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Evaluation of nebulized lignocaine versus intravenous lignocaine for attenuation of pressor response to laryngoscopy and intubation in controlled hypertensive patients

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Abstract
Objectives
1. To assess the degree of cardiovascular responses to laryngoscopy and endotracheal intubation in controlled hypertensive patients with standard induction techniques.
2. To assess the effectiveness of nebulised lignocaine in attenuating these pressor responses.

Methodology: Total 60 patients aged between 18-65 years k/c/o hypertension taking some antihypertensive medications scheduled for elective surgical procedures belonging to ASA class II under GA were included in our study. The study population was randomly divided into two groups with 30 patients in each group using computer generated randomized table.
Group A (n=30): received 8 ml of 4% lignocaine nebulization 10 min prior to induction.
Group B (n=30): received intravenous lignocaine 2% 1.5 mg/kg 90 seconds prior to induction.
Result: The study revealed that there is significant difference between (p value < 0.05) the two groups during laryngoscopy and endotracheal intubation with respect to heart rate, systolic, diastolic and mean arterial BP.
Conclusion: Study concludes that both nebulized and intravenous lignocaine are effective in attenuating pressor response but nebulized lignocaine gave slightly better results than intravenous lignocaine in attenuating pressor response to laryngoscopy and endotracheal intubation without any significant side effects in controlled hypertensive patients.

Keywords: Intravenous lignocaine, intubation, laryngoscopy, nebulized lignocaine, hypertension, hemodynamic changes

1. Introduction
To secure airway providing adequate ventilation to patient during general anaesthesia is the major responsibility of an anaesthesiologists. Tracheal intubation is the gold standard of securing airway. Even with adequate depth of anaesthesia, direct laryngoscopy and intubation elicits a significant adreno-sympathetic response [1, 2]. The response is manifested by increase in SBP, DBP, MAP & HR, arrhythmias etc. In normotensive patients these effects subside within 3 minutes. But in hypertensive patients these responses are not only exaggerated but also persist for approximately 7 minutes and can result in potentially deleterious effects like left ventricular failure, pulmonary edema [3], myocardial infarction [4], ventricular dysrhythmias and cerebral hemorrhage [5]. Thus, there is necessity to blunt these unwanted laryngoscopic responses. Lignocaine is an amide synthetic local anaesthetic. It has cardio stabilizing action. To prevent these responses nebulized lignocaine is being widely used. Topical anaesthesia with lignocaine applied to the larynx and trachea in a variety of ways remains a popular method used alone or in combination with other techniques.
Hence this study is designed to compare the effectiveness of nebulized lignocaine against intravenous lignocaine.

1.1 Aim
To study the evaluation of Nebulized Lignocaine versus Intravenous Lignocaine for Attenuation of Pressor Response to Laryngoscopy and Intubation in controlled hypertensive patients.

1.2 Objectives

1. To assess the degree of cardiovascular responses to laryngoscopy and endotracheal intubation in controlled hypertensive patients with standard induction techniques.
2. To compare the efficacy of nebulised lignocaine with intravenous lignocaine in attenuating these pressor responses.

2. Materials and Methods

This study was carried out in the Department of Anaesthesiology, Smt. Kashibai Navale Medical College & General Hospital, Pune.

2.1 Study Design: Prospective, randomized double blinded study. Ethical committee clearance was obtained prior to the study.

2.2 Study Period: From August, 2019 to December, 2020 (18 months)

2.3 Sample size: Total 60 patients aged between 18-65years k/c/o hypertension taking antihypertensive medications scheduled for elective surgical procedures belonging to ASA class II under GA.

2.4 Mode of Selection: The subjects were selected by computer generated randomized table and divided in 2 groups 30 each. Anaesthesiologists who prepared and administered drug was different from observer.
Group A (n=30): received 8 ml of 4% lignocaine nebulization 10 min prior to induction.
Group B (n=30): received intravenous preservative free lignocaine 2% 1.5mg/kg 90 seconds prior to induction.

2.5 Inclusion Criteria

a. Patients of either sex, aged between 18-65 years.
b. Patients belonging to American Society of Anaesthesiologist Grade II.
c. Patient scheduled for elective surgeries under general anaesthesia.

2.6 Exclusion Criteria

a. Patients belonging to American Society of Anaesthesiologist Grade III & IV.
b. Patients with known allergies to local anaesthetic drugs.
c. Patients with anticipated difficult airway.

