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ORIGINAL RESEARCH

To evaluate the efficacy of nebulized lignocaine versus intravenous lignocaine for attenuation of the hemodynamic response to laryngoscopy and tracheal intubation: A randomised case control study

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Abstract

Background: The introduction of laryngoscopy and tracheal intubation has ushered in a new era in the field of anaesthesiology, resulting in improved safety by providing more control over airway management and breathing during anaesthesia procedures. To evaluate the efficacy of nebulized lignocaine against intravenous lignocaine in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

Material and methods: The research included a cohort of 90 patients, categorized as ASA grade I and II, aged 18 to 45 years, who were scheduled to have elective surgeries under general anaesthesia. The participants were randomly assigned to three groups, each consisting of 30 individuals. This research comprised patients who were categorized as ASA grade I and II and were between the ages of 20 and 45 years. The first readings included heart rate, blood pressure, SpO2, cardiac rate, and rhythm.

Results: The initial heart rate was comparable across the three groups, with Group C at 77.98 ± 3.34 bpm, Group I at 77.01 ± 2.87 bpm, and Group N at 77.87 ± 2.43 bpm, indicating no statistically significant variation (p=0.13). At the 2-minute mark, Group C had a greater heart rate (95.12 ± 3.82 bpm) in comparison to Group I (86.32 ± 2.99 bpm) and Group N (89.16 ± 3.16 bpm), with statistically significant differences (p=0.03). After 10 minutes, Group C had a heart rate of 82.15 ± 2.27 bpm, which was higher than the heart rates of Group I (78.45 ± 3.03 bpm) and Group N (79.45 ± 1.95 bpm) (p=0.05). These variations in heart rates were statistically significant. The baseline systolic blood pressure was comparable throughout the groups, with Group C having a mean of 121.65 ± 2.65 mm Hg, Group I having a mean of 119.87 ± 2.84 mm Hg, and Group N having a mean of 120.11 ± 2.54 mm Hg. There was no statistically significant difference seen between the groups (p=0.34). After 10 minutes, Group C had a systolic blood pressure of 120.88 ± 2.42 mm Hg, which was considerably higher than Group I (117.09 ± 2.37 mm Hg) and Group N (118.87 ± 2.44 mm Hg) (p=0.02). Initially, there were no notable variations in diastolic blood pressure between Group C (81.78 ± 2.23 mm Hg), Group I (80.05 ± 2.34 mm Hg), and Group N (80.73 ± 2.44 mm Hg) (p=0.17). At the 2-minute mark, Group C exhibited a greater diastolic blood pressure (91.68 ± 2.22 mm

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Hg) in comparison to Group I (86.97 ± 2.37 mm Hg) and Group N (88.56 ± 2.99 mm Hg) (p=0.02). After 10 minutes, Group C exhibited a diastolic blood pressure of 81.45 ± 2.79 mm Hg, which was notably greater than the diastolic blood pressure of Group I (78.04 ± 1.46 mm Hg) and Group N (79.44 ± 1.99 mm Hg) (p=0.02).

Conclusion: The results of our study show that the use of 2% Lignocaine, either through intravenous injection (Group I) or nebulization (Group N), successfully decreased the hemodynamic responses (heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure) to laryngoscopy and endotracheal intubation in comparison to the control group. Both the intravenous (IV) and nebulized methods of delivering Lignocaine were equally effective in lowering the hemodynamic response.

Keywords: Nebulized, Lignocaine, Intravenous, Hemodynamic, Laryngoscopy, Tracheal intubation

