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A Comparative Study of Lignocaine Nebulization with Intravenous Lignocaine in Attenuation of Pressor Response to Laryngoscopy and Intubation

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Abstract

Background: Laryngoscopy and endotracheal intubation is often associated with hypertension and tachycardia because of sympatho-adrenal stimulation which is usually transient. In patients with cardiovascular and cerebrovascular diseases, the sudden hemodynamic response can produce deleterious effects in the form of myocardial ischaemia or infarction, arrhythmias, cardiac failure, raised ICP and cerebral haemorrhage. In view of this, the present study was undertaken to evaluate and compare the effects of 2% Lignocaine 2 mg/kg nebulization given 10 minutes and 2% Lignocaine 2 mg/kg iv given 90 seconds before induction for attenuation of intubation response. *Materials & methods:* Sixty ASA Grade I & II patients in the age group 20-60 years of either sex scheduled for elective surgeries under general anaesthesia were allocated into Group A and Group B with the sample size of 30 in each. Group A received nebulization with 2% lignocaine 2 mg/kg 10 minutes and Group B received 2% lignocaine 2 mg/kg intravenous 90 sec before induction. Heart rate, systolic and diastolic blood pressure and mean arterial pressure and SpO_2 were recorded, basal values and subsequently at 1st, 3rd, 5th, 7th and 10th minute after intubation. *Results:* It was noted that, Group A, the rise of HR, SBP, DBP, MAP at 1 min after intubation were found to be 24.86 bpm, 9.6 mm Hg, 20.44 mm Hg, 22.30 mm Hg respectively. In Group B, the rise of HR, SBP, DBP, MAP were found to be 11.7 bpm, 3 mm Hg, 2.61 mm Hg, 4.77 mm Hg respectively. *Conclusion:* It was seen that use of lignocaine has suppressed heart rate and blood pressure changes to laryngoscopy and endotracheal intubation. In fact intravenous lignocaine has better suppressing property than nebulization of lignocaine.

Keywords: Laryngoscopy; Endotracheal intubation; Cardiovascular response.

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Introduction

The major responsibility of an anaesthesiologist is to secure airway to provide adequate ventilation to the patient during general anaesthesia. Endotracheal intubation is the gold standard of securing the airway. However, endotracheal intubation requires time, a skilled anaesthesiologist, appropriate instruments and adequate circumstances with respect to space and illumination.

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Laryngoscopy and endotracheal intubation is associated with intense sympatho-adrenal stimulation resulting in increase in heart rate (HR) and blood pressure (BP) consequent to the release of catecholamines [1,2]. The cardiovascular response is a reflex phenomenon. This is mediated by Vagus and Glossopharyngeal cranial nerves. They carry the afferent stimulus from epiglottis and infraepiglottic region activating vasomotor centre to cause a peripheral sympathetic adrenal response resulting in hypertension, tachycardia and arrhythmias [3,4,5].

The hemodynamic response, being transient in nature may not be of much clinical significance in normal healthy individuals [6]. However, in patients with limited myocardial reserve, with raised intracranial pressures (ICP) or intraocular pressures (IOP), the laryngoscopic reaction may predispose to development of pulmonary edema [7], myocardial insufficiency [8] and cerebrovascular accidents [9]. Thus, there is necessity to blunt these harmful laryngoscopic reactions.

Attenuation of stress response to laryngoscopy and intubation has been practiced either by non-pharmacological or pharmacological methods.

The non-pharmacological methods used are smooth and gentle intubation with a shorter duration of laryngoscopy, insertion of Laryngeal Mask Airway (LMA) [10,11] or advanced airways [12] and blocking glossopharyngeal & superior laryngeal nerves. [13]. Pharmacological methods like topical or intravenous lignocaine, high dose of opioids [14], α & β adrenergic blockers [15,16], calcium channel antagonists [17] like diltiazem, verapamil, vasodilators like nitroglycerine [18] and α2 agonists like clonidine [19] & dexmedetomidine [20,21] are used.

Topical anaesthesia with lignocaine in forms of viscous gargles [22], lignocaine aerosols [23] or oropharyngeal sprays [24] remains a popular method alone or in combination with others to attenuate the stress response.

