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Original Research Article

Study Comparing the Efficacy of Nebulized and Intravenous Administration of 2% Lidocaine in Reducing the Sympathetic Response to Laryngoscopy

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Conflict of interest: Nil

Abstract

Aim: The aim of the present study was to compare the attenuation of sympathetic response to laryngoscopy and endotracheal intubation while using lidocaine in its aerosolized form and intra-venous form and establish a safer and better route of administration of the drug.

Methods: The present study was conducted at Department of Anesthesiology for one year. we included 120 ASA I & II patients of both the genders who were posted for elective surgeries under general anaesthesia. Patients in all the three groups were equally distributed in variables of age (20-65 years), ASA grades and obvious difficult airway.

Results: In all the 3 groups post endotracheal intubation demonstrated an increase in heart rate, systolic BP, Diastolic BP and mean arterial BP. However, the minimum change in hemodynamic and thus most effective way of attenuating the sympathetic response to laryngoscopy and intubation was seen in the subgroup where lidocaine was administered intravenously.

Conclusion: In the present study we concluded that the use of lignocaine when used in combination with opioid for laryngoscopy and endotracheal intubations reduces the increase in heart rate and blood Pressure. However, the attenuation of exaggerated sympathetic response was much more in the subset of patients where intravenous lidocaine was used.

Keywords: Laryngoscopy, Endotracheal Intubation, Cardiovascular Response, Nebulized Lidocaine.

Introduction

Laryngoscopy and tracheal intubation after induction of anesthesia generate pressure and sympathoadrenal responses which are thought to be somatovisceral reflexes caused by the stimulation of epipharynx and laryngopharynx. [1] Laryngoscopy alone generates essentially the same pressor response as done by laryngoscopy followed by intubation. [2] It starts within 5 s, reaches a peak in 1–2 min, and returns to baseline within 5 min. [3] These responses result in increased circulatory catecholamines, heart rate (HR), blood pressure, myocardial oxygen demand, and dysrhythmias. The rise in HR and blood pressure is usually transient, variable, and unpredictable. Average increase in HR has been reported to be 23 beats and increase in blood pressure by 53/54 mmHg and decrease in the left ventricular ejection fraction by approximately 20%. [4,5]

Although such a response would likely be tolerated by healthy patients, these changes may be associated with myocardial ischemia and cerebral hemorrhage in those with a significant coronary artery or cerebrovascular diseases. [6] Several techniques have been tried in an effort to attenuate adverse hemodynamic responses to intubation. Commonly used techniques include increasing the depth of anesthesia by heavy premedication, potent narcotics such as fentanyl [7] and inhalational anesthetic agents. [8] Others include intravenous (IV) and topical lignocaine, clonidine, calcium channel blockers, sodium nitroprusside, beta-adrenergic blockers, and magnesium sulfate but none is ideal.

Different studies evaluate the effects of lignocaine on the hemodynamic response of intubated patients. Most of these studies, however, attempt to

investigate the effect of IV lignocaine in comparison to other drugs or control groups. For instance, by comparing the effects of lignocaine and fentanyl on the hemodynamic response of tracheal intubation patients, Splinter and Cervenko [9] observe that IV lignocaine reduces the patients' systolic blood pressure (SBP) more effectively than fentanyl. It is also shown that lignocaine complications are less than those of fentanyl. Feng et al [10] on the other hand, do not report any effect of IV lignocaine on the hemodynamic response in patients undergoing intubation and laryngoscopy, as opposed to the reported for fentanyl. According to the existing studies, the effects of lignocaine on improving the hemodynamic response of intubation patients are yet to be fully understood [11,12] and the use of lignocaine spray – rather than IV lignocaine – is more convenient and brings about fewer complications. [12,13] Hence, it seems quite feasible to carry out a clinical study to evaluate the effects of lignocaine spray on the hemodynamic response of intubation patients. In addition, due to the complications associated with the hemodynamic response of intubation patients, improving such responses is of great significance.