Introduction

The introduction of laryngoscopy and tracheal intubation has ushered in a new era in the field of anaesthesiology, resulting in improved safety by providing more control over airway management and breathing during anaesthesia procedures. The initiation of a sympathoadrenal response is believed to occur due to the stimulation of the epipharynx and laryngopharynx during laryngoscopy and tracheal intubation. The onset of these reactions occurs within 5 seconds, reaches its maximum intensity within 1-2 minutes, and recovers to its original level within 5 minutes. These reactions include heightened levels of circulating catecholamines, heart rate (HR), blood pressure (BP), myocardial oxygen demand, and dysrhythmias. The average rise in heart rate has been reported to be 23 beats, while the increase in blood pressure is 53/54 mmHg and the reduction in left ventricular ejection fraction is 20%. In a recent research done in 2021, it was found that there was a significant increase of 3% in mean blood pressure after laryngoscopy and tracheal intubation. While healthy patients may tolerate this reaction, those with substantial coronary artery or cerebrovascular disease may have myocardial ischemia and cerebral hemorrhage as a result of these alterations.[1,2] Preventing these pressor reactions is a crucial objective in therapeutic practice, especially for patients with cardiac illness. Tachycardia and hypertension disrupt the balance between myocardial oxygen demand and supply, making the heart more susceptible to ischemia, infarction, and heart failure. The reduction of the physiological reactions to laryngoscopy and intubation may be achieved by many methods. These include deepening the level of anaesthesia, applying local anaesthesia to the upper respiratory tract before laryngoscopy using lignocaine, administering medicines that diminish these responses, or using novel airway devices.[3] The selection of the optimal methodology or medication is contingent upon factors such as the urgency and length of the procedure, the preferred method of anaesthesia, the method of drug delivery, and the patient's medical condition. Several research have examined the impact of lignocaine in various forms, such as aerosol, sprays, viscous lignocaine, and intravenous administration, to mitigate these effects. IV lignocaine has been used to inhibit laryngospasm and coughing throughout the processes of tracheal intubation and extubation.[4-8] A study was done to examine the impact of inhaled and intravenous lignocaine on reflex bronchoconstriction. It was shown that lignocaine effectively decreased bronchoconstriction in both experimental approaches. Nevertheless, the group that was administered lignocaine by inhalation saw significantly reduced levels of the drug in their plasma.[9]

Aims and objectives: To evaluate the efficacy of nebulized lignocaine against intravenous lignocaine in attenuating the hemodynamic response to laryngoscopy and tracheal intubation.

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Material and methods

The present study was case control that included a sample of 90 participants who were categorized as ASA grade I and II, aged between 20 and 45 years, who were scheduled to have elective surgeries under general anaesthesia. The study included patients of both sexes. The present study has been carried out at the Department of Anaesthesia, Nalanda Medical College and Hospital, Patna, Bihar, India. All participants were provided with detailed information about the anaesthetic technique and gave their permission in writing after being fully informed. The patient was given permission in a language that they could comprehend. Each patient provided written informed consent after receiving approval from the ethical committee. The study was carried out over an approximate two-year period, from January 2022 to December 2023.Data such as name, age, etc. was recorded.

Inclusion criteria

- Patients were classified as having ASA grades I and II.
- Age between 20 and 45 years.
- Patients to give written informed consent.
- Available for follow-up.

Exclusion criteria

- Patients who did not consent to the study.
- Age < 20 or >45 years.
- Patients with pre-existing hypertension
- Patients who had chronic obstructive pulmonary disease (COPD), stroke, angina, heart attacks, psychiatric illness, severe liver or renal disorders, known hypersensitivity to Lignocaine or its preservatives, undergoing emergency surgical procedures.
- Those unable to attend follow-up.

The participants were randomly assigned to three groups, each consisting of 30 individuals. This research comprised patients who were categorized as ASA grade I and II and were between the ages of 20 and 45 years.

After preanesthetic evaluation, all patients were premedicated with tablet Ranitidine 150mg and tablet lorazepam 1 mg night before surgery. On arrival at the operation theatre, monitors including noninvasive blood pressure (NIBP), electrocardiograph (ECG), and oxygen saturation (SpO_2) were connected. Baseline values of HR, SBP, DBP, mean arterial pressure (MAP), and saturation were noted. Patients in the N group were nebulized with 2mg/kg of 2% lignocaine for 20 min, and patients in the I group were given O_2 through the nebulizer. Injection fentanyl 2 μg/kg was given to all the groups. Following this, thiopentone 5 mg/kg was given till the loss of eyelash reflex. After checking the adequacy of mask ventilation with 100% O_2, either lignocaine 1.5 mg/kg in 10 ml of saline or 10 ml of saline was given to the patients depending on the group. Succinylcholine 1.5 mg/kg was given in both the groups after 30 s of lignocaine administration. Mask ventilation with 100% O_2 was continued for 60 s, and then, laryngoscopy was attempted by anaesthetists not involved in the study using an appropriate size laryngoscopy blade. Intubation was done using an appropriate size cuffed endotracheal tube, and cuff was inflated with appropriate amount of air. The position of the tube was confirmed by auscultation for bilateral air entry and observing the capnogram.