Intravenous lignocaine has been used to suppress cough during tracheal intubation [25], laryngospasm and cough during extubation [26]. It has also been used to suppress airway hyperactivity and mitigate bronchoconstriction [27]. Intravenous lignocaine with its well established centrally depressant and anti-arrhythmic effects is found to be more suitable alternative to attenuate the stress response as compared to other forms of lignocaine [28,29,30]. The purpose of our study is to compare the effect of nebulization of lignocaine with intravenous lignocaine on blunting the hemodynamic response to laryngoscopy and tracheal intubation.

The study will also help us standardize minimal safe dose of lignocaine to attenuate the stress response which can be safely practiced prior to induction making it simple, practical, effective and economical prophylactic method.

Material and Methods

After obtaining approval from ethics committee and informed consent from patients, 60 Patients with ASA grade I and II in the age group of 20 to 60 years of either sex posted for elective surgery to be done under general anaesthesia were divided into two groups randomly as,

Group A - received Lignocaine (2%) nebulization 2 mg/kg 10 minutes prior to induction, N- 30.

Group B - received intravenous Lignocaine (2%) 2 mg/kg 90 seconds prior to induction, N- 30.

A detailed pre-anaesthetic evaluation including history of previous illness, previous surgeries, general physical examination and systemic examination was done. Baseline investigations were carried out. A written informed and valid consent was taken after explaining the anaesthetic procedure in detail. Patient arrived to the preoperative room 30 minutes before surgery and preoperative basal heart rate, non-invasive blood pressure readings, SpO_2, cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II were recorded. The patient in Group A received Lignocaine (2%) nebulization 2 mg/kg undiluted using a simple fitting face mask with Compressor Nebulizer (DeVilbiss-3655I) 10 min before induction. Patient was taken inside Operation Theatre and standard monitors such as Electrocardiogram (ECG), pulse oximeter (SpO_2) and Non-invasive sphygmomanometer (NIBP) cuff were attached. All patients were pre-oxygenated with 100% oxygen for 3 minutes by a face mask. All patients were pre-medicated with Inj. Midazolam 1mg, Inj. Fentanyl 2 µg/kg iv.

The patient in Group B received 2% lignocaine 2 mg/kg body weight 90 sec before induction.

Anaesthesia was induced with inj. propofol 2 mg/kg iv as 1% solution, after loss of consciousness and confirmation of adequacy of mask ventilation endotracheal intubation was facilitated with succinylcholine 1.5 mg/kg iv. Laryngoscopy was performed using Macintosh laryngoscope,

under visualization of vocal cords a lubricated (2% lignocaine jelly) cuffed endotracheal tube of appropriate size was passed. After confirming bilaterally equal air entry, the endotracheal tube was secured. Anaesthesia was maintained using 50% nitrous oxide and 50% of oxygen and 1% sevoflurane. After the patients recovered from succinylcholine further neuromuscular blockade was maintained with non-depolarizing muscle relaxant atracurium 0.5 mg/kg iv.

Heart rate (HR), Systoloic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP) were noted as below-

1. Basal before giving any study drugs and premedication
2. at 1, 3, 5, 7 and 10 minutes after laryngoscopy and intubation

At the end of the procedure patients were reversed with Neostigmine 0.05 mg/kg iv and glycopyrrolate 0.01 mg/kg iv and extubated after recovery of adequate muscle power and consciousness.

Results

Statistical methods:

The collected data was compiled in EXCEL sheet and Master sheet was prepared. Data was presented by visual impression like Bar-Diagram, Histogram. Qualitative was represented in form values & percentages. Chi-square test was used for qualitative data. For comparison of Quantitative variables of two groups unpaired t-test was used.

p- Value < 0.05 – Statistically significant (S)

p- Value > 0.05 – Not significant (NS)

Samples are age matched (Group A mean 38.20 ± 10.63, Group B mean 39.80 ± 12.45) with t = 1.68 and p = 0.098**.** Samples are weight matched (Group A mean 57.00 ± 10.35, Group B mean 60.63 ± 9.92) with t = 1.08 and p = 0.248. There was no significant difference in age, gender and weight distribution in the two groups.

