Original Article

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Assessment of Effect of Nebulised Lidocaine on Haemodynamic Responses during Nasotracheal Intubation

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Abstract

Background: Aim: To assess the effect of nebulised lidocaine on haemodynamic responses during nasotracheal intubation. **Subjects and Methods:** Eighty- four patients of American Society of Anaesthesiologists' classes I- II age ranged 18-65 years planned for head and neck surgery were randomized into 2 groups based on lottery method. Group I patients were nebulised with 5 ml of normal saline. Group II patients were nebulised with 5 ml (200 mg) of lidocaine 4% (40 mg/ml) solution. Parameter such as systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial blood pressure (MAP) were observed before and after nebulisation, just before intubation, immediately after intubation and at 3, 5 10 min after intubation. **Results:** The mean BMI was 25.8 Kg/m2 in group I and 26.2 Kg/m2 in group II. ASA grade I was seen in 32 in group I and 30 in group II and ASA II in 9 in group I and 11 in group II. The mean duration of nasotracheal intubation was 57.2 seconds in group I and 59.4 seconds in group II. The mean SBP (mm Hg) and DBP in both groups were significant statistically ($P< 0.05$). The mean MAP (mm Hg) at baseline was 95.7 in group I and 92.1 in group II, just before intubation was 82.3 in group I and 76.4 in group II, immediately after intubation was 101.9 in group I and 86.2 in group II, at 3 minutes was 93.2 in group I and 81.7 in group II, at 5 minutes was 89.5 in group I and 84.2 in group II and at 10 minutes was 88.2 in group I and 83.0 in group II. A significant difference was observed ($P< 0.05$). **Conclusion:** Administration of nebulised 4% lidocaine inhalation before induction attenuates cardiovascular response and sympathetic stimulation that occurs due to nasotracheal intubation.

Keywords: nasotracheal intubation, lidocaine, haemodynamic responses.

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Introduction

Nasotracheal intubation (NTI) is one of the commonest methods used to provide anaesthesia for surgeries on the head and neck region. NTI involves the tracheal tube passing through the nose hence allowing better isolation and good surgical access for intraoral procedures.[1] Despite adequate anesthetic depth, direct laryngoscopy and intubation which are essential steps in general anesthesia needing endotracheal intubation elicit a significant adreno-sympathetic response.[2] This response, as manifested by increased systolic blood pressure (SBP), diastolic BP (DBP), mean blood pressure (BP), heart rate (HR), arrhythmias, etc., would result in unacceptable morbidity and mortality in patients with compromised and borderline cardiac reserve.[3] This has necessitated the use of various adjuvants such as opioids, beta-blockers, nitroglycerine, nitroprusside, lignocaine, and magnesium sulfate, with varying degrees of effectiveness.[4] Nebulised lidocaine has been used in clinical practice for a variety of indications. Nebulized lignocaine (NL), a novel method to prevent this response, has rekindled interest in the recent past. Aerosol anesthesia is a method that has been rather widely used for bronchoscopy and bronchography.[5] It has been tried in patients with bronchial asthma to decrease airway reactivity. Also, it has been found in various studies that the use of nebulised lidocaine preoperatively decreases the haemodynamic responses during laryngoscopy and orotracheal intubation.[6] Considering this, we planned present study to assess the effect of nebulised lidocaine on haemodynamic responses during nasotracheal intubation.

Subjects and Methods

After considering the utility of the study and obtaining approval from ethical review committee of the institute, we selected eighty- four patients of American Society of Anaesthesiologists' physiologic status classes I- II age ranged 18-65 years planned for head and neck surgery of either gender.

Demographic data of each patient was recorded. All patients were given injection glycopyrrolate 0.2mg iv and 0.1% oxymetazoline nasal drops were instilled in both the nasal passage. Patients were randomized into 2 groups based on lottery method. Group I patients were nebulised with 5 ml of normal saline. Group II patients were nebulised with 5ml

(200 mg) of lidocaine 4% (40 mg/ml) solution. Parameter such as systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean arterial blood pressure (MAP) were observed before and after nebulisation, just before intubation, immediately after intubation and at 3, 5 10 min after intubation. The results were compiled and subjected for statistical analysis using Mann Whitney U test. P value less than 0.05 was set significant.