The aim of the present study was to compare the attenuation of sympathetic response to laryngoscopy and endotracheal intubation while using lidocaine in its aerosolized form and intra-venous form and establish a safer and better route of administration of the drug.

Materials and Methods

The present study was conducted at Department of Anesthesiology, Patna Medical College and Hospital, Patna, Bihar, India for one year. we included 120 ASA I & II patients of both the genders who were posted for elective surgeries under general anaesthesia. Patients in all the three groups were equally distributed in variables of age (20-65 years), ASA grades and obvious difficult airway.

Group A received standard anaesthesia techniques using Ondansetron 4mg iv, Fentanyl @2mcg/Kg, Propofol @2mg/Kg and Atracurium @ 0.5mg/kg. In addition to the standard anaesthesia techniques Group B received nebulization with 2% lignocaine @ 1.5 mg/kg 10 minutes prior to the endotracheal intubation and Group C received 2% lignocaine @ 1.5 mg/kg intravenous 90 sec before induction. Standard anaesthesia monitoring techniques were ensured in all the 3 groups. The variables we included in the study are heart rate, systolic and diastolic blood pressure and mean arterial pressure. Basal and subsequent values of the included variables were recorded at 1st, 3rd, 5th, and 10th minute after intubation were recorded. Patients who were either unwilling to participate in the study or already on beta/ alpha blocking agents and those requiring laryngoscopy for more than 30 secs or more than 2 attempts were excluded from the study.

Group B received nebulization with 1.5 mg/kg of 2% Lidocaine diluted in 5 ml of 0.9% normal saline using Compressor Nebulizer (DeVilbiss-3655I) face mask10 min before induction.

Group C received 1.5 mg/kg of intra venous preservative free Lidocaine (Loxicard) 90 seconds before starting intra venous induction.

A conventionally trained anaesthesiologist performed Laryngoscopy and endotracheal intubation in all the subgroups using Macintosh size 3 blade and Gum elastic bougie to prevent airway manipulation and decrease the duration of laryngoscopy after loss of verbal response. Anaesthesia was maintained using combination 50% nitrous oxide with 50% of oxygen and 1% sevoflurane. Minimum monitoring as per ASA standards were used in all the subgroups.

Statistical Methods

Data was compiled in EXCEL sheet and Master sheet was prepared. For comparison of Quantitative variables of three groups unpaired t-test was used. p- Value < 0.05 was considered to be statistically significant (S) while p Value > 0.05 was considered not significant (NS).

Results

Table 1: Mean of heart rate at basal, 1, 3, 5 and 10 minutes

Heart Rate	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	P value
Pre-Anaesthesia	85.95±10.22	86.94±12.28	88.32±12.08	0.94
1 Mins	136.74±17.3	112.88±16.94	98.00±12.28	0.001
3 Mins	135.35±18.2	106.84±16.44	97.93±15.02	0.034
5 mins	122.4±19.1	95.34±14.84	94.36±12.98	0.450
10 mins	82.78±9.1	88.32±12.58	87.93±12.48	0.910

Mean Heart Rate Group A Mean ± SD Group B Mean ± SD and Group c Mean ± SD P-value Basal 85.95±10.22, 86.94±12.28 and 88.32±12.08 and P=0.94 NS. Post-intubation 1 Minute 136.74±17.3, 112.88±16.94 and 98.00±12.28 P=0.001 which was considered statistically significant. Post intubations at 3 Minute 135.35±18.2, 106.84±16.44 and 97.93±15.02 P=0.034 and found to be statistically

significant. At 5 Minute post intubation 122.4±19.1, 95.34±14.84 and 94.36±12.98 with P=0.450. These results were not found to be statistically significant At 10 Minutes 82.78±9.1, 88.32±12.58 and 87.93±12.48 and p value of 0.910 which was found to be statistically not significant.