2.7 Procedure

Informed consent was taken from the subjects. Pre-anaesthetic evaluation was done before surgery. A routine preanesthetic examination was conducted assessing: General condition of the patient, Airway by Mallampatti grading, Nutritional status, Body weight of the patient, Detailed examination of the cardiovascular system and Respiratory system. The following investigations were done in all patients: Haemoglobin estimation, Urine examination for albumin, sugar and microscopy, standard 12-lead electrocardiogram, X-ray chest, Blood sugar, LFT, RFT, PT/INR. All patients included in the study were kept nil per orally 10 pm onwards on the previous night. Patients were advised to take morning dose of antihypertensive drug with sip of water on the day of surgery. On arrival of the patients from Group A in the preoperative room, the baseline systolic, diastolic blood pressure, mean arterial pressure and heart rate were recorded in the preoperative room. Then 8 ml of 4% lignocaine nebulization was given 10 min prior to induction. Patient was taken inside OT. The patients were connected to Drager multiparameter monitor which records heart rate, non-invasive measurements of SBP, DBP,
MAP, EtCO2 and continuous ECG monitoring and oxygen saturation. The cardiac rate and rhythm were also monitored from a continuous visual display of electrocardiogram from lead II. After recording the baseline reading, all patients were premedicated with intravenous (IV) glycopyrrolate (4mcg/kg) midazolam 0.03mg/kg,
IV fentanyl (2µg/kg). The patients were pre-oxygenated for 3 minutes via a face mask. The patient in Group B received 2% lignocaine 1.5 mg/kg body weight 90 sec before induction. Induction was done with inj. Propofol till loss of verbal response. Endotracheal intubation was facilitated with IV Vecuronium 0.1mg/kg three minutes prior to laryngoscopy and intubation. Laryngoscopy and oral intubation were performed using appropriately sized Macintosh blade and after confirmation of bilateral equal air entry, the endotracheal tube was fixed. Anaesthesia was maintained using 50% nitrous oxide and 50% of oxygen with sevoflurane and inj. vecuronium. No surgical or any other stimulus was applied during 10 minutes of study period. At the end of the procedure patients were reversed with neostigmine 0.05 mg/kg body weight and glycopyrrolate 8mcg/kg. Hemodynamic parameters of patients including systolic BP (SBP), diastolic BP (DBP), mean arterial pressure (MAP), and heart rate (HR), were recorded as:

1. Baseline before giving study drugs and premedication.
2. At 1,3,5,7 and10 minutes after endotracheal intubation.

Hypotension was defined as SBP ≤ 20% of baseline value.
Tachycardia was defined as HR > 25% of baseline value.
Bradycardia was defined as HR ≤ 20% of baseline value.
Any dysrhythmia was defined as any ventricular or supra ventricular beat or any rhythm other than sinus. Incidences of all these parameters were recorded in both the groups.

3. Results

3.1 Comparison of Demographic Data

Both the groups under study were comparable to each other with respect to age, weight, height and gender. (Table number 1 and 2)

Table 1: Demographic Data

Demographic Variables	Group A: NL (N=30) (Mean ± SD)	Group B: IVL (N=30) (Mean ± SD)	P value
Age	24.23 ± 3.730	25.07 ± 5.382	0.983
Weight	75.48 ± 16.15	73.42 ± 15.40	0.615

The age distribution in group A and group B was from 18 – 65 years with p value 0.983 which is statistically not significant. The mean weight of the patients in both the groups was comparable with p value being 0.615.
There was no significant difference in age, gender and weight distribution in the two groups.

3.2 Heart Rate Variation

Table 2: Comparison of Heart Rate Variation

Time Interval	Group A:NL(N=30) (Mean ± SD)	Group B: IVL (N=30) (Mean ± SD)	P value
Baseline	84.33±5.579	85.27±5.620	0.5182
1 min	78.53±5.303	79.87±4.547	0.2978
3 min	76.93±2.959	79.40±4.207	0.0109
5 min	73.93±2.753	74.60±4.039	0.0115
7 min	69.33±5.390	67.93±6.938	0.3864
10 min	68.60±3.286	70.80±4.089	0.0252

Statistically significant reduction in heart rate was seen (p value < 0.05) at 3, 5 and 10 minutes after endotracheal intubation in group A compared to group B.