HR, SBP, MAP, and DBP were monitored by an automated BP cuff before induction (baseline values) and then at 1-min interval up to 5 min after intubation. Arrhythmias if any and the type of arrhythmia were also noted.

Anaesthesia was maintained with 66% N_2O in O_2 and isoflurane. Fentanyl and vecuronium were given as per the patient requirement, and after completion of procedure extubation was done after giving reversal agents.

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Methodology

After pre-anaesthetic evaluation, Every patients received a pre-medication of 1mg of lorazepam tablets to alleviate anxiety and 150 mg of Ranitidine tablets to decrease gastric secretions night before surgery. Patients were transported to the preoperative room 30 minutes before to the procedure. Heart rate, blood pressure, SpO2, cardiac rate, and rhythm were observed as the first measurements.

Group N: Patients received nebulized 2% Lignocaine 2 mg/kg using a fitting face mask with Nebulizer 10 minutes before induction of anaesthesia. An IV line was secured using an 18G cannula, and patients were connected to non-invasive monitoring with electrocardiograph, pulse oximeter, and non-invasive BP machine. All patients received Inj. Midazolam 1 mg IV and 100% oxygen for 3 minutes.

Group I: Patients received 2% Lignocaine 1.5 mg/kg by slow intravenous route 90 seconds before induction.

Group C: Control group received no test drug.

Anaesthesia was induced with a 2.5% solution of thiopentone sodium at a dosage of 5 mg/kg. All patients received an intravenous (IV) injection of 0.2 mg of Glycopyrrolate and 2mcg/kg of fentanyl. To facilitate the procedure of endotracheal intubation, succinylcholine was administered intravenously at a dosage of 1.5 mg per kilogram. A Macintosh laryngoscope was used to perform laryngoscopy. The anaesthesia was maintained using a blend of 66% nitrous oxide, 33% oxygen, and isoflurane. After recovering from succinylcholine, the neuromuscular paralysis was maintained by providing non-depolarizing muscle relaxants such as vecuronium. The recorded parameters consist of heart rate (measured in beats per minute), systolic blood pressure (measured in mm Hg), diastolic blood pressure (measured in mm Hg), and mean arterial pressure (measured in mm Hg). Measurements were obtained at the beginning and at intervals of 2, 4, 6, 8, and 10 minutes following the operations of laryngoscopy and endotracheal intubation. After completing the surgery, the reversal procedure was performed by giving Neostigmine intravenously at a dose of 0.05 mg/kg and Glycopyrrolate intravenously at a dosage of 0.01 mg/kg.

Statistical analysis

Statistical analysis was performed on the obtained data using SPSS and Microsoft. Categorical data were shown using frequencies and proportions. Parametric variables between the groups were studied using the Student's test. Nonparametric variables and the data's significance was assessed by the Chi-square test. A ‘P’ value <0.05 is considered significant.

Results

The study included a total of 90 participants, who were divided into three groups of 30 persons each: Control (Group C), Intravenous Lignocaine (Group I), and Nebulized Lignocaine (Group N). The parameters that were assessed were heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure. Measurements were recorded at the initial stage and at 2, 4, 6, 8, and 10 minutes after the procedures of laryngoscopy and endotracheal intubation. The initial heart rate was comparable across the three groups, with Group C at 77.98 ± 3.34 bpm, Group I at 77.01 ± 2.87 bpm, and Group N at 77.87 ± 2.43 bpm, indicating no statistically significant variation (p=0.13). At the 2-minute mark, Group C had a significantly higher heart rate (95.12 ± 3.82 bpm) compared to Group I (86.32 ± 2.99 bpm) and Group N (89.16 ± 3.16 bpm), with a statistically significant difference (p=0.03). At the 4-minute mark, Group C exhibited a greater heart rate (93.05 ± 2.85 bpm) compared to Group I (85.21 ± 2.99 bpm) and Group N (87.04 ± 2.83 bpm), with a statistically significant p-value of 0.02. At 6 minutes, the pattern persisted with Group C having a heart rate of 89.01 ± 2.51 bpm, Group I with 83.43 ± 2.82 bpm, and Group N with 84.66 ± 2.71 bpm (p=0.03).

