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Original Article

Comparison of Esmolol and Lidocaine for Blunting of Stress Response during Laryngoscopy and Endotracheal Intubation

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Abstract

Background: Direct laryngoscopy and endotracheal intubation lead to stimulation of strong cardiovascular responses. Various attempts have been made to attenuate these responses. The aim of this study was to compare the efficacy and safety of intravenous esmolol and lidocaine in suppressing the cardiovascular response to laryngoscopy and tracheal intubation in normotensive patients undergoing general anesthesia.

Materials and Methods: This randomized controlled study was conducted in 90 normotensive patients of age group 18–60 years and American Society of Anaesthesiologists (ASA) physical Status I or II undergoing elective surgeries. The patients were randomly divided into three groups of 30 patients each (n = 30) - control, (C) lignocaine (L), and esmolol (E). Group - "C" received 10 ml normal saline, Group -"L" received 2 mg/kg diluted up to 10 ml preservative-free lidocaine, and Group -"E" received 2 mg/kg esmolol IV diluted up to 10 ml, 2 min before intubation. Induction was done with thiopentone 5 mg/kg, fentanyl 2 μg/kg, and vecuronium 0.1 mg/kg uniformly as per protocol. Thereafter, changes in heart rate (HR), systolic blood pressure (SBP), diastolic BP (DBP), and mean arterial BP (MAP), were measured before induction of general anesthesia (baseline), 1, 3, and 5 min after tracheal intubation. Patients were also observed for any complications. Statistical analysis was performed by ANOVA and *post hoc* tukey test.

Results: Group C had statistically highly significant ($P \leq 0.0001$) value of HR, SBP, DBP, and MAP at all time interval after intubation when compared to Group L and Group E, and, Group L had statistically significant ($P \leq 0.05$) higher values of hemodynamic variables at all time interval when compared to Group E.

Conclusions: Both esmolol and lignocaine are effective in attenuating the stress response due to laryngoscopy and intubation, but esmolol maintains hemodynamic variables more stable.

Key words: Esmolol, Intubation, Laryngoscopy, Lidocaine, Stress response

INTRODUCTION

Hypertension and tachycardia usually accompany laryngoscopy and tracheal intubation; it is generally transient occurring 30 s after intubation and lasting for <10 min due to reflex sympathetic stimulation.[1,2] This stress response should be avoided, especially in patients with cardiovascular or intracranial diseases. Various pharmacological agents have been used to attenuate the pressure responses.[3] Topical and intravenous lidocaine, opioids, inhaled anesthetics, calcium channel blocker, vasodilators or beta/adrenergic blockers, magnesium sulfate, pregabalin, etc., have been tried to blunt these hemodynamic responses. Lidocaine has been found to be inconsistently effective.[4-6] Esmolol (beta-adrenergic receptor antagonist + ultra-short-acting) provides hemodynamic stability during laryngoscopy and tracheal intubation without side effects.[7]

Previous studies have shown that the control of the cardiovascular response to endotracheal intubation is important to reduce adverse cardiovascular outcomes.[8]

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The main purpose of this study was to determine the efficacy and safety of intravenous lidocaine and esmolol in attenuating hemodynamic response to laryngoscopy and intubation in normotensive patients undergoing elective general surgeries under general anesthesia requiring endotracheal intubation.

MATERIALS AND METHODS

After an institutional approval by the Ethics Committee of the institution, the study was started from December 2016, to April 2017. Informed consent was obtained. Proper preanesthetic check-up and routine investigations were done. 90 consenting adult patients aged 18–65 years of age of either sex being non-hypertensive, American Society of Anaesthesiologists Grade I or II undergoing elective surgery other than cardiac surgery under general anesthesia with endotracheal intubation, oropharyngeal anatomy of Mallampati Class I and II, were included in our study protocol. Whereas, patients who were morbidly obese, pregnant and lactating females, patients with cardiopulmonary and renal disease, heart rate (HR) <60 beats per minute, basal systolic blood pressure (SBP) <100 mm/Hg, and other conditions such as bronchial asthma, diabetes mellitus, drug allergies, anticipated difficult intubation, and in cases where duration of laryngoscopy exceeded 15 s were excluded. Allocation concealment was ensured with sealed opaque envelopes. Our study was comparative, prospective, randomized, and double-blinded in a normotensive healthy population. The sample size taken was 90 generated using a sample size calculator. The study groups were randomly divided into three groups (C- control/L- lignocaine/E-esmolol) by a computer-generated randomization table. Dose used: Esmolol 2 mg/kg BW and lignocaine 2 mg/kg BW. A anesthetist colleague (Person A) who was not involved in the randomization process was made to prepare the study drugs by diluting to 10 ml. All three drugs were color-coded to improve blinding. Another Person B monitored the HR, SBP, and diastolic BP (SBP/DBP) mean arterial pressure (MAP) at various time intervals while Person C was responsible for intubation of the patients having a good experience of the technique. Person A and C were kept constant throughout the study. Person B, C, and the patient were kept unaware of the drug injected to enable double-blinding.

