

Comparative Study of Haemodynamic Responses to Laryngoscopy and Oral Endotracheal Intubation in Healthy Normotensive Adults with Prior Administration of Lignocaine and Esmolol Hydrochloride

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ABSTRACT

BACKGROUND

The frequent occurrence of cardiovascular responses to laryngoscopy and tracheal intubation has attracted the attention of anaesthesiologists for more than five decades. The reason for this is the occasional report of sudden death immediately after intubation and increasing awareness about the common occurrence of potentially dangerous responses such as tachycardia, hypertension and arrhythmias. We wanted to study, measure and compare the haemodynamic responses to laryngoscopy and oral endotracheal intubation in healthy normotensive adults with prior administration of injection lignocaine and injection esmolol hydrochloride.

METHODS

75 patients belonging to ASA 1, ASA 2 status, in the age group of 18-60 years scheduled for elective non-cardiac surgical procedures under general anaesthesia were selected for this prospective randomized controlled study conducted after obtaining institutional approval. They were randomly divided into 3 groups of 50 each. Group C (control group), Group Lignocaine and Esmolol Group. All the patients irrespective of group to which they belonged received tablet diazepam 0.15 mg/Kg the previous night followed by intramuscular Pethidine 1 mg/Kg 1 hour prior to the scheduled surgery.

RESULTS

In esmolol group, there has significant attenuation of heart rate, SAP, DAP and mean arterial pressure following laryngoscopy and endotracheal intubation compared to lignocaine group. Lignocaine group has significant attenuation of heart rate, SAP, DAP and mean arterial pressure following laryngoscopy and endotracheal intubation compared to control group.

CONCLUSIONS

Esmolol hydrochloride given in the dose of 1.5 mg/Kg body weight 3 minutes prior to intubation provided consistent and reliable protection against increase in mean heart rate. SAP, DAP and MAP during laryngoscopy and intubation compared to lignocaine (1.5 mg/Kg).

KEY WORDS

Esmolol, Lignocaine, Sympathoadrenal Stimulation, Laryngoscopy and Endotracheal Intubation

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BACKGROUND

There have been occasional reports of sudden death immediately after intubation and increasing awareness about the common occurrence of potentially dangerous responses such as tachycardia, hypertension and arrhythmias after endotracheal intubation. They elicit adrenergic responses that precipitate transient but intense increase in heart rate, BP and serum concentrations of catecholamine. The circulatory responses to laryngeal and tracheal stimulation were known since 1940. These facts are interpreted as being the result of reflex sympathoadrenal stimulation. Although this response is well tolerated in healthy patients, in susceptible individuals this response results in increased cardiac workload; which in turn may culminate in preoperative myocardial ischemia, infarction, rhythm disturbances, acute heart failure, pulmonary oedema and ventricular arrhythmias owing to sudden increase in myocardial oxygen demand. This response is undesirable, especially in patients with hypertension, cardiovascular diseases, cerebrovascular diseases, PIH, hyperthyroidism etc.

Antihypertensives modify the response but do not inhibit it completely. Attempt to reduce these untoward cardiovascular response during laryngoscopy and endotracheal intubation lead to the trial of various systemic as well as topical agents.¹ Various methods used to modify this reflex are deeper planes of anaesthesia, narcotics like fentanyl and alfentanil, intravenous Lignocaine, topical Lignocaine, vasodilators such as sodium nitroprusside, isosorbide dinitrate, alpha blockers, premedication with clonidine, induction with propofol, use of calcium channel blockers like nifedipine, verapamil, diltiazem, use of beta blockers like propranolol, labetalol, Esmolol etc.^{1,2} In our study, we compared I.V Lignocaine and IV Esmolol to find out how effective these drugs are in suppressing haemodynamic responses to laryngoscopy and intubation. I.V Lignocaine is commonly used in our hospital. We selected Esmolol as its ultra-short action seems ideal to control the intense but brief sympathetic stimuli following laryngoscopy and endotracheal intubation.³

We wanted to study, measure and compare the haemodynamic responses to laryngoscopy and oral endotracheal intubation in healthy normotensive adults with prior administration of injection Lignocaine and injection Esmolol hydrochloride.