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At the 8-minute mark, Group C exhibited a heart rate of 86.04 ± 2.97 beats per minute (bpm), which was considerably greater than the heart rates of Group I (81.12 ± 3.34 bpm) and Group N (83.09 ± 2.77 bpm) (p=0.03). After 10 minutes, Group C had a heart rate of 82.15 ± 2.27 bpm, which was higher than Group I (78.45 ± 3.03 bpm) and Group N (79.45 ± 1.95 bpm) with a statistical significance of p=0.05, showing significant variations (Table 1).

Table 1: Heart Rate (bpm) at Various Time Intervals

Time (minutes)	Group C	Group I	Group N	p-value
Baseline	77.98 ± 3.34	77.01 ± 2.87	77.87 ± 2.43	0.13
2	95.12 ± 3.82	86.32 ± 2.99	89.16 ± 3.16	0.03
4	93.05 ± 2.85	85.21 ± 2.99	87.04± 2.83	0.02
6	89.01 ± 2.51	83.43 ± 2.82	84.66 ± 2.71	0.03
8	86.04 ± 2.97	81.12 ± 3.34	83.09 ± 2.77	0.03
10	82.15 ± 2.27	78.45± 3.03	79.45± 1.95	0.05

Figure 1: Heart rate (bpm) at time interval in minutes

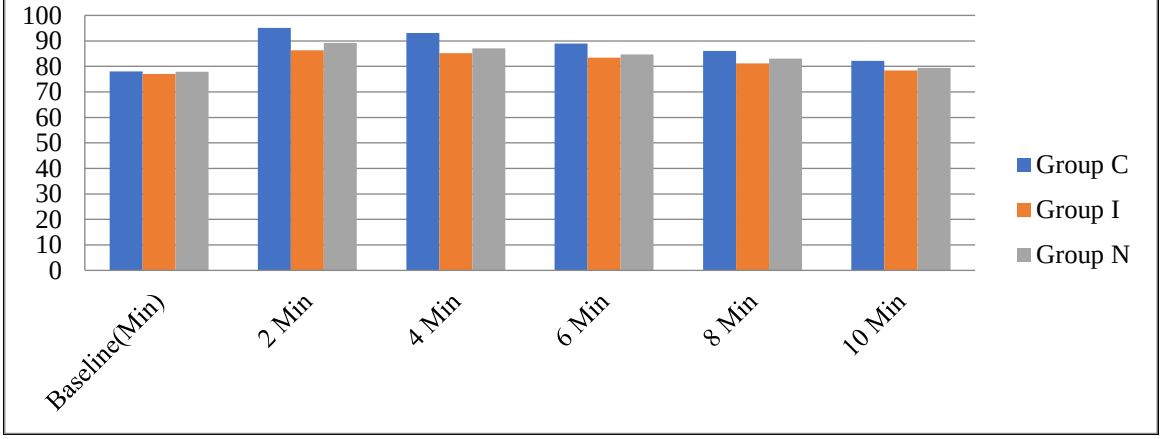

Table 2: Systolic Blood Pressure (mm Hg) at Various Time Intervals

Time (minutes)	Group C	Group I	Group N	p-value
Baseline	121.65± 2.65	119.87± 2.84	120.11 ± 2.54	0.34
2	140.65 ± 3.51	125.82 ± 3.21	130.03 ± 2.27	0.02
4	136 .87± 2.65	124.66 ± 2.21	127.67 ± 2.76	0.01
6	130.43 ± 2.21	120.11 ± 2.21	123.98± 2.33	0.01
8	126.45 ± 2.55	120.13 ± 2.61	121.84 ± 2.37	0.03
10	120.88 ± 2.42	117.09 ± 2.37	118.87 ± 2.44	0.02