Table 1: Table showing changes in Mean Heart Rate

HR	Group A Mean ± SD	Group B Mean ± SD	t-value	P-value
Basal	86.97 ± 11.23	87.30 ± 13.09	0.145	P=0.928 NS
Post-intubation				
1 Minute	111.83 ± 15.91	99.00 ± 12.25	3.50	P=0.001 S
3 Minute	105.87 ± 16.46	96.90 ± 15.01	2.41	P=0.031 S
5 Minute	95.33 ± 14.81	93.36 ± 12.91	0.743	P=0.477 NS
7 Minute	92.30 ± 14.93	89.73 ± 12.79	0.715	P=0.467 NS
10 Minute	88.33 ± 12.56	87.93 ± 12.41	0.124	P=0.902 NS

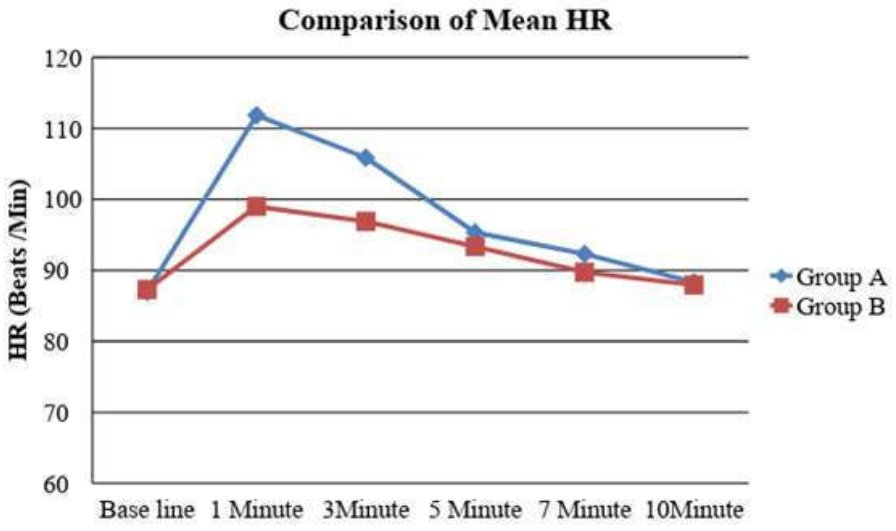

Fig. 1: Graph showing changes in Mean Heart Rate (HR)

Table 2: Table showing changes in Mean Systolic Blood Pressure (SBP)

	Group A Mean ± SD	Group B Mean ± SD	t-value	P-value
Base Line	130.17 ± 11.13	132.10 ± 15.18	0.247	P=0.629 NS
Post-intubation				
1 Minute	139.77 ± 13.39	135.10 ± 14.68	2.09	P=0.039 S
3Minute	130.13 ± 19.53	121.97 ± 17.85	1.98	P=0.031 S
5 Minute	125.73 ± 18.84	114.23 ± 17.08	2.44	P=0.016 S
7 Minute	119.53 ± 15.25	113.10 ± 18.46	3.07	P=0. 003 S
10 Minute	116.66 ± 11.56	113.73 ± 17.35	1.26	P=0.102 NS

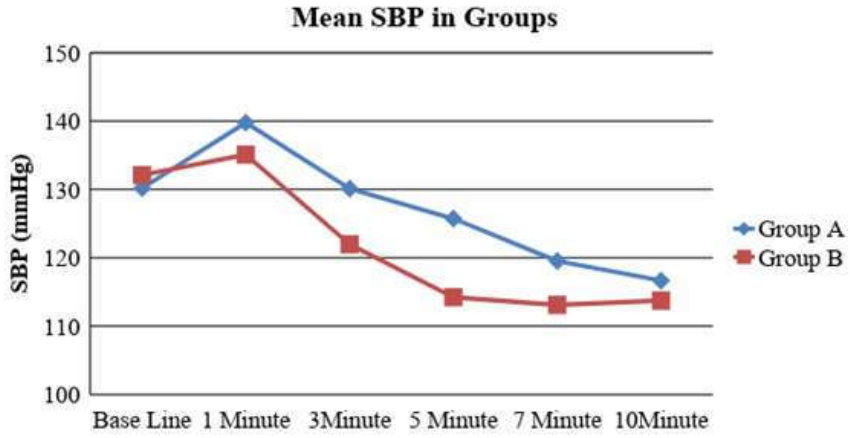

Fig. 2: Graph showing changes in Mean Systolic Blood Pressure (SBP)