After patient confirmation, short pre-operative history taking and clinical examination were done. Procedure explained to the participants, and a written informed consent was obtained from each participant. Baseline vital parameters of patients' including HR, systolic arterial pressure (SAP), diastolic arterial pressure; MAP, and oxygen saturation were recorded in the operation theater through routine standard monitors. In the operating room, IV line was secured with 18-G venous cannula, and Ringer's lactate infusion (8 ml/kg) was started. All the patients were uniformly pre-medicated with IV midazolam 0.05 mg/kg and glycopyrrolate 0.004 mg/kg, and 10 min before induction. The study drugs were prepared to a volume of 10 ml.

Patients were pre-oxygenated with four to five breaths of 100% oxygen. Induction done with 5 mg/kg IV thiopentone sodium in incremental doses until loss of eyelash reflex, fentanyl 2 μg/kg and 0.1 mg/kg IV vecuronium bromide given slowly, followed by administration of the study drugs (normal saline, esmolol, or lignocaine) 2 min before laryngoscopy and intubation.

Patients were ventilated with oxygen and 2% sevoflurane using IPPV with a fresh gas flow of 8 l/min by Bains circuit until intubation. About 2 min after study drug being given, laryngoscopy was performed with a Macintosh laryngoscope blade and trachea intubated by a trained anesthetist with an appropriate size cuffed endotracheal tube. After confirmation of correct placement of ET tube, anesthesia was then maintained with O_2/N_2O/Sevo (50:50:1%). Additional doses of vecuronium bromide and fentanyl 1 μg/kg if necessary were administered to maintain surgical relaxation and analgesia. Surgery was allowed to start only after 10 min of intubation. At the end of surgery neuromuscular blockade reversal was done with injection neostigmine 0.05 mg/kg and injection glycopyrrolate 0.1 mg/kg. IV ondansetron 0.08 mg/kg was given to patients 30 min before the completion of the surgery. The tracheal tube was removed after the adequate spontaneous ventilation established.

HR, SBP, DBP, MAP, SpO_2 (oxygen saturation), and electrocardiogram changes were recorded before induction (Basal) and after tracheal intubation at 1, 3, and 5 min for the study.

Parameters and Statistical Analysis

Summary statistics of patient gender, age, weight, SpO_2, for all three groups were reported as means ± standard deviation. HR, SBP, DBP, and MAP were recorded, before induction (baseline), after tracheal intubation at 1, 3, and 5 min. Patients were also observed for complications such as hypotension, hypertension, arrhythmias, and hypoxemia. For statistical analysis, hemodynamic variables were represented by mean ± standard deviation (SD). Results on continuous measurements are presented as mean ± SD. Significance was assessed at 5% level of significance. Analysis of variance was used to assess the significance of study parameters between three or more groups of patients. *Post hoc* Tukey test was used to find the pairwise significance.

$P < 0.05$ and $P < 0.001$ were considered significant and highly significant, respectively, for the study.

RESULTS

All the three groups were comparable in demographic data [Table 1]. The basal readings of HR, SBP, DBP, and MAP were similar in all three groups. Maximum laryngoscopy response was recorded at 1 min all three groups. The vital parameters never reached near baseline by 5 min in Group C. In Group E, hemodynamic variables reached near baseline by 3 min, and at 5 min they fell below the baseline. In Group L, all vital parameters reached near baseline by 5 min. Group C had statistically highly significant ($P \leq 0.0001$) higher value of HR, SBP, DBP, and MAP at all time interval after intubation when compared to Group L and Group E, and Group L had statistically significant ($P \leq 0.05$) higher values of hemodynamic variables at all time interval when compared to Group E. Therefore, it can be inferred that the lignocaine and esmolol both are effective in attenuating intubation response, but esmolol seems to be more effective than lignocaine in attenuating laryngoscopic and intubation response [Tables 2-5].