Table 2: Means of Systolic BP at basal, 1, 3, 5 and 10 minutes

Mean Systolic BP	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	P value
Pre Anaesthesia	114.1±12.4	130.16±11.14	132.18±15.15	0.634
1 Mins	147.73±22.5	138.82±12.38	136.14±14.66	0.045
3 Mins	152.38±18.2	131.19±19.51	122.92±17.83	0.036
5 mins	150.4±19.5	126.74±18.82	115.25±17.03	0.018
10 mins	82.78±9.1	117.63±11.59	114.76±17.33	0.106

Mean Systolic Blood Pressure (SBP) Group A Mean ± SD Group B Mean ± SD P-value Base Line 114.1±12.4, 130.16±11.14, 132.18±15.15. P=0.634 Not significant Post-intubation 1 Minute 147. 73±22.5, 138.82±12.38, 136.14±14.66 P=0.045 Statistically significant. 3 Minute 152.38±18.2, 131.19±19.51, 122.92±17.83 P=0.036 S. 5 Minute 150.4±19.5, 126.74±18.82, 115. 25±17 .03 P=0.018 S. 10 Minute 82.78±9.1,117.63±11. 59, 114.76 ±17 .33P=0.106 NS.

Table 3: Means of Diastolic BP at basal, 1, 3, 5 and 10 minutes

Mean Diastolic BP	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	P value
Pre Anaesthesia	79.11±9.01	82.28±7.73	85.45±8.82	0.590
1 Mins	102±10.26	88.0±11.19	86.04±18.62	0.000
3 Mins	101±12.88	89.91±10.75	81.29±11.35	0.001
5 mins	98±10.10	82.98±11.29	76.80±12.08	0.032
10 mins	75±6.74	78.92±5.35	75.15±10.80	0.098

Mean Diastolic Blood Pressure (SBP) Group A Mean ± SD Group B Mean ± SD P-value Base Line 79.11±9.01, 82.28±7.73, 85.45±8.82 and p value 0.590 which was statistically not significant. At 1 minute post-intubation 102±10.26, 88.0±11.19, 86.04±18.62 and p of 0.0 which was statistically significant. At 3 mins post intubation 101±12.88, 89.91±10.75, 81.29±11.35 and p of 0.001 which was statistically significant. At 5 minutes post intubation 98±10.10, 82.98±11.29, 76.80±12.08 and p value of 0.028 which was statistically significant. At 10 minutes post-intubation 75±6.74, 78.92±5.35, 75.15±10.80 with p value of 0.094 which was statistically non-significant.

Table 4: Means of mean arterial pressure at basal, 1, 3, 5 and 10 minutes

Mean MAP	Group A Mean ± SD	Group B Mean ± SD	Group C Mean ± SD	P value
Pre Anaesthesia	99±7.99	98.63±7.83	100.23±10.05	0.648
1 Mins	127±12.38	121.99±12.58	106.00±12.01	0.750
3 Mins	107.93±10.32	106.84±12.48	95.25±12.25	0.001
5 mins	112.38±9.90	96.94±12.48	88.12±12.78	0.001
10 mins	98±5.55	90.88±7.3	87.93±12.4	0.132

Mean of Mean Arterial Pressure Group A Mean ± SD Group B Mean ± SD P-value Base Line 99±7.99, 98.63±7.83, 100.23±10.05 p value 0.648 which was statistically not significant. At 1 minute post-intubation 127±12.38, 121.99±12.58, 106.00±12.01 and p=0.750 which was statistically not significant. At 3 mins post intubation 107.93±10.32, 106.84±12.48, 95.25±12.25 and p of 0.001 which was statistically significant. At 5 minutes post intubation 112.38±9.90, 96.94±12.48, 88.12±12.78 and p value of 0.001 which was statistically significant. At 10 minutes post intubation 98±5.55, 90.88±7.3, 87.93±12.4 with p value of p=0.132 which was statistically non-significant.