Table 3: Table showing changes in Mean Diastolic Blood Pressure (DBP)

DBP	Group A Mean ± SD	Group B Mean ± SD	t-value	P-value
Base Line	83.26 ± 7.83	84.46 ± 8.92	0.553	P=0.582 NS
Post-intubation				
1 Minute	103.70 ± 11.20	87.07 ± 18.69	4.18	P=0.000 S
3 Minute	89.90 ± 10.79	80.26 ± 11.32	3.37	P=0.001 S
5 Minute	81.90 ± 11.27	75.80 ± 12.04	2.25	P=0.028 S
7 Minute	79.80 ± 8.05	76.30 ± 12.47	1.29	P=0. 202 NS
10 Minute	78.93 ± 5.36	75.17 ± 10.85	1.70	P=0.094 NS

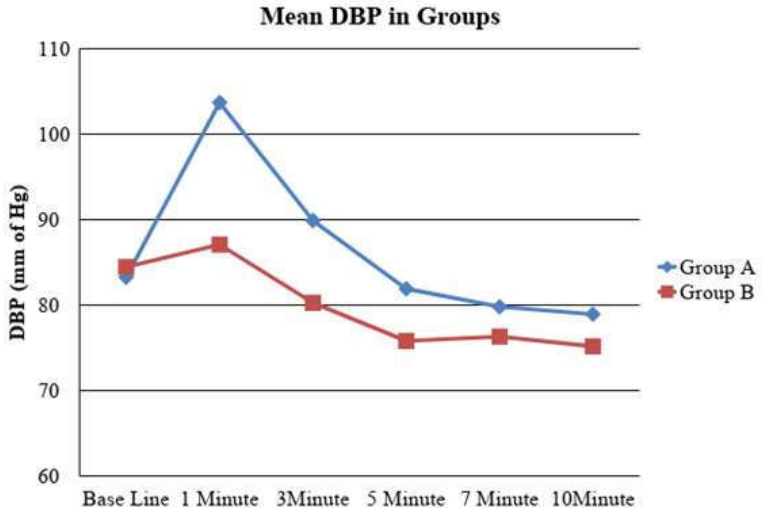

Fig. 3: Graph showing changes in Mean Diastolic Blood Pressure (DBP)

Table 4: Table showing changes in Mean Arterial Pressure (MAP)

MAP	Group A Mean ± SD	Group B Mean ± SD	t-value	p-value
Base Line	98.63 ± 7.83	100.23 ± 10.05	0.763	p=0.642 NS
Post-intubation				
1 Minute	120.93 ± 11.64	105.00 ± 11.01	5.44	p=0.000 S
3 Minute	105.86 ± 12.44	94.23 ± 12.23	3.65	p=0.001 S
5 Minute	95.90 ± 12.43	88.16 ± 12.77	3.29	p=0.001 S
7 Minute	92.03 ± 10.39	88.83 ± 13.42	1.43	p=0. 104 NS
10 Minute	90.86 ± 7.90	87.96 ± 12.38	1.12	p=0.128 NS