METHODS

This was a randomized controlled study conducted after obtaining institutional approval. One hundred and fifty patients belonging to ASA Grade I or II participated in the study. They were scheduled for elective non-cardiac surgical procedures under general anaesthesia.⁴

Preoperative Evaluation

On the evening before surgery, all the patients underwent a thorough systemic and physical examination including evaluation of the airway. Patient's age and weight was noted.

Exclusion Criteria

1. Patients in whom difficulty in tracheal intubation was anticipated.
2. Patients with conduction blocks or congestive heart failure.
3. Patients with diabetes mellitus.
4. Arrhythmias.
5. Bronchial asthma.
6. Use of B-blockers within the twenty-four hours preceding surgery.
7. Those in whom attempts at intubation failed in the first attempt and in whom laryngoscopy exceeded 30 seconds.

Patients were divided randomly into three groups of 50 each-

Control Group:

A Who did not receive any drug.

Lignocaine Group:

B Received 1.5 mg/Kg of Lignocaine Intravenously.

Esmolol Group:

C Received 1.5 mg/Kg of Esmolol Hcl intravenously.

All the patients irrespective of group to which they belonged received tablet diazepam 0.15 mg/Kg the previous night followed by Inj. Midazolam 1 mg given IV before to reduce the anxiety, Fentanyl given IV before starting of surgery as an Intraoperative analgesic at the dose of 2µg/Kg. After transferring the patient into the operating room, the patient was placed supine with a small pillow under the head. The NIBP cuff was attached to one of the arms of the patient, pulse oximetry probe was attached to the finger of the other arm of the patient.⁵ Basal values of pulse rate, systolic arterial pressure (SAP), Diastolic arterial pressure (DAP) and MAP were noted. Then an intravenous line using an appropriately sized cannula was started on one of the forearm veins and a Ringer lactate run slowly.

Induction Technique

The induction technique included preoxygenation for 5 minutes with a face mask. 1.5 mg/Kg of Esmolol in Esmolol group, 1.5 mg/Kg of Lignocaine^{6, 7} in Lignocaine group was given slowly over a period of one minute and control group did not receive any medication. After one-minute induction of anaesthesia was done with 5 mg/Kg of 2.5%. Thiopentone sodium administered slowly over a period of 30 seconds to minimize its effect on cardiovascular system. Following the loss of eyelash reflex ventilation was checked and then suxamethonium 2 mg/Kg was administered. The patient was then ventilated using 100% oxygen with positive pressure ventilation. After 90 seconds laryngoscopy was done with a Macintosh curved blade and tracheal intubation completed within 15 seconds using an appropriate size cuffed endotracheal tube and the cuff inflated.

Maintenance of Anaesthesia

The endotracheal tube was connected to a closed circle absorption system and IPPV commenced. Anaesthesia was then maintained with 66% Nitrous Oxide and 33% Oxygen. No muscle relaxant was given in the first 5 minutes following intubation. No other anaesthetic agent or drug was

administered during the said period. No surgical stimulation was permitted during this period. Pulse rate, systolic arterial pressure, diastolic arterial pressure was recorded. The parameters were recorded at the start of Oxygenation, and also at the end of 1, 3, 5 minutes following tracheal intubation. At the end of the study period, any changes in anaesthetic technique was made according to the choice of the anaesthesiologist responsible for the further conduct of the anaesthesia.

Statistical Analysis

Analysis of variance (ANOVA) was done to assess the extent of variation between the three groups. Student’s ‘t’ test was used to find the significance of difference between the control, Lignocaine, and esmolol.