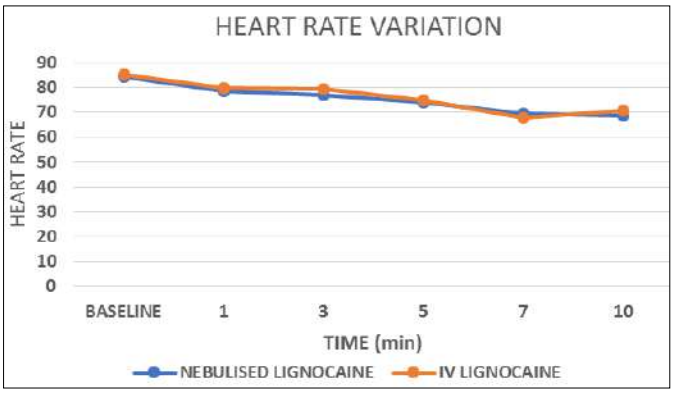

Fig 1: Line diagram showing mean Heart rate Variation

3.3 Systolic Blood Pressure Variation

Table 3: Comparison of Systolic Blood Pressure Variation between Group A and B

Time Interval	Group A:NL (N=30) (Mean ± SD)	Group B: IVL(N=30) (Mean ± SD)	P value
Baseline	129.13±3.884	130.21±4.536	0.326
1 min	112.33±6.216	113.0±5.401	0.657
3 min	97.87±4.812	100.6±5.117	0.0375
5 min	100.80±5.268	103.80±6.178	0.0476
7 min	107.80±8.143	110.53±7.257	0.175
10 min	112.80±8.430	113.87±6.684	0.588

Statistically significant reduction in systolic blood pressure occurred in group A as compared with B group at 3 and 5 minutes after endotracheal intubation.

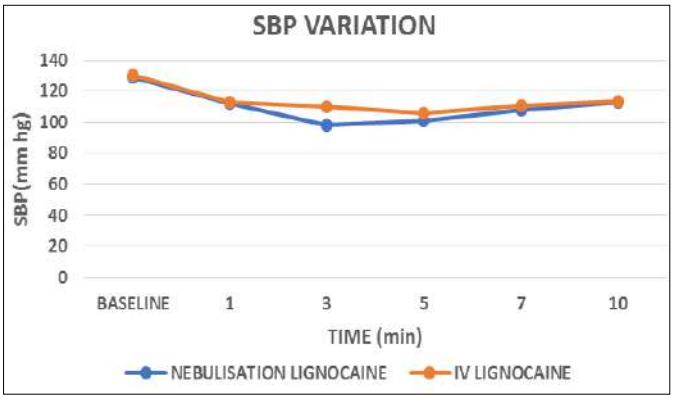

Fig 2: Line diagram showing Mean SBP Variation

3.4 Diastolic Blood Pressure Variation

Table 4: Comparison of Diastolic Blood Pressure Variation

Time Interval	Group A:NL (N=30) (Mean ± SD)	Group B: IVL(N=30) (Mean ± SD)	P value
Baseline	85.40±3.793	85.13±2.501	0.746
1 min	67.93±8.111	68.07±3.769	0.932
3 min	64.73±6.528	66.00±5.356	0.413
5 min	63.73±5.219	64.47±4.718	0.942
7 min	65.13±4.599	65.20±4.506	0.015
10 min	72.23±4.235	73.32±3.712	0.293

The statistically significant reduction is observed in patients with group A as compared with group B at 7 minutes only after endotracheal intubation.

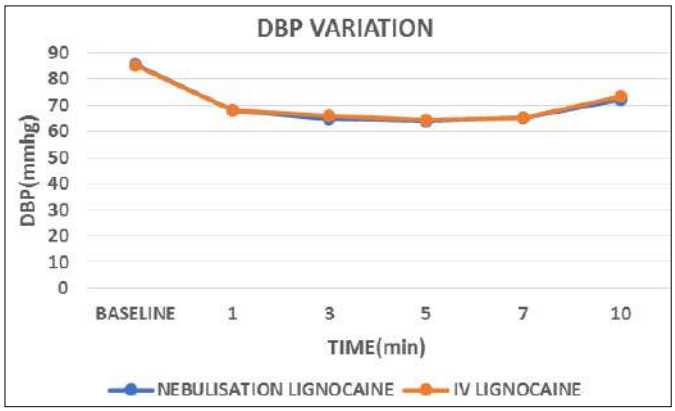

Fig 3: Line diagram showing Mean DBP Variation

3.5 Mean Arterial Pressure Variation

Table 5: Comparison of Mean Arterial Pressure Variation

Time Interval	Group C: NL (N=30) (Mean ± SD)	Group P: IVL (N=30) (Mean ± SD)	P value
Baseline	99.72±2.62	99.16±2.15	0.3692
1 min	82.73±7.11	83.04±3.74	0.8334
3 min	75.78±5.59	78.62±3.96	0.0182
5 min	76.09±3.58	78.24±4.18	0.0366
7 min	79.36±4.46	80.31±3.51	0.3630
10 min	81.60±3.70	81.96±3.05	0.6824

The statistically significant reduction is observed in patients with group A as compared with group B at 3 & 5 minutes after endotracheal intubation.