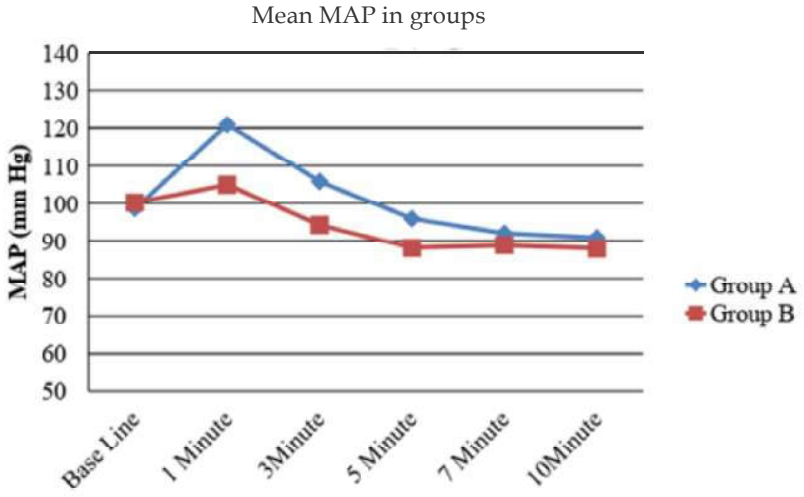

Fig. 4: Graph showing changes in Mean Arterial Pressure (MAP)

Discussion

Local anaesthetic like lignocaine has been the most common agent used for blunting the hemodynamic responses to laryngoscopy and tracheal intubation. Lignocaine has been used by the following routes to blunt the hemodynamic responses to intubation:

- As lignocaine gargle for oropharyngeal analgesia [22]
- As lignocaine aerosol for intratracheal analgesia [23]
- As intravenous infusion for analgesia [31]
- As a topical spray [24]

Lignocaine has been successfully used to blunt the hemodynamic responses to intubation. The mechanisms explained for this action of lignocaine and desirable properties are as follows:

1. Suppression of airway reflexes elicited by irritation of epipharyngeal and laryngopharyngeal mucosa [32].
2. Effectively prevents and treats laryngospasm [26].
3. Excellent cough suppressant [25].
4. Myocardial depression [23].
5. Peripheral vasodilatation [23].
6. Antiarrhythmic properties [33].
7. Increasing depth of general anaesthesia, reduction in anaesthetic requirements of nitrous oxide and halothane [34].
8. Depression of autonomic nervous system [35].
9. Analgesic properties when given intravenously [31].

Gianelly et al. [36] concluded that the concentration of lignocaine in the blood following intravenous administration was directly related to the dose given. They also concluded that an effective safe blood level of 2 to 5 µg/ml is obtained by intravenous bolus of 1 to 2 mg/kg and major side effects may occur with blood levels 9 µg/ml. Adriani [37] asserts that the topical anaesthetic agents applied to the larynx and trachea are readily absorbed from the pulmonary alveoli.

The blood levels achieved after oropharyngeal anaesthesia with viscous lignocaine (25 ml of 2% as mouth wash and gargle 15 min before laryngoscopy) was found to be 0.5 µg/ml at the time of laryngoscopy [20]. The average lignocaine level following aerosol anaesthesia of the upper airway (6-8 ml of a mixture of 1/3 of 2% viscous lignocaine and 2/3 of 4% aqueous lignocaine) was 1.2 µg/ml at 1 minute and 1.4 µg/ml at 2 minutes did prevent PVC [23] in the treated patients even though minimum blood levels effective in suppression of premature ventricular contractions range from 0.6 – 2 µg/ml.

Inhalation of lignocaine aerosol is a safe, simple, effective and generally accepted method. Obvious limitations are small children, uncooperative patients, patients in whom there is danger due to regurgitation and vomiting and lack of time is another limitation. With all the advantages and ease of administration of lignocaine and minimal side effects the present study was carried out to evaluate the efficacy of lignocaine in blunting the hemodynamic response to laryngoscopy and endotracheal intubation using two different routes of administration at similar dosage and look for any side effects.