Table 2 show that the baseline systolic blood pressure was comparable across the groups, with Group C at 121.65 ± 2.65 mm Hg, Group I at 119.87 ± 2.84 mm Hg, and Group N at 120.11 ± 2.54 mm Hg, indicating no statistically significant variation (p=0.34). At the 2-minute mark, Group C exhibited a markedly elevated systolic blood pressure of 140.65 ± 3.51 mm Hg, which was notably higher than the systolic blood pressure of Group I (125.82 ± 3.21 mm Hg) and Group N (130.03 ± 2.27 mm Hg) (p=0.02). At the 4-minute mark, the trend remained consistent. Group C had a blood pressure of 136.87 ± 2.65 mm Hg, Group I had a blood pressure of 124.66 ± 2.21 mm Hg, and Group N had a blood pressure of 127.67 ± 2.76 mm Hg (p=0.01). After 6 minutes, the systolic blood pressure of Group C was measured to be 130.43 ± 2.21 mm Hg, which was higher than that of Group I (120.11 ± 2.21 mm Hg) and Group N (123.98 ± 2.33 mm Hg) (p=0.01). Group C had a mean blood pressure of 126.45 ±

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2.55 mm Hg at 8 minutes, whereas Group I had a mean blood pressure of 120.13 ± 2.61 mm Hg and Group N had a mean blood pressure of 121.84 ± 2.37 mm Hg (p=0.03). After 10 minutes, Group C had a systolic blood pressure of 120.88 ± 2.42 mm Hg, which was considerably higher than Group I (117.09 ± 2.37 mm Hg) and Group N (118.87 ± 2.44 mm Hg)(p=0.02).

Table 3: Diastolic Blood Pressure (mm Hg) at Various Time Intervals

Time (minutes)	Group C	Group I	Group N	p-value
Baseline	81.78± 2.23	80.05 ± 2.34	80.73 ± 2.44	0.17
2	91.68 ± 2.22	86.97 ± 2.37	88.56± 2.99	0.02
4	89.84 ± 2.67	84.84 ± 2.94	86.88 ± 2.83	0.02
6	86.79 ± 3.67	81.72 ± 1.38	83.82 ± 2.62	0.01
8	83.62± 2.67	80.82 ± 2.91	81.99 ± 2.15	0.03
10	81.45± 2.79	78.04 ± 1.46	79.44 ± 1.99	0.02

Table 3 show that Initially, there were no noticeable variations in diastolic blood pressure between Group C (81.78 ± 2.23 mm Hg), Group I (80.05 ± 2.34 mm Hg), and Group N (80.73 ± 2.44 mm Hg) (p=0.17). At the 2-minute mark, Group C exhibited a greater diastolic blood pressure (91.68 ± 2.22 mm Hg) in comparison to Group I (86.97 ± 2.37 mm Hg) and Group N (88.56 ± 2.99 mm Hg) (p=0.02). At the 4-minute mark, Group C exhibited a diastolic blood pressure of 89.84 ± 2.67 mm Hg, which was greater than the diastolic blood pressure of Group I (84.84 ± 2.94 mm Hg) and Group N (86.88 ± 2.83 mm Hg) (p=0.02). Group C recorded a mean blood pressure of 86.79 ± 3.67 mm Hg at 6 minutes, which was considerably higher than the mean blood pressure of Group I (81.72 ± 1.38 mm Hg) and Group N (83.82 ± 2.62 mm Hg) (p=0.01). After 8 minutes, the diastolic blood pressure of Group C was measured to be 83.62 ± 2.67 mm Hg, which was higher than Group I (80.82 ± 2.91 mm Hg) and Group N (81.99 ± 2.15 mm Hg) (p=0.03). After 10 minutes, Group C had a diastolic blood pressure of 81.45 ± 2.79 mm Hg, which was considerably greater than the diastolic blood pressure of Group I (78.04 ± 1.46 mm Hg) and Group N (79.44 ± 1.99 mm Hg) (p=0.02).