Results

Table 1: Patients distribution

Groups	Group I	Group II
Drug	5 ml of normal saline	5ml (200 mg) of lidocaine 4% (40 mg/ml) solution
M:F	25:16	20:21

Group I comprised of 25 male and 16 female and group B had 20 male and 21 female [Table 1]

Table 2: Baseline characteristics

Parameters	Group I	Group II	P value
BMI (Kg/m2)	25.8	26.2	0.94
ASA I	32	30	0.01
ASA II	9	11	
Duration of (nasotracheal intubation)	57.2	59.4	0.72

The mean BMI was 25.8 Kg/m2 in group I and 26.2 Kg/m2 in group II. ASA grade I was seen in 32 in group I and 30 in group II and ASA II in 9 in group I and 11 in group II. The mean duration of nasotracheal intubation was 57.2 seconds in group I and 59.4 seconds in group II. The difference was significant (P< 0.05) [Table 2].

Table 3: Baseline characteristics

SBP (mm Hg)	Group I	Group II	P value
Baseline	126.2	123.2	0.05
Just before intubation	107.5	100.8	0.04
Immediately after intubation	132.4	118.4	0.001
At 3 minutes	127.4	113.2	0.02
At 5 minutes	121.0	112.0	0.01
At 10 minutes	119.5	100.8	0.03

The mean SBP (mm Hg) at baseline was 126.2 in group I and 123.2 in group II, just before intubation was 107.5 in group I and 100.8 in group II, immediately after intubation was 132.4 in group I and 118.4 in group II, at 3 minutes was 127.4 in group I and 113.2 in group II, at 5 minutes was 121.0 in group I and 112.0 in group II and at 10 minutes was 119.5 in group I and 100.8 in group II. A significant difference was observed (P< 0.05) [Table 3].

Table 4: Comparison of diastolic blood pressure (DBP)

DBP (mm Hg)	Group I	Group II	P value
Baseline	76.4	76.2	0.92
Just before intubation	68.4	65.3	0.05
Immediately after intubation	86.2	70.5	0.001
At 3 minutes	74.2	65.2	0.03
At 5 minutes	70.4	68.9	0.81
At 10 minutes	68.5	67.4	0.90

The mean DBP (mm Hg) at baseline was 76.4 in group I and 76.2 in group II, just before intubation was 68.4 in group I and 65.3 in group II, immediately after intubation was 86.2 in group I and 70.5 in group II, at 3 minutes was 74.2 in group I and 65.2 in group II, at 5 minutes was 70.4 in group I and 68.9 in group II and at 10 minutes was 68.5 in group I and 67.4 in group II. A significant difference was observed (P< 0.05) [Table 4].

Table 5: Comparison of mean arterial blood pressure (MAP)

MAP (mm Hg)	Group I	Group II	P value
Baseline	95.7	92.1	0.03
Just before intubation	82.3	76.4	0.01
Immediately after intubation	101.9	86.2	0.01
At 3 minutes	93.2	81.7	0.02
At 5 minutes	89.5	84.2	0.05
At 10 minutes	88.2	83.0	0.05

The mean MAP (mm Hg) at baseline was 95.7 in group I and 92.1 in group II, just before intubation was 82.3 in group I and 76.4 in group II, immediately after intubation was 101.9 in group I and 86.2 in group II, at 3 minutes was 93.2 in group I and 81.7 in group II, at 5 minutes was 89.5 in group I and 84.2 in group II and at 10 minutes was 88.2 in group I and 83.0 in group II. A significant difference was observed (P< 0.05) [Table 5].