DISCUSSION

King *et al.* first described the hemodynamic stress response due to laryngoscopy and intubation more than 60 years ago.[9] Orotracheal intubation consists of two phases: Direct laryngoscopy and passing of endotracheal tube through the vocal cords and trachea.[10] It has been seen in various studies that increase in HR occurs during endotracheal intubation whereas the greatest increase in BP occurs during laryngoscopy.[11] 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.[12]

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.[13] All other organs but most important to this discussion, the brain, heart, and kidneys depend on systemic pressure to maintain perfusion pressure. Therefore, it is inferred that certain patients such as with coronary artery disease, hypertension, raised intracranial, or intraocular pressure cannot tolerate the consequences of the hemodynamic response to laryngoscopy and intubation.[8]

Among the available β-adrenergic blocking drugs, 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

Table 1: Distribution of patient's demographic profile

Parameter	Group C (*n*=30)	Group L (*n*=30)	Group E (*n*=30)	*P* value
Age (in years)	42.45±11.55	41.25±13.45	43.70±14.17	0.770 (ANOVA test)
Weight (in kg)	65.12±8.84	62.88±9.30	66.10±9.20	0.378 (ANOVA test)
Ratio (M: F)	16:14	18:12	16:14	0.895 (Fischer exact test)
ASA status (I/II)	8.22	12.18	14.16	-
MP grade (I/II)	20.10	20.10	17.13	-
Baseline SpO_2	99.47±0.48	99.54±0.37	99.63±0.55	0.425 (ANOVA test)

Table 2: Changes in hemodynamic variables (HR) in control and experimental groups

HR (beats/min)	Group C	Group L	Group E	*P* value	Pairwise significance		
					C versus L	C versus E	L versus E
Baseline	81.40±5.67	79.50±5.30	80.47±5.28	0.4016	0.3675	0.7846	0.7681
1	113.23±5.80	90.57±5.41	85.37±5.51	<0.0001*	<0.0001*	<0.0001*	0.0015
3	105.03±4.63	84.73±4.93	80.83±5.40	<0.0001*	<0.0001*	<0.0001*	0.0091
5	93.87±5.08	80.47±4.65	75.03±5.80	<0.0001*	<0.0001*	<0.0001*	0.0003

*Highly significant (test of significance used is ANOVA and *post hoc* Tukey test), HR: Heart rate

Table 3: Changes in hemodynamic variables (SBP) in control and experimental groups

SBP (mm of Hg)	Group C	Group L	Group E	*P* value	Pairwise significance		
					C versus L	C versus E	L versus E
Baseline	127.07±7.80	129.73±8.82	128.07±8.11	0.4549	0.4284	0.8859	0.7169
1	161.13±6.08	137.60±8.74	132.27±7.75	<0.0001*	<0.0001*	<0.0001*	0.0216
3	145.33±5.87	132.87±8.75	127.67±8.51	<0.0001*	<0.0001*	<0.0001*	0.0311
5	139.60±4.94	128.07±8.87	121.80±7.59	<0.0001*	<0.0001*	<0.0001*	0.0038

*Highly significant (test of significance used is ANOVA and *post hoc* Tukey test). SBP: Systolic blood pressure

Table 4: Changes in hemodynamic variables (DBP) in control and experimental groups

DBP (mm of Hg)	Group C	Group L	Group E	*P* value	C versus L	C versus E	L versus E
Baseline	77.40±6.84	78.37±4.91	75.87±5.26	0.2400	0.7898	0.5577	0.2151
1	92.32±6.11	84.67±5.12	80.33±5.26	<0.0001*	<0.0001*	<0.0001*	0.0085
3	89.13±5.64	82.67±5.22	76.47±5.16	<0.0001*	<0.0001*	<0.0001*	0.0001
5	85.67±5.33	77.33±5.02	72.53±4.66	<0.0001*	<0.0001*	<0.0001*	0.0011

DBP: Diastolic blood pressure. *Highly significant (test of significance used is ANOVA and *post hoc* Tukey test)

Table 5: Changes in hemodynamic variables (MAP) in control and experimental groups

MAP	Group C	Group L	Group E	*P* value	Pairwise significance		
					C versus L	C versus E	L versus E
Baseline	92.73±6.36	95.70±5.25	94.27±5.73	0.1458	0.1222	0.5609	0.6070
1	115.57±5.14	101.87±5.76	97.33±5.40	<0.0001*	<0.0001*	<0.0001*	0.0049
3	109.47±5.97	98.83±5.53	94.27±5.69	<0.0001*	<0.0001*	<0.0001*	0.0077
5	104.37±5.51	93.80±5.58	89.47±5.18	<0.0001*	<0.0001*	<0.0001*	0.0075