Discussion

Balanced anaesthesia techniques mandate minimum hemodynamic disturbances during the peri-operative period. Although transient, laryngoscopy and endotracheal intubation by far have been described as the most important causes in increasing the heart rate and blood pressure peri-operatively. [14] Tachycardia and hypertension due to laryngoscopy and endotracheal intubation can increase systolic blood pressure by 40-50% and heart rate to 20 per minute. [15] More common than not these responses are well tolerated in healthy individuals, nonetheless they may lead to various complications including but not restricted to myocardial ischemia, cerebral haemorrhage in susceptible subset of patients. [16] The cardio-vascular response is mediated vide the Xth and XIth Cranial nerves via the afferent impulses carried from epiglottis and infra-glottic regions which then activate the vasomotor centres leading

to intense sympathetic response causing hypertension, tachycardia and arrhythmias. [17,18]

In all the 3 groups post endotracheal intubation demonstrated an increase in heart rate, systolic BP, Diastolic BP and mean arterial BP. However the minimum change in hemodynamic and thus most effective way of attenuating the sympathetic response to laryngoscopy and intubation was seen in the subgroup where lidocaine was administered intravenously. Nebulized lignocaine was found least effective in all the groups, and all the hemodynamic parameters were significantly increased after intubation when compared with other groups. Combining nebulized lignocaine and IV fentanyl was found most effective, but it did not reach the significant level when compared with IV fentanyl alone. Therefore, nebulization with lignocaine did not offer any additional advantage in attenuating hemodynamic response. Our study has shown nebulized lignocaine as least effective in attenuating hemodynamic response to intubation. This probably may be due to several reasons. Laryngoscopy and intubation are a two part process, each contributing to the hemodynamic response independently. Pressure by laryngoscope blade not only on the mucous membrane but also to the submucosal deep proprioceptors is responsible for these reactions which cannot be blocked by topical anesthesia. [19] The inhalation method provides inadequate anesthesia of trachea. A major part of the drug is lost to air and in patient's mouth during nebulization which is supported by findings of Chinn et al [20] who demonstrated that up to 60% of lignocaine dose can be lost via nebulized route.

Gianelly et al established 2 to 5 mcg/ml plasma concentration of lidocaine as safe for preventing hemodynamic disturbance. However at concentration of greater than 9 mcg/ml the side effects may occur. [21] Adriani et al postulated that the absorption of aerosolized lidocaine is vide the pulmonary alveoli and this method is generally considered to be safe, simple, and effective. [22] Various other studies viz. Mounir Abou-Madi et al [23] and Stanlay Tarn et al [24] 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. Sarvanan S et al., conducted a study to compare the effect of lignocaine nebulisation with intravenous lignocaine on stress response to laryngoscopy and tracheal Intubation. [25] Ninety patients scheduled for elective surgeries under general anaesthesia were allocated into three groups, Group C, Group I, Group N with the sample size of 30 in each. Group I received 2% lignocaine 2 mg/kg intravenous 90 seconds and Group N received nebulisation with 2% lignocaine 2 mg/kg 10 minute before induction. Miller CD and Warren SJ, noticed rise in SBP to be 33 mmHg, DBP to be 37 mmHg. [26] Sarvanan S et al., noticed that increase in SBP, DBP and MAP in Group I, one minute following laryngoscopy and intubation, was 17.54 mmHg, 13.84 mmHg and 16.1 mmHg, that is maximum above the baseline value, then started settling down towards the baseline value by 10 to 15 minutes. [25]

In the present study we also used 1.5 mg/ kg of 2% lignocaine intravenous for attenuation of pressor response and preferred to give 90 sec before induction and intubation while following standard anaesthesia techniques. We also observed that the use of nebulized Lidocaine along with fentanyl was not as effective in decreasing the pressor response to endotracheal intubation as the combination of intra venous form of lidocaine.

Conclusion

In the present study we concluded that the use of lignocaine when used in combination with opioid for laryngoscopy and endotracheal intubations reduces the increase in heart rate and blood Pressure. However the attenuation of exaggerated sympathetic response was much more in the subset of patients where intravenous lidocaine was used.

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