Discussion

Laryngoscopy and tracheal intubation provoke a significant sympathoadrenal response. In patients who are at risk for developing arterial hypertension or myocardial ischemia, such changes may be harmful.[7] The exact mechanism of this sympathoadrenal response is unclear but perhaps due to intense stimulation of the upper respiratory tract. Suppressing a hypertensive response to intubation is one of the important prerequisites for a properly administered general anesthesia.[8] Drugs such as inhalational agents, narcotics, β-blockers, α-blockers, calcium channel blockers, and vasodilators have been used to suppress these responses. Intubation in a deeper plane also alleviates this response.[9] Unfortunately, they can be ineffective or have adverse effects including bradycardia and hypotension. In our study, we used lignocaine, an amide local anesthetic to blunt the hemodynamic response during laryngoscopy and intubation. Lignocaine has been used in various modalities such as IV, topical, instillation, or inhalation.[10] We assessed the effect of nebulised lidocaine on haemodynamic responses during nasotracheal intubation.

Our results showed that Group I comprised of 25 male and 16 female and group B had 20 male and 21 female. Verma et al.[11] compared 94 patients who underwent head and neck surgery under general anaesthesia requiring nasotracheal intubation were randomised into two groups. Group A (control group) was nebulised with 5 ml of normal saline. Group B was nebulised with 5 ml of lidocaine 4% solution. Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), and SpO2 were observed before and after nebulisation, just before intubation, immediately after intubation and at 3, 5 10 min after intubation. On comparing SBP, DBP, MAP and

heart rate immediately after intubation with pre-intubation values, there was a significant surge in both groups but an increase in the control group was more in comparison to the lidocaine group.

Our results revealed that the mean BMI was 25.8 Kg/m2 in group I and 26.2 Kg/m2 in group II. ASA grade I was seen in 32 in group I and 30 in group II and ASA II in 9 in group I and 11 in group II. The mean duration of nasotracheal intubation was 57.2 seconds in group I and 59.4 seconds in group II. Ganesan et al.[12] compared the efficacy of nebulized lidocaine versus intravenous (IV) lidocaine in suppressing the pressor response to laryngoscopy and intubation. 100 patients within the age group of 18–65 years undergoing elective surgery under general anesthesia were randomly allocated into two groups: group IV lignocaine (IVL) (n = 50) and group nebulized lignocaine (NL) (n = 50). Baseline values of heart rate (HR), systolic blood pressure (BP), diastolic BP, mean arterial pressure (MAP), and saturation were noted. Patients received either nebulized or IV lidocaine according to the group. HR, saturation, BP, MAP, and arrhythmias were noted every minute from laryngoscopy up to 5 min postlaryngoscopy and intubation. There was an increase in HR and BP from baseline in both the groups with laryngoscopy and intubation, and the increase is significantly less in NL ($P < 0.05$). The parameters in both the groups attained the baseline values at the 3rd min post intubation. However, the 4th- and 5th-min readings showed values below baseline in the nebulization group

We found that the mean SBP (mm Hg) at baseline was 126.2 in group I and 123.2 in group II, just before intubation was 107.5 in group I and 100.8 in group II, immediately after intubation was 132.4 in group I and 118.4 in group II, at 3 minutes was 127.4 in group I and 113.2 in group II, at 5 minutes was 121.0 in group I and 112.0 in group II and at 10 minutes was 119.5 in group I and 100.8 in group II. We observed that the mean DBP (mm Hg) at baseline was 76.4 in group I and 76.2 in group II, just before intubation was 68.4 in group I and 65.3 in group II, immediately after intubation was 86.2 in group I and 70.5 in group II, at 3 minutes was 74.2 in group I and 65.2 in group II, at 5 minutes was 70.4 in group I and 68.9 in group II and at 10 minutes was 68.5 in group I and 67.4 in group II. Lee S Y et al.[13] found that MAP and HR at 2.5 and 5 min after orotracheal intubation were significantly higher in the control group than in the lidocaine group where 10% lidocaine was sprayed on a laryngoscope blade or trachea.

