table 10** shows that bradycardia occurred in 5 patients (10%) in Group E compared to 1 patient (2%) in Group L, reflecting the expected pharmacological effect of β1-blockade. Hypotension was observed in 3 patients (6%) in Group E and 2 patients (4%) in Group L, while arrhythmias were rare in both groups. Overall, adverse effects were minimal and clinically manageable in both groups.

DISCUSSION

The present prospective randomized comparative study done over a period of 12 months in 100 patients proves the definite efficacy of intravenous esmolol and lignocaine in attenuating hemodynamic response to laryngoscopy and intubation and compares the efficacy of both. Esmolol was more effective than lignocaine in attenuation of heart rate and arterial pressure responses during the immediate post intubation period [9,10]. Laryngoscopy and intubation cause a powerful but transient sympathetic discharge with tachycardia and hypertension mediated by catecholamines and resulting from stimulation of laryngeal and tracheal structures. In the present study, heart rate and BP increased in the immediate period after intubation and peak values were seen at 1 minute post intubation in both groups [11]. However, the magnitude of the increase was lower in the esmolol group. In esmolol group, other than the attenuated chronotropic response (<20% increase in HR as compared to lignocaine) in the immediate post intubation period, it was seen that the incidence of >100/min heart rate or tachycardia was 3 times higher in the lignocaine group [12,13]. The β1-adrenergic blockade suppressed the sympathetic cardiac stimulation better. This is feasible pharmacologically since esmolol blocks β1-receptors in the myocardium thereby attenuating heart rate and myocardial contractility and reducing the hemodynamic effect of catecholamine surge during laryngoscopy and intubation.Similarly, systolic blood pressure, diastolic blood pressure and mean arterial pressure increased in both groups following intubation; however, the rise was significantly higher in the lignocaine group. The incidence of hypertension (MAP rise >20% from baseline) was significantly higher in patients receiving lignocaine [14]. The better control of bloodpressure exerted by esmolol is due to its property of blunting sympathetic cardiac output response which subsequently dampens pressor effect of airway manipulation. Lignocaine is considered to attenuate airway reflexes and minimizes sympathetic response; however, the mechanism of action is primarily suppression of neuronal transmission and a partial nuisance value for sympathetic outflow. Though this depresses the rise moderately, it does not block β adrenergic receptors. This could explain why lignocaine was less effective in blunting peak heart rate and blood pressure response as compared to esmolol in our study. Interestingly, hemodynamic values in esmolol group returned

towards baseline rapidly in contrast to lignocaine group [15]. This could be because esmolol not only reduces peak response but also shortens duration of sympathetic overactivity. Being short acting, with rapid elimination, esmolol provides a precise and transitory control of hemodynamics and does not leave patients with uncontrolled hypotension or bradycardia for prolonged durations. Safety: bradycardia was noted to be marginally more common in esmolol group; as expected due to its pharmacodynamic effect of β1 blockade. However, the episodes were mild and could be sustained with no major clinical relevance. Hypotension and arrhythmias were observed infrequently in both groups [16]. None of the patients has serious adverse events during the study period; hence, both the drugs are safe in used studied doses. Clinically, these observations become more important in patients where exaggerated hemodynamic responses maybe detrimental [17]. Patients suffering from ischaemic heart disease, uncontrolled hypertension or cerebrovascular pathology could suffer harm if the drawbacks of uneventful intubation with associated exaggerated hemodynamic response is repeated in multiple cases. Although our study population consists of ASA I and II patients, presence of superior attenuation by esmolol in comparison to lignocaine makes the former more appealing for high-risk populations as well, provided precaution is taken with monitoring for complications [18].

The study was conducted at single centre with 100 patients, which would have comprised adequate sample size to demonstrate a significant difference between both agents but may exclude the generalisation. High risk cardiac were not included in study and the results cannot be directly applied our findings to patients with severe cardiovascular disease. Dyas including higher risk populations and larger sample sizes would provide more substantiate evidence [19]. Intravenous esmolol is more effective than intravenous lignocaine in attenuating the hemodynamic response to laryngoscopy and endotracheal intubation, specially in controlling heart rate and mean arterial pressure during early post intubation period [20].

CONCLUSION

In this prospective randomized comparative study, carried out over 12 months on 100 patients undergoing elective surgery under general anaesthesia, intravenous emolol is clinically better than intravenous lignocaine in attenuation of the hemodynamic responses to laryngoscopy and endotracheal intubation. Esmolol more effectively attenuated heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure at intubation and especially at the first minute after intubation when sympathetic stimulation is at a peak. Fewer patients in the esmolol group experienced tachycardia and hypertension. Bradycardia occurred slightly more in patients given esmolol but it was not of clinical importance and was without any serious complications. The commonest arrythmias are also clinically insignificant. Both the drugs are safe and well tolerated and both effectively attenuated the pressor response but the esmolol attenuation response is more reliable. It appears that this drug can be used more effectively than lignocaine for suppressing the transient sympathetic responses which are associated with direct laryngoscopy and endotracheal intubation.

Ethical Approval

Approval for conducting the study was obtained from the Institutional Ethics Committee. All methods were carried out in accordance with the relevant guidelines for work with human participants.

Informed Consent

Informed written consent from patients was obtained for study inclusion.

Data Availability

Access to the datasets generated and analysed is available when requested from the author of correspondence.

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No funding was used in writing this paper.

Conflict of Interest

The authors declare that they have no conflict of interest.

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