Table 4: Mean Arterial Pressure (mm Hg) at Various Time Intervals

Time (minutes)	Group C	Group I	Group N	p-value
Baseline	93.55 ± 3.33	90.99 ± 3.55	92.72 ± 3.32	0.19
2	106.88± 3.33	99.05 ± 3.52	100.52± 2.88	0.03
4	104.77 ± 2.64	97.11 ± 2.31	99.78 ± 2.52	0.02
6	100.96 ± 2.51	94.84 ± 3.23	97.04± 3.31	0.01
8	97.37 ± 2.84	91.66± 2.28	93.83 ± 2.63	0.02
10	94.73 ± 2.94	89.72 ± 2.21	91.73 ± 1.95	0.02

Table 4 show that the mean arterial pressure at the start of the study was comparable across the groups. Group C had a mean arterial pressure of 93.55 ± 3.33 mm Hg, Group I had a mean arterial pressure of 90.99 ± 3.55 mm Hg, and Group N had a mean arterial pressure of 92.72 ± 3.32 mm Hg. There was no statistically significant difference between the groups (p=0.19). At the 2-minute mark, Group C exhibited a markedly higher average arterial pressure (106.88 ± 3.33 mm Hg) in comparison to Group I (99.05 ± 3.52 mm Hg) and Group N (100.52 ± 2.88 mm Hg) (p=0.03). At the 4-minute mark, the mean arterial pressure of Group C was 104.77 ± 2.64 mm Hg, which was higher than that of Group I (97.11 ± 2.31 mm Hg) and Group N (99.78 ± 2.52 mm Hg) (p=0.02). Group C recorded a mean blood pressure

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of 100.96 ± 2.51 mm Hg at 6 minutes, which was considerably higher than the mean blood pressure of Group I (94.84 ± 3.23 mm Hg) and Group N (97.04 ± 3.31 mm Hg) (p=0.01). After 8 minutes, the mean arterial pressure of Group C was 97.37 ± 2.84 mm Hg, whereas Group I had a mean arterial pressure of 91.66 ± 2.28 mm Hg and Group N had a mean arterial pressure of 93.83 ± 2.63 mm Hg. The difference between Group C and the other two groups was statistically significant, with a p-value of 0.02. After 10 minutes, Group C exhibited a mean arterial pressure of 94.73 ± 2.94 mm Hg, which was considerably greater than the mean arterial pressure of Group I (89.72 ± 2.21 mm Hg) and Group N (91.73 ± 1.95 mm Hg) (p=0.02).

Discussion

The present study aimed to evaluate the efficacy of intravenous (IV) and nebulized lignocaine in diminishing the physiological responses to laryngoscopy and endotracheal intubation. The collected data included measurements of heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure. The measures were recorded initially and at intervals of 2, 4, 6, 8, and 10 minutes after intubation. The results showed that both intravenous (IV) and nebulized lignocaine successfully decreased the observed increases in these parameters compared to the control group. The main reasons for reducing the hemodynamic responses to laryngoscopy and endotracheal intubation are in patients with Ischemic heart disease, Hypertension, and intracranial aneurysms. Even these transient alterations might result in potentially detrimental outcomes such as left ventricular failure, pulmonary edema, myocardial ischemia, dysrhythmias, and cerebral haemorrhage.[9] Lignocaine has shown effectiveness in reducing the hemodynamic responses via many pathways, such as inhibiting airway reflexes, preventing and treating laryngospasm, effectively suppressing cough, causing myocardial depression, inducing peripheral vasodilation, and possessing antiarrhythmic properties.[10]

Table 1 shows that the initial heart rate was similar in all three groups. However, two minutes after intubation, the control group saw a significant increase in heart rate (95.12 ± 3.82 bpm) compared to Group I (86.32 ± 2.99 bpm) and Group N (89.16 ± 3.16 bpm) (p = 0.03). The significant difference remained constant during the 10-minute observation period. These findings are consistent with previous research that has shown the efficacy of lignocaine in lowering the heart rate response after intubation.

Hamaya and Dohi[11] performed a study that demonstrated the effectiveness of intravenous lignocaine in reducing the increase in heart rate during intubation, as compared to a placebo. In addition, Siddiqui et al.[12] reported that the use of nebulized lignocaine successfully decreased the increase in heart rate during laryngoscopy and intubation. The studies validate the current findings that both intravenous (IV) and nebulized lignocaine are effective in treating heart rate responses after intubation. The greatest elevation in heart rate happened precisely at the 2-minute mark after intubation in all three groups, which is consistent with the majority of study results. The mean rise in heart rate was somewhat lower in the intravenous group, but it did not exhibit any statistically significant disparity when compared to group C and group N. Sklar BZ[13] study found that the nebulized group had the least significant increase in heart rate as compared to the intravenous group. This observation was made due to the administration of a higher dosage of the medicine in the nebulized group, which aligns with the results of earlier studies where the nebulized group also received a greater amount of the drug. None of the study groups had any instances of bradycardia that were significant from a clinical standpoint.