The mean MAP (mm Hg) at baseline was 95.7 in group I and 92.1 in group II, just before intubation was 82.3 in group I and 76.4 in group II, immediately after intubation was 101.9 in group I and 86.2 in group II, at 3 minutes was 93.2 in group I and 81.7 in group II, at 5 minutes was 89.5 in group I and 84.2 in group II and at 10 minutes was 88.2 in group I and 83.0 in group II. Venus B et al.[14] conducted a study in which topical anaesthesia of the oropharynx with lidocaine aerosol was given to assess the stress response of laryngoscopy and orotracheal intubation and they reported that a rise in mean BP, SBP and HR was significantly less than that of their control group.

Conclusion

Administration of nebulised 4% lidocaine inhalation before induction attenuates cardiovascular response and sympathetic stimulation that occurs due to nasotracheal intubation.

References

1. Ahmed A, Sen S, Das T, Dasgupta A. Reflex circulatory responses after three stages of nasotracheal intubation and two stages of orotracheal intubation: A comparative study. Asian J Med Sci. 2017;8(5):41–7.
2. Splinter WM, Cervenko F. Haemodynamic responses to laryngoscopy and tracheal intubation in geriatric patients: effects of fentanyl, lidocaine and thiopentone. Can J Anaesth. 1989;36:370–6.
3. Sklar BZ, Lurie S, Ezri T, Krichelli D, Savir I, Soroker D. Lidocaine inhalation attenuates the circulatory response to laryngoscopy and endotracheal intubation. J Clin Anesth. 1992;4(5):382–5.
4. Hoda A, Khan F. Effect of one minimum alveolar concentration sevoflurane with and without fentanyl on hemodynamic response to laryngoscopy and tracheal intubation. J Anaesthesiol Clin Pharmacol. 2011;27(4):522–6.
5. Altintas F, Bozkurt P, Kaya G, Akkan G. Lidocaine 10% in the endotracheal tube cuff: Blood concentrations, haemodynamic and clinical effects. Eur J Anaesthesiol. 2000;17(7):436–42.
6. Gupta A, Anand R. Comparative study of nebulised lignocaine with intravenous lignocaine on stress response to endotracheal intubation. Int J Anesthesiol. 2018;8(2):84–7.
7. Ganesan P, Balach H, Elakkumanan L. Evaluation of nebulised lidocaine versus intravenous lidocaine for attenuation of pressor response to laryngoscopy and intubation. Curr Med Issues. 2020;18(3):184–8.
8. Patil V, Barker GL, Harwood RJ, Woodal NM. Training course in local anaesthesiaof the airway and fibreoptic intubation using course delegates as subjects. Br J Anaesth. 2002;89(4):586–93.
9. Kumar A, Seth A, Prakash S, Deganwa M, Gogia A. Attenuation of the hemodynamic response to laryngoscopy and tracheal intubation with fentanyl, lignocaine nebulization, and a combination of both: A randomized controlled trial. Anesth Essays Res. 2016;10(3):661–6.
10. Parkes SB, Butler CS, Muller R. Plasma lignocaine concentration following nebulization for awake intubation. Anaesth Intensive Care. 1997;25(4):369–71.
11. Verma R, Gaurav S, Verma H, Verma S, Kuswaha B, Chaudhary AK, Singh D. A prospective study evaluating the effect of nebulised lidocaine on haemodynamic responses during nasotracheal intubation. Indian J Clin Anaesth 2022;9(4):439-444.
12. Ganesan P, Balachander H, Elakkumanan LB. Evaluation of nebulized lignocaine versus intravenous lignocaine for attenuation of pressor response to laryngoscopy and intubation. Curr Med Issues 2020;18:184-8.
13. Lee SY, Min JJ, Kim HJ, Hong DM, Kim HJ, Park HP. Hemodynamic effects of topical lidocaine on the laryngoscope blade and trachea during endotracheal intubation: a prospective, doubleblind, randomized study. J Anesth. 2014;28(5):668–75.
14. Venus B, Polassani V, Pham CG. Effects of aerosolized lidocaine on circulatory responses to laryngoscopy and tracheal intubation. Crit Care Med. 1984;12(4):391–4.

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