Mounir Abou-Madi et al. [28] compared two doses of 2% lignocaine when given intravenously for suppression of pressor response and suggested 1.5 mg/ kg provided better control of pressor response compared to 0.75 mg/ kg when given 2 to 3 min before laryngoscopy. Stanlay Tarn et al. [29] observed that intravenous lignocaine at a dose of 1.5 mg/kg attenuated the increase in Heart rate (HR) and Arterial Blood Pressure (ABP), only when given 3 min, before intubation and did not give any protection when given at 1 min, 2 min and 5 min before intubation. Mohan K, Mohana Rupa L [38] stated that Intravenous lignocaine 2% in the dose of 1.5 mg/kg given 3 minutes before laryngoscopy and intubation is helpful in attenuating the cardiovascular response to intubation. Gulabani M et al. [39] said that lignocaine in a dose of 1.5 mg/kg given 3 min before laryngoscopy and intubation was more effective than dexmedetomidine 0.5 µg/kg in attenuating the increase in systolic and DBP at 3 min and 5 min after endotracheal intubation. We used 2 mg/ kg of 2% lignocaine intravenous for attenuation of pressor response and preferred to give 90 sec before induction and intubation was done 90 sec after induction as we used succinylcholine; thus duration between iv lignocaine and intubation was 3 minutes.

Bahaman Venus [40] studied the effects of nebulization of 6ml of 4% lignocaine on cardiovascular response to laryngoscopy and intubation 5 min before induction compared to control with saline nebulization. The pressor response and tachycardia was successfully prevented by the aerosol group than the control. Ahmed M. et al. [41] used Lidocaine 2% (2 mg/kg) in 5 ml saline was added to a standard nebulizer with a full face mask attached with O_2 flow at 3 L/min., then the patient was asked to inhale the local anesthetic vapor deeply for 15 minutes. Patient's tolerance to endotracheal tube in the study group showed a highly significant increase in numbers of patients in grade 0 and highly significant decrease in numbers in grades 1 and 2 in comparison with the control group.

Data Analysis

Heart rate changes (as shown in Table 1 and Figure 1)

In Group A, where nebulization of 2% Inj. Lignocaine 2 mg/kg 10 minutes before laryngoscopy and intubation was used to blunt the pressor response, the base line value of Heart rate (HR) was 86.97 bpm. One minute following laryngoscopy and intubation, the heart rate (HR) increased to 111.83 bpm, representing a rise of 24.86 bpm above the baseline value. Thus the maximal rise in heart rate (HR) seen was by an average of 24.86 bpm. It was seen that the elevated heart rate (HR) started settling down towards base line value by 10 min. In Group B, where 2% Inj. Lignocaine 2mg/kg iv was administered to attenuate the hemodynamic response to laryngoscopy and intubation, the baseline value of Heart rate (HR) was 87.30 bpm. One minute following laryngoscopy and intubation, the heart rate (HR) increased to 99 bpm, representing a rise of 11.70 bpm above the baseline value. Thus the maximal rise of Heart rate (HR) seen in the Group B was by an average of 11.70 bpm. It was seen that the elevated Heart rate (HR) started settling down towards the baseline value by 7 min. The maximum rise in heart rate was noted at 1 min following intubation in both the groups which concurs well with mentioned studies above. The mean rise in Heart rate at 1 min in Group A was 24.86 bpm compared to 11.70 bpm in Group B. The mean rise in the heart rate was comparatively lesser in the intravenous group and statistically significant when compared to the Group A.

Blood Pressure Changes (as shown in table 2, 3, 4 and figure 2, 3, 4)

In Group A, where nebulization of 2% Lignocaine 2 mg/kg 10 min before laryngoscopy and intubation

to blunt the pressor response, the maximal increase in the SBP, DBP and MAP was found to be 9.60 mm Hg, 20.4 4 mm Hg and 22.30 mm Hg respectively.

In Group B, where 2% Lignocaine 2 mg/kg iv was employed 90 sec before laryngoscopy and intubation to blunt the pressor response, the maximal increase in the SBP, DBP and MAP was found to be 3.0 mm Hg, 2.41 mm Hg and 4.77 mm Hg respectively. The attenuation of pressor response was highly significant in the Intravenous group

Conclusion

Lignocaine in both routes is easy, safe and effective method to blunt hemodynamic response to laryngoscopy and intubation. Intravenous lignocaine 2% in the dose of 2 mg/kg 90 sec before induction effectively controlled the hemodynamic response to laryngoscopy and endotracheal intubation. Nebulized lignocaine 2% in the dose of 2 mg/kg was less effective in controlling the hemodynamic changes as compared to intravenous lignocaine 2%.

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