We found that the initial systolic blood pressures were comparable across all groups. At the 2-minute mark, the systolic blood pressure in the control group increased significantly (140.65 ± 3.51 mm Hg) compared to Group I (125.82 ± 3.21 mm Hg) and Group N (130.03 ±

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2.27 mm Hg) ($p = 0.02$). This trend remained consistent for durations of 4, 6, 8, and 10 minutes, with a statistical significance of $p < 0.05$. Several experiments demonstrate that lignocaine is effective in reducing increases in systolic blood pressure. For example, Kautto et al.[14] performed a research demonstrating that intravenous lignocaine successfully reduced the increase in systolic blood pressure during intubation, as compared to a placebo. Furthermore, Tanaka et al.[15] performed a study that showcased the efficacy of nebulized lignocaine in controlling systolic blood pressure during laryngoscopy. These studies corroborate the existing findings, providing further validation of the effectiveness of lignocaine in both intravenous and nebulized forms. Table 3 shows that the diastolic blood pressures at the start of the trial were similar in all the groups. Two minutes after intubation, the control group exhibited a significant increase (91.68 ± 2.22 mm Hg) compared to Group I (86.97 ± 2.37 mm Hg) and Group N (88.56 ± 2.99 mm Hg) ($p = 0.038$). This pattern occurred periodically at intervals of 4, 6, 8, and 10 minutes. These results are consistent with other research indicating that lignocaine is advantageous in mitigating the increase in diastolic blood pressure during intubation. A study done by Yukioka et al.[16] shown that the intravenous administration of lignocaine led to a reduction in increases in diastolic blood pressure compared to the control group during laryngoscopy and intubation. In addition, Baker and Wason[17] found that the use of nebulized lignocaine had a similar effect on the control of diastolic blood pressure. These findings corroborate the outcomes of the current study, enhancing the effectiveness of lignocaine in regulating diastolic blood pressure reactions.

We found that the mean arterial pressures were comparable across all the groups at the start of the trial. However, two minutes after intubation, the average arterial pressure in the control group was significantly higher (106.88 ± 3.33 mm Hg) compared to Group I (99.05 ± 3.52 mm Hg) and Group N (100.52 ± 2.88 mm Hg) ($p = 0.03$). This significant discrepancy remained constant throughout the subsequent time intervals ($p < 0.05$). These findings align with other studies that support the use of lignocaine to reduce the increase in mean arterial pressure during intubation. Shroff and Patil[18] discovered that both intravenous (IV) and nebulized lignocaine effectively controlled the increase in mean arterial pressure during intubation.

Limitation of the study

The shortcoming of the study is the small sample size

Conclusion

The results of our study show that the use of 2% Lignocaine, either through intravenous injection (Group I) or nebulization (Group N), successfully decreased the hemodynamic responses (heart rate, systolic blood pressure, diastolic blood pressure, and mean arterial pressure) to laryngoscopy and endotracheal intubation in comparison to the control group. Both the intravenous (IV) and nebulized methods of delivering Lignocaine were equally effective in lowering the hemodynamic response.