MAP: Mean arterial pressure. *Highly significant (test of significance used is ANOVA and *post hoc* Tukey test)

with no significant drug interaction with commonly used anesthetics.[14,15] 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. There have been several reports showing the effects of esmolol on both HR and arterial BP during laryngoscopy and ET intubation compared with placebo. Miller *et al.*[16] in their study have reported that 100 mg of a single bolus dose of esmolol was effective for controlling the hemodynamic response to tracheal intubation in a Canadian multicenter trial. Liu *et al.* who used esmolol infusion to control hemodynamic responses associated with intubation, found significant decreases in HR and SAP before induction and post-intubation, the increase was 50% less in the esmolol-treated patients compared to the placebo group.[17] Korpinen *et al.* reported that the administration of bolus esmolol 2 mg/kg IV 2 min before laryngoscopy and intubation suppressed the increase in the HR rather than arterial BP.[18] Bostana and Eroglu reported that IV esmolol in dose of 1 mg/kg before intubation was effective in suppressing the HR and arterial BP.[19] Kumar *et al.* have also claimed optimal results while using higher doses of esmolol in Asian population, i.e., 2 mg/kg without any incidence of unplanned hypotension or bradycardia.[20]

Studies disagreeing to esmolol's response on both tachycardia and hypertensive response following ET intubation are also available, namely, Oxorn *et al.* in their study concluded that esmolol in bolus doses of 100 mg and 200 mg affected solely the chronotropic response, i.e., it reduced the HR only significantly.[21] Kindler *et al.* found that esmolol administration before laryngoscopy was sufficient to control HR after intubation, but it did not affect SAP.[22] In our study, esmolol 2 mg/kg was found to be quite effective in attenuating the hypertensive response (MAP) as well as the HR during laryngoscopy and tracheal intubation till 5 min.

Lignocaine has been a popularly used agent for attenuating circulatory responses during intubation. The beneficial effect of lidocaine is due to its direct cardiac depression and peripheral vasodilation, ability to suppress airway reflexes due to irritation of tracheal mucosa, analgesic as well as antiarrhythmic properties. Abou-Madi *et al.* compared the efficacy of intravenous lidocaine 0.75 mg/kg and 1.5 mg/kg as protection against cardiovascular responses associated with laryngoscopy and endotracheal intubation.[23] These

researchers found that 1.5 mg/kg of lidocaine afforded complete protection against cardiac arrhythmias of all types; the lower dose was ineffective in preventing ventricular arrhythmias. The higher dose also offered "borderline" protection against a rise in HR and BP. The lower dose only prevented a rise in SBP. Lev and Rosen reviewed the use of prophylactic lignocaine as a pre-intubation medication.[24] They used a dose of 1.5 mg/kg intravenously 3 min before intubation, and it was found to be optimal for attenuation of the sympathoadrenal pressure response to laryngoscopy and intubation without any harmful effects. Wilson *et al*. in their study stated that IV lignocaine is beneficial in preventing the hemodynamic changes to laryngoscopy and intubation.[25] Bruder *et al*. in a review article wrote that in clinical practice, lignocaine is particularly effective in preventing the pressor response to tracheal intubation, whatever its route of administration (intravenous or intratracheal), but not the increase in HR.[26] From our statistical analysis, we infer that there is a general decline in HR after administration of lignocaine at the time interval corresponding to 1 (maximum), 3 and 5 min post-intubation, which is in contrast to previous studies owing to the dose of lignocaine being 2 mg/kg in our case suggesting complete abolished reflex at this dose till 5 min.

However, recent studies have doubted the lignocaine's efficacy. In studies by Singh *et al*.[27] van den Berg *et al*.,[28] and Kindler *et al*.[23] IV lignocaine 1.5 mg/kg was not found to be effective in controlling the acute hemodynamic response following laryngoscopy and intubation. Hence, we thought of modifying the dose to 2 mg/kg and found better results comparatively with no serious side effects. From the interpretation of the results of our study, we concluded that lignocaine (2 mg/kg) blunted the pressure response to laryngoscopy and intubation for a longer duration compared to previous studies with dose 1.5 mg/kg.

CONCLUSION

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. However, esmolol 2 mg/kg appears to be more effective and a potential agent for attenuating hemodynamic changes during induction of anesthesia.

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