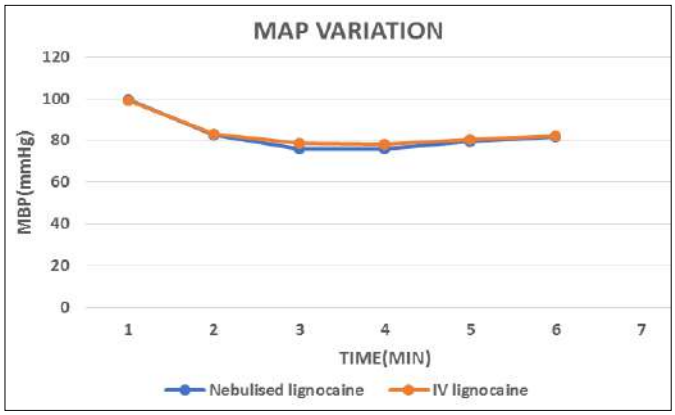

Fig 4: Line diagram showing Mean MAP Variation

4. Discussion

General anaesthesia apart from providing adequate analgesia, amnesia and muscle relaxation should also provide haemodynamic stability ideally. Adequate sedation and antisialogogue effects are also highly desirable. Laryngoscopy and tracheal intubation are associated with marked haemodynamic changes and autonomic reflex activity like rise in heart rate, blood pressure and cardiac arrhythmias. These potentially dangerous changes normally disappear within 5 minutes of laryngoscopy. But in patients with cardiovascular compromise like hypertension, Ischemic heart disease, Cerebrovascular disease and in patients with intracranial aneurysms even these transient changes in haemodynamic can result in potentially harmful effects like left ventricular failure [6], pulmonary oedema, myocardial ischemia [6], ventricular dysrhythmias [7], and cerebral haemorrhage [6]. This is by far the most important indication for attenuation of haemodynamic response to laryngoscopy and tracheal intubation. Many methods like use of inhalational anaesthetic agents [8], lidocaine [9, 10], opioids [11, 12], direct acting vasodilators [6, 7], calcium channel blockers [13, 14], and β-blockers [15, 16], have been tried by various authors for blunting haemodynamic responses to laryngoscopy and intubation. But all such manoeuvres had their own limitations.

For example, with opioids respiratory depression and chest wall rigidity were potential problems, use of halothane was associated with dysrhythmias, calcium channel blockers produced reflex tachycardia, direct acting vasodilators needed invasive haemodynamic monitoring. Beta blockers are also one group of pharmacological agents employed for blunting haemodynamic response to laryngoscopy and intubation. But they blunt the heart rate response better than blood pressure response [17]. Hence a drug which can blunt both the heart rate response and blood pressure response of laryngoscopy and intubation, without having any adverse effects like respiratory depression and post-operative nausea and vomiting, was required for the purpose.

Lignocaine has been successfully used to blunt the hemodynamic responses to intubation.

4.1 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 [18].
2. Effectively prevents and treats laryngospasm [19].
3. Excellent cough suppressant [20].
4. Myocardial depression [21].
5. Peripheral vasodilatation [21].
6. Antiarrhythmic properties [22].
7. Increasing depth of general anaesthesia, reduction in anaesthetic requirements of nitrous oxide and halothane [23].
8. Depression of autonomic nervous system [24].
9. Analgesic properties when given intravenously [25].

Gianelly *et al*. [26] 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 [27] 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 [28]. 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) were 1.2 µg/ml at 1 minute and 1.4 µg/ml at 2 minutes did prevent PVC [29] in the treated patients even though minimum blood levels effective in suppression of premature ventricular contractions ranges 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. Bahaman Venus [30] 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 were successfully prevented by the aerosol group than the control. Ahmed M. *et al*. [31] used Lidocaine 2% (2 mg/kg) in 5 ml saline was added to a standard nebulizer with a full-face mask attached with O2 flow at 3 L/min., then the patient was asked to inhale the local anaesthetic 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. The pulse rate is important as the increase in pulse rate decreases the diastolic time for coronary blood flow and increases myocardial oxygen consumption. The systolic blood pressure can be considered as an indirect estimate of afterload another factor that determines myocardial oxygen consumption.

5. Conclusion

Study concludes that both nebulized and intravenous lignocaine are effective in attenuating pressor response but nebulized lignocaine gave slightly better results in our study than intravenous lignocaine in attenuating pressor response to laryngoscopy and endotracheal intubation without any significant side effects in controlled hypertensive patients.

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