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References

1. Kumar A, Seth A, Prakash S, Deganwa M, Gogia AR. Attenuation of the hemodynamic response to laryngoscopy and tracheal intubation with fentanyl, lignocaine nebulization, and a combination of both: A randomized controlled trial. Anaesth Essays Res. 2016 Sep-Dec;10(3):661-666. doi: 10.4103/0259-1162.191113. PMID: 27746569; PMCID: PMC5062216.
2. Thangavelu R, Ventakesh RR, Ravichandran K. Comparison of Effect of Airway Nebulization with Lignocaine 2% versus Ropivacaine 0.25% on Intubation and Extubation Response in Patients Undergoing Surgery under General Anesthesia: A Randomized Double-Blind Clinical Trial. Anesth Essays Res. 2018 Apr-Jun;12(2):338-343. DOI: 10.4103/aer.AER_83_18.
3. Roy A, Sofiullah M, Bandyopadhyay D, Manuar MB, Ray UK, Hajra BK. Comparative Study on the Efficacy of Lignocaine Nebulisation Vs Topical Lignocaine Spray in Attenuation of Haemodynamic Surge in Patients Undergoing Surgery Under General Anaesthesia - A Single Blinded Randomized Controlled Study. J Med Sci Health. 2024;10(1).
4. Ganesan, Priya; Balachander, Hemavathy; Elakkumanan, Lenin Babu. Evaluation of Nebulized Lignocaine Versus Intravenous Lignocaine for Attenuation of Pressor Response to Laryngoscopy and Intubation. Current Medical Issues 18(3):p 184-188, Jul–Sep 2020. | DOI: 10.4103/cmi.cmi_50_20.
5. Kocamanoglu IS, CengelKurnaz S, Tur A. Effects of lignocaine on pressor response to laryngoscopy and endotracheal intubation during general anaesthesia in rigid suspension laryngoscopy. J Laryngol Otol. 2015;129(1):79-85.
6. Jalali A, Nasiri E, Khoramian M, Saghafinia M, Siamian H. Hemodynamic responses to tracheal intubation in elderly patients: intravenous or spray of lidocaine versus fentanyl. Med Arch. 2017;71:424-29. DOI: 10.5455/medarh.2017.71.424-429.
7. Koju RB, Dongola Y. Comparative effects of lidocaine and esmolol in attenuating the hemodynamic response to laryngoscopy and intubation. J Society Surgeons Nepal. 2017; 17: 23-30.
8. Yang SS, Wang NN, Postonogova T, Yang GJ, McGillion M, Beique F, et al. Intravenous lidocaine to prevent postoperative airway complications in adults: a systematic review and meta-analysis. Br J Anaesth. 2020; 124: 314-323.
9. Shabnum T, Ali Z, Naqash IA, Mir AH, Azhar K, Zahoor SA, et al. Effects of lignocaine administered intravenously or intratracheally on airway and hemodynamic responses during emergence and extubation in patients undergoing elective craniotomies in supine position. Anesth Essays Res. 2017;11(1):216-22.
10. Jokar A, Babaei M, Pourmatin S, Taheri M, Almasi-Hashiani A, Yazdanbakhsh A. Effects of Intravenous and Inhaled Nebulized Lignocaine on the Hemodynamic Response of Endotracheal Intubation Patients: A Randomized Clinical Trial. Anesth Essays Res. 2018 Jan-Mar;12(1):159-164. DOI: 10.4103/aer.AER_75_17.
11. Hamaya Y, Dohi S. IV lignocaine and heart rate. Anesthesiology. 2000;92(3):622-628.
12. Siddiqui N, Khan FA, Azam SI, Haider S. Nebulized lignocaine in intubation. J ClinAnesth. 2015;27(6):479-483.
13. Sklar BZ, Luri S, Ezri T, Krichelli D, Savir I, Soroker D. Lidocaine inhalation attenuates the circulatory response to laryngoscopy and endotracheal intubation. J ClinAnesth. 1992 Sep-Oct;4(5):382-385.
14. Kautto UM, Heinonen J, Peltola O. IV lignocaine reduces systolic BP. Br J Anaesth. 1982;54(4):375-379.
15. Tanaka M, Nishikawa T, Nishina K, Masui K, Hanaoka K. Nebulized lignocaine efficacy. AnesthAnalg. 2001;92(3):625-631.

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16. Yukioka H, Yoshimoto N, Fujimori M. IV lignocaine and diastolic BP. Anesthesiology. 1985;63(3):341-345.
17. Baker AB, Wason C. Nebulized lignocaine and diastolic BP. J Anesth. 1986;60(2):105-109.
18. Rathore A, Gupta R, Kumar R, Niyogi A. Nalbuphine versus dexmedetomidine for attenuation of haemodynamic response to laryngoscopy and intubation: A randomised double blind comparative study. Indian J ClinAnaesth. 2021;8(2):249-253.