

Effect of Nebulised Lidocaine on Haemodynamic Responses during Nasotracheal IntubationKhyati Makwana¹, Pooja Fumakiya², Jagdishbhai Mer³¹Assistant Professor, Department of Anaesthesia, Gujarat Adani Institute of Medical Science, Bhuj, Kutch, Gujarat²Assistant Professor, Department of Anaesthesia, Gujarat Adani Institute of Medical Science, Bhuj, Kutch, Gujarat³Assistant Professor, Department of Anaesthesia, Shantabaa Medical College and General Hospital, Amreli, Gujarat

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Corresponding author: Dr Jagdishbhai Mer

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Abstract:

Background and Aim: A common side effect of laryngoscopy and nasotracheal intubation is hypertension and tachycardia, which are brought on by temporary sympatho-adrenal activation. In this work, we compared the experimental group to the control group to examine the effects of nebulized lidocaine on the haemodynamic stress responses during nasotracheal intubation. This study's main goal was to compare the differences in mean arterial pressure between two groups following nasotracheal intubation.

Material and Methods: The current prospective comparison study was carried out over the course of a year in a tertiary healthcare facility. The study comprised 120 patients scheduled for head and neck surgery who were classified as physiologic status Classes I–II by the American Society of Anaesthesiologists. 5 ml of ordinary saline were nebulized to Group A (the control group). Lidocaine 4% solution in 5ml was nebulized for Group B. Before and after nebulization, just before intubation, immediately after intubation, and at 3, 5, and 10 minutes after intubation, heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), and SpO₂ were measured.

Results: There was a substantial increase in both groups when SBP, DBP, MAP, and heart rate were compared to pre-intubation values, although the increase in the control group was greater than it was in the lidocaine group (p 0.05). After that, MAP, SBP, DBP, and HR all gradually fell until the study's 10-minute end.

Conclusion: Nebulized 4% inhalation given before to induction reduces the sympathetic activation and cardiovascular response brought on by nasotracheal intubation.

Keywords: Heart rate, Lidocaine, Nasotracheal Intubation, Systolic Blood Pressure.

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Introduction

Securing the patient's airway so that they receive enough ventilation while under general anaesthetic is the primary duty of an anesthesiologist. One of the most popular techniques for administering anaesthesia during head and neck procedures is nasotracheal intubation (NTI). The tracheal tube must enter through the nose during NTI, which improves isolation and provides adequate surgical access for intraoral treatments. King first characterised the considerable reflex circulatory alterations brought on by endotracheal intubation and laryngoscopy in 1951.[1] A laryngoscope pressing on the tongue's base and raising the epiglottis causes these alterations to occur. Although a response like this would probably be well tolerated by healthy patients, in people with severe coronary artery or cerebrovascular disorders, these alterations may be linked to

myocardial ischemia and brain haemorrhage.[2] In comparison to orotracheal intubation, nasotracheal intubation causes a more severe and long-lasting hypertensive reaction.[3] Because the hemodynamic response is brief in duration, it could not have much therapeutic relevance in otherwise healthy patients.[4] The laryngoscopic reaction may, however, put patients with low myocardial reserve, elevated intracranial pressures (ICP), or elevated intraocular pressures (IOP) at risk for developing pulmonary edoema, ciency, and cerebrovascular-myocardial insuf ities.[5-7] Therefore, it's important to reduce these negative laryngoscopic reactions. Laryngoscopy and intubation stress response attenuation techniques have been used both pharmacologically and nonpharmacologically. Inhalational anaesthetic drugs, powerful opioids like

fentanyl, and substantial pre-medication are a few procedures that are frequently utilised to increase the degree of anaesthesia.[8-10] Others include calcium channel blockers, magnesium sulphate, lidocaine, both topical and intravenously (IV).[11-14] In order to reduce the stress reaction, topical anaesthetic with lignocaine in the form of viscous gargles, lignocaine aerosols, or oropharyngeal sprays continues to be widely used. Laryngospasm, cough during extubation, and cough during tracheal intubation have all been treated with intravenous lignocaine. Additionally, it has been utilised to lessen bronchoconstriction and decrease airway hyperactivity. In comparison to other types of lignocaine, intravenous lignocaine is shown to be a more effective choice to reduce the stress response due to its well-established centrally depressive and anti-arrhythmic effects.[15-18] In this work, we compared the experimental group to the control group to examine the effects of nebulized lidocaine on the haemodynamic stress responses during nasotracheal intubation. This study's main goal was to compare the differences in mean arterial pressure between two groups following nasotracheal intubation.