RESULTS

Details	Control	Lignocaine	Esmolol	F-Value	p Value	Significance
Pulse Rate	86.36	84.24	86.44	3.548	0.00313	NS
SAP	124.8	122.56	123.92	2.185	0.1161	NS
DAP	83.28	83.44	84	0.444	0.4681	NS
MAP	98.72	97.84	98.12	0.763	0.4681	NS

Table 1. Mean Haemodynamic Values at Baseline

Details	Control	Lignocaine	Esmolol	F-Value	p Value	Significance
Pulse Rate	104.36	96.4	83.6	261.138	0.0001	S
SAP	157.2	143.2	121.04	277.472	0.0001	S
DAP	103.84	102.48	82.24	266.488	0.0001	S
MAP	120.04	116.04	95.08	419.678	0.0001	S

Table 2. Mean Hemodynamic Values at 1 Minute after Intubation

Details	Control	Lignocaine	Esmolol	F-value	p Value	Significance
Pulse Rate	97.12	94.96	79.44	275.737	0.0001	S
SAP	151.2	135.4	117.52	398.856	0.0001	S
DAP	98.16	95.64	79.2	208.448	0.0001	S
MAP	115.4	108.68	91.88	325.292	0.0001	S

Table 3. Mean Haemodynamic Values 3 Minutes after Intubation

Details	Control	Lignocaine	Esmolol	F-value	p Value	Significance
Pulse Rate	94.64	93.6	74.56	531.350	0.0036	S
SAP	135.12	129.12	113.36	181.254	0.0036	S
DAP	90.64	85.2	74.16	157.897	0.0036	S
MAP	105.56	102.8	87.2	8.915	0.0036	S

Table 4. Mean Haemodynamic Values at 5 Minutes after Intubation

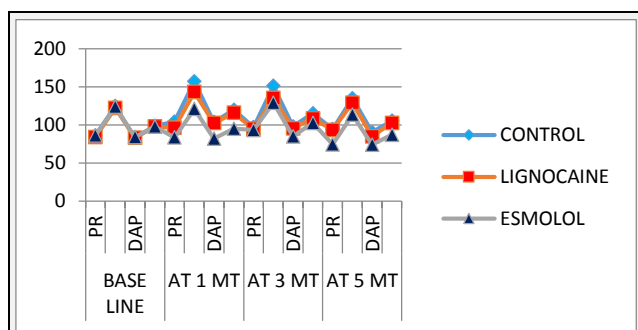


Figure 1. Mean Haemodynamic Values at Base Line, 1, 3 and 5 Minutes after Intubation

At baseline the mean pulse rate, mean SAP(systolic arterial pressure), mean DAP(Diastolic arterial pressure), mean MAP (mean arterial pressure) were comparable in all the three groups, i.e. there is no significant difference between control, Lignocaine and Esmolol groups.⁸ Table 2 shows the mean haemodynamic values of control, Lignocaine and Esmolol groups 1 minute after intubation. ANOVA

revealed that there is significant difference in pulse rate, SAP (systolic arterial pressure), mean DAP (diastolic arterial pressure), mean MAP (mean arterial pressure) between the three groups.

Table 3 shows the mean haemodynamic values of three groups after 3 mins intubation. From the above table it is clear that there statistically significant variation between three groups regarding the haemodynamic values (pulse rate, SAP, DAP and MAP). The ANOVA conducted revealed that there is significant difference between the three groups in the haemodynamic values. The mean pulse rate in the control group at baseline was 86.36±10.60 per minute and at 1, 3 and 5 minutes after intubation were 104.36±10.28 beats per minute, 97.12±7.2 beats per minute, and 94.64±6.58 beats per minute respectively.

At 1, 3 and 5 minutes after intubation, there was significant different between control and Lignocaine groups. The mean pulse rate in Lignocaine group at 1 minute (96.4), 3 min (94.96) and 5 min (91.6) were lower than the control group at 1 min (104.36) 3 min (97.120) and 5 min (94.64)

Mean pulse rate in Esmolol group at baseline was 86.44±7.4 beats/minute and 1, 3 and 5 minutes after intubation were 83.60±7.3 per minute, 79.44±7.10 beats per minute and 74.56±5.8 beats per minute.