Material and Methods

The current prospective comparison study was carried out over the course of a year in a tertiary healthcare facility. In the randomised control double-blind research, 120 patients between the ages of 18 and 65 with physiologic status Classes I to II according to the American Society of Anaesthesiologists were slated for head and neck surgery. Exclusion criteria for the trial included patients with hypertension, allergies to local anaesthetics, convulsion histories, pregnancy, predicted difficult intubation, patients needing a second attempt at intubation, high risk of aspiration, and patients with a recent history of URTI.

A computer-generated random number was used to place each patient into one of the two groups. Alprazolam 0.5mg tablets were given to patients the night before surgery as a premedication. Following the patient's transportation to the operating room, blood pressure, heart rate, oxygen saturation, and ECG were all continually monitored. An 18G cannula was used to ensure intravenous (IV) access, and baseline readings of SBP, DBP, and HR were taken. Glycopyrrolate 0.2 mg intravenously was administered to all patients, along with 0.1% oxymetazoline nasal drops, in both nasal passages. Following that, each patient received a random dose of the study medicine administered in a sequence determined by a computer. Five cc of sterile saline were nebulized to the individuals in Group A (the control group). Lidocaine 4% (40 mg/ml) solution was nebulized onto the patients in Group B in a volume of 5ml (200 mg). Nebulizing was used to provide the medication as an aerosol. A face mask

attached with a nebulizer was used to nebulize a gas through a 200 cm tubing connected from the oxygen port to the nebulizer. Up until the entire solution in the nebulizer became aerosolized, nebulization was continued.

All of the patients received 2 micrograms/kg of fentanyl after nebulization was finished prior to induction. Before the onset of anaesthesia and the continuation of the maintenance infusion, all patients received 10 ml/kg of Ringer lactate solution over a 10-min interval. After 3 minutes of preoxygenation with 100% oxygen, all patients were then induced with injections of propofol 1.5–2.5 mg/kg until verbal response was lost, followed by vecuronium 0.1 mg/kg, a muscle relaxant. Patients were intubated nasally with the appropriate size endotracheal tube by direct laryngoscopy technique utilising Macintosh Blade after 3 min of using the bag and mask and ventilation with 100% O₂ at 6 l/min. O₂:N₂O (1:1) and 2% sevoflurane were used to maintain anaesthesia. The respiratory rate was set at 12/min, the inspiration/expiration ratio was set at 1:2, and the tidal volume was 6 ml/kg. Prior to induction (baseline), right before intubation, immediately after intubation, and at 3, 5, and 10 min. after intubation, all parameters were assessed.

Statistical analysis

The collected data was organised, inputted, and exported to the data editor page of SPSS version 15 (SPSS Inc., Chicago, Illinois, USA) after being combined and entered into a spreadsheet programme (Microsoft Excel 2007). The level of significance and confidence level for each test were set at 5% and 95%, respectively.

Results

Between the two groups, the demographic profile was comparable (Table 1). Both groups saw a substantial increase in SBP, DBP, MAP, and HR when compared to pre-intubation readings, however the increase in the control group was greater than it was in the lidocaine group (p 0.05). In the lidocaine group, MAP increased by 11% while it increased by more than 21% in the control group. The increase in SBP was 24% for the control group and 15% for the lidocaine group, respectively. The increase in DBP in the control group and the lidocaine group, respectively, was 24% and 9%. In the control group, HR was raised to 19%, whereas in the lidocaine group, it was raised to 4%. MAP, SBP, DBP, and HR then gradually fell in both groups at 3, 5 and 10 minutes. From the moment of intubation to the end of the study, the MAP and SBP in the lidocaine group were both considerably lower than those in the control group in the inter-group comparison (p 0.05). Before intubation, immediately after intubation, and three minutes later, DBP was considerably lower in the lidocaine group. The heart rate in the group receiving lido-

caine was considerably lower both immediately and three minutes after intubation.

Table 1: Demographic characteristics of patients

Variables	Group A	Group B	P value
Male (n, %)	10 (16.6)	12 (20)	0.47
Female (n, %)	50 (83.3)	48 (80)	
Age in years (Mean \pm SD)	45.2 \pm 7.2	51.4 \pm 09.14	0.1
BMI (Mean \pm SD)	24.9 \pm 2.2	26.8 \pm 4.3	0.2
ASA I (n, %)	39 (65)	37 (65)	0.64
ASA II (n, %)	21 (35)	23 (37.3)	

Statistically significance at $p \leq 0.05$

Table 2: Mean difference of changes in mean arterial blood pressure (MAP) between two group

Variables	Group A (mean \pm SD)	Group B (mean \pm SD)	P value
Baseline	93.2 \pm 4.8	94.1 \pm 4.6	0.47
Just before intubation	82.1 \pm 4.3	78.1 \pm 5.2	
Immediately after intubation	102.3 \pm 3.4	87.5 \pm 3.2	0.1
At 3 min	92.8 \pm 5.4	82.5 \pm 4.2	0.2
At 5 min	88.9 \pm 6.9	84.5 \pm 5.3	0.64
At 10 min	86.9 \pm 5.4	82.7 \pm 5.1	

Statistically significance at $p \leq 0.05$

Discussion

The safest method for managing patients with problematic airways would be awake intubation, preferably by flexible fibre optic bronchoscopy, which is the industry standard for handling difficult airways while under topical anaesthesia.

The most popular medication for reducing the hemodynamic reactions to laryngoscopy and tracheal intubation is local anaesthetic, particularly lignocaine. It has been proven effective to utilise lignocaine to reduce the hemodynamic reactions following intubation. Gianelly et al.[19] came to the conclusion that the dose administered was directly correlated with the levels of lignocaine in the blood after intravenous delivery. They also came to the conclusion that blood levels as high as 9 g/l may cause serious adverse effects and that an effective safe blood level of 2 to 5 g/ml is attained by intravenous bolus of 1 to 2 mg/kg. According to Adriani [20], the pulmonary alveoli quickly absorb topical anaesthetics that are administered to the larynx and trachea.

Pre-intubation MAP, SBP, and DBP were shown to be considerably lower in the lidocaine group than in the control group in the current study. It could be as a result of lidocaine's systemic absorption and subsequent impact on the peripheral nervous system. Although lidocaine has a biphasic action on the peripheral vascular system. It has a vasoconstricting impact at low concentrations and a vasodilating effect at high concentrations. In addition, Weinberg L. et al. found that intravenous lidocaine 1.5 mg/kg loading dosage followed by an infusion of 1.5 mg/hr decreases the need for volatile anaesthesia and lowers blood pressure and heart rate in patients undergoing open radical prostatectomy.[21]

In our work, we employed the amide local anaesthetic lidocaine 4% in nebulization form to reduce the haemodynamic response to nasotracheal intubation. To eliminate the stress reaction during laryngoscopy and intubation during orotracheal intubation, lidocaine, an inexpensive and widely accessible medication, has been utilised in spray, intravenous, and nebulized forms.[22,23] However, there are few research examining how nebulized lidocaine affects haemodynamic reactions during nasotracheal intubation. Comparing nebulized lidocaine to intravenous lidocaine, Gupta A et al. and Gansesan P et al. found that nebulised lidocaine was better able to reduce haemodynamic reactions.[24,25] It was found in the current study that the rise in MAP, SBP, DBP, and HR immediately following intubation was less in the lidocaine group than in the control group. Similar results were obtained by Lee S Y et al., who discovered that MAP and HR were considerably higher in the control group than in the lidocaine group after orotracheal intubation, when 10% lidocaine was sprayed on the laryngoscope blade or trachea.[11] To measure the stress response of laryngoscopy and orotracheal intubation, Venus B et al. conducted a study in which topical anaesthetic of the oropharynx with lidocaine aerosol was administered. They reported that an increase in Mean BP, SBP, and HR was much smaller than that of their control group.[26] Additionally, Patil V. et al. noted that 4% lidocaine nebulization was efficient in reducing the hemodynamic reaction to oral intubation and direct laryngoscopy.[27] They examined 2% and 4% lidocaine in nebulization in their study. Similar results were found by Kumar A et al, who found that 4% lidocaine nebulized with fentanyl was more successful at reducing haemodynamic reactions to intubation than fentanyl or nebulization alone.[28] At all post-

intubation periods in our investigation, MAP was significantly lower in the lidocaine group compared to the control group. ($p < 0.05$) Ahmed M. et al.[29] utilized a normal nebulizer with a full face mask connected and Lidocaine 2% (2 mg/kg) in 5 ml saline. The patient was instructed to inhale the local anaesthetic vapour deeply for 15 minutes. In contrast to the control group, neither the number of patients in grade 0 nor the number of patients in grades 1 or 2 in the trial group showed an increase in endotracheal tube tolerance. Using awake fibre optic intubation, Hawkyard et al. investigated the hypertensive response to laryngoscopy and endotracheal intubation.[30] They carried out a study in which measures of blood pressure and heart rate were taken from 35 patients undergoing endotracheal intubation under general anaesthesia and 35 patients undergoing awake fibre optic intubation under local anaesthesia. As a result of intubation, Group A's mean arterial pressure increased on average by 35 mmHg, according to the results, whereas Group B's mean arterial pressure dropped on average by 9 mmHg.

Our study's use of 5 ml of 4% lidocaine, which is considerably less than the dosage that would be advised given the weight range we employed in our investigation, constitutes a study limitation.

Conclusion

Nebulized 4% lidocaine is administered prior to induction to reduce the sympathetic stimulation and cardiovascular response brought on by nasotracheal intubation. Large-scale studies are needed, nonetheless, to prove that 4% lignocaine is a superior topical anaesthetic for nasotracheal intubation.

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