DISCUSSION

The circulatory responses to laryngeal and tracheal stimulation were the result of reflex sympathoadrenal stimulation. Although this response is well tolerated in healthy patients, in susceptible individuals this response results in increased cardiac work load; which in turn may culminate in preoperative myocardial ischemia, infarction, rhythm disturbances, acute heart failure, pulmonary oedema and ventricular arrhythmias owing to sudden increase in myocardial oxygen demand.^{9,10} This response is undesirable, especially in patients with hypertension, cardiovascular diseases, cerebrovascular diseases, PIH, hyperthyroidism etc.

In our study we compared I.V. Lignocaine and IV Esmolol to find out how effective these drugs are in suppressing haemodynamic responses to laryngoscopy and intubation. Injection of Esmolol (1.5 mg/Kg) given 3 minutes prior to intubation provided consistent and reliable protection against increase in heart rate during laryngoscopy and endotracheal intubation. Injection Lignocaine (1.5 mg/Kg) given 3 minutes prior to intubation failed to attenuate the increase in heart rate to the same extent as Esmolol during laryngoscopy and endotracheal intubation.

The mean pulse rate in the control group at baseline was 86.36±10.60 per minute and at 1, 3 and 5 minutes after intubation were 104.36±10.28 beats per minute, 97.12±7.2 beats per minute, and 94.64±6.58 beats per minute respectively. This shows that laryngoscopy and endotracheal intubation has caused significant increase in pulse rate in control group which did not come back to baseline level even after 5 minutes. At baseline, the mean pulse rate of control and Lignocaine groups are 86.36±10.6 and 84.24±7.60 respectively. t-test reveals that there is no significant difference between control and Lignocaine groups. At 1, 3 and 5 minutes after intubation, there was significant different

between control and Lignocaine groups. The mean pulse rate in Lignocaine group at 1 minute (96.4), 3 min (94.96) and 5 min (91.6) were lower than the control group at 1 min (104.36) 3 min (97.120 and 5 min (94.64).

Mean pulse rate in Esmolol group at baseline was 86.44 ± 7.4 beats/minute and 1, 3 and 5 minutes after intubation were 83.60 ± 7.3 per minute, 79.44 ± 7.10 beats per minute and 74.56 ± 5.8 beats per minute. This shows that in Esmolol group there was significant attenuation of heart rate following laryngoscopy and endotracheal intubation.

The show data reveals that there is statistically significant difference between the Lignocaine and Esmolol groups. In Lignocaine group, there was increase to pulse rate at 1 minute from baseline value and it decreased at 3 and 5 minutes after intubation. But it did not return to the baseline value even after 5 minutes. In Esmolol group, there was decrease in pulse rate at 1, 3 and 5 minutes after intubation from baseline value. The same observed in all other haemodynamic values like SAP, DAP and MAP at 1, 3 and 5 minutes after intubation and there was statistically significant difference between the two groups.

CONCLUSIONS

A study was done to compare the efficiency of Lignocaine and Esmolol in attenuating the haemodynamic responses to laryngoscopy and intubation in 150 patients. It was observed that injection of single bolus dose of Esmolol hydrochloride^{11,12,13} in the dose of 1.5 mg/Kg body weight given 3 minutes prior to intubation provided consistent and reliable protection and against increase in mean heart rate. SAP, DAP and MAP during laryngoscopy and intubation compared to group-I and -II, whereas singly, bolus I.V. Lignocaine (1.5 mg/Kg) given 3 minutes prior to intubation failed to attenuate^{14,15} the raise in all haemodynamic values to the same extent compared to group-III. In control group all the haemodynamic values significantly increased from baseline and failed to return to baseline early. Tachycardia and hypertension are undesirable in patients with ischemic heart disease. Tachycardia increases myocardial oxygen consumption more than hypertension and leads to greater incidence of myocardial infarction.

From this study, it is concluded that Esmolol¹⁶ a cardioselective beta blocker given 3 minutes prior to intubation attenuates both raise in heart rate and blood pressure, Thus, Esmolol^{17,18} stands out as near ideal drug for attenuating the haemodynamic responses accompanying laryngoscopy and intubation.

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