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Comparison of sublingual nitroglycerine spray with oropharyngeal lignocaine spray for blunting response to laryngoscopy and intubation

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Abstract

Background: Endotracheal intubation is a common mode of securing the airway for administering general anaesthesia. Direct laryngoscopy and endotracheal intubation is almost always associated with haemodynamic changes due to reflex sympathetic stimulation caused by laryngopharyngeal stimulation.

Methods: A prospective, randomized controlled clinical study was undertaken to compare the efficacy of oropharyngeal lignocaine spray (group L,n=30) and sublingual nitroglycerine spray (group N, n=30) in blunting of haemodynamic response to laryngoscopy and intubation belonging to ASA I, posted for surgery under general anaesthesia.

Results: There was statistically significant difference in the mean heart rate between the groups for the first 3 minutes after intubation. Statistical evaluation between the groups showed the mean SBP was statistically significant (p=0.036) for initial 3 mins after intubation and also at 5th 6th & 10th minute. Statistical evaluation between the groups showed that mean DBP 1st minute after intubation was statistically significant (p=0.008). The difference was significant at 4, 5, 6 and 9 min after intubation. Statistical evaluation between the groups did not show any statistical difference in the MAP except at 5th & 6th minute. However, there was no clinically significant difference in any of the above groups.

Conclusion: Both sublingual NTG & lignocaine group successfully blunted the intubation response, sublingual NTG spray was better in suppressing the BP response to laryngoscopy and intubation than oropharyngeal lignocaine spray but lignocaine controlled the HR response better than NTG.

Keyword: attenuation, sublingual, nitroglycerine, lignocaine

Introduction

Endotracheal intubation is a common mode of securing the airway for administering general anaesthesia. Direct laryngoscopy and endotracheal intubation is almost always associated with haemodynamic changes due to reflex sympathetic stimulation caused by laryngo-pharyngeal stimulation[1].These changes are usually transient, variable and highly unpredictable. Though these changes are usually well tolerated by healthy individuals they can be fatal in patients with hypertension, coronary artery disease and intra cranial hypertension. The IV anesthetic agents used for induction, alone, might not adequately suppress the haemodynamic response evoked by laryngoscopy and endotracheal intubation. So, many additional methods have been tried and used to obtund these responses[2, 3].

Lignocaine has been used both as surface anesthetic and also by IV route to attenuate haemodynamic response to intubation[4]. Lignocaine, by its local surface analgesic property depresses the circulatory response to endotracheal intubation.5 But lignocaine administered as nebulization and as spray have been found to be safer and simpler techniques[5].

NTG and organic nitrates act principally on venous capacitance vessels causing peripheral pooling of blood and reduced cardiac ventricular wall tension[6].NTG generates nitric oxide in vascular smooth muscles which produce vasodilatation leading to decrease in blood pressure[7].Its use for blunting intubation response has been more popular because of its immediate action, short half-life and lack of sedative properties.

This study is undertaken with the objective of comparing the efficacy of sublingual spray of NTG and oropharyngeal spray of lignocaine for attenuation of haemodynamic responses during laryngoscopy and intubation.

Materials and Method

The Study was undertaken in ESIC-PGIMSR hospital, Bengaluru during November 2016 to October 2019. Ethical clearance was obtained for the study.

The study was conducted on 60 ASA grade I patients in the age group of 18 to 40 years of either sex scheduled for elective surgeries done under general anaesthesia. Patients were allocated into two groups with the sample size of 30 each.

Group L (n=30) received oropharyngeal lignocaine 10% spray, 10 puffs, each puff contains 10mg, 3 min before intubation.

Group N (n=30) received NTG spray sublingually 2 puffs, each of 400 mcg.

Inclusion criteria

1) American Society of Anesthesiologists physical status 1 (ASA-PS 1) patients.
2) Aged between 18-40 years posted for elective surgeries under general anaesthesia with endotracheal intubation.

Exclusion criteria

1) Unwilling to participate in the study.
2) Allergy to the study drugs.
3) Anticipated difficult airway.
4) Emergency surgical procedures.
5) Patients requiring rapid sequence induction and intubation.
6) Patients with chronic obstructive lung disease, cerebrovascular disease, cardiovascular diseases, psychiatric illness and liver disorders.
7) Patients having known allergy either to Lignocaine or its preservatives.
8) Patients with history of laryngeal, tracheal surgery or any pathology.

A detailed pre-anaesthetic evaluation including history of previous illness, previous surgeries, general physical examination, and detailed examination of cardiovascular system, Respiratory system and other relevant systems were done. Baseline investigations were carried out and recorded in the proforma.

An informed and written consent was taken from all patient. Detailed pre-anaesthetic evaluation was done. Demographic (age, gender) morphologic (weight) and vital parameters were recorded in patients fulfilling the criteria were selected. The patients were randomly divided into 2 groups of 30 each using a computer generated randomization tables. The patient and the anesthesiologists were blinded to the drug to be used. All patients were pre-medicated with tab alprazolam 0.25 mg and Tab. Ranitidine 150 mg on the night before surgery. On arrival in the operating room, basal parameters-heart rate, systolic blood pressure, diastolic blood pressure, mean arterial pressure, baseline ECG and oxygen saturation using standard monitoring equipment were recorded.

Induction of anaesthesia

After pre-oxygenation for 3 min, all patients received IV inj. Midazolam 0.02mg/kg, inj. fentanyl 2mcg/kg and inj. propofol 2mg/kg. After confirming adequacy of mask ventilation inj. vecuronium 0.1mg/kg was given IV.

An observer 1(anaesthesiology resident) administered the study drugs. Patients in Group L received lignocaine 10% oro-pharyngeal spray 100 mg, 3 min before intubation. Patients in Group N received NTG sublingual spray 0.8 mg 3 min before intubation. Observer 2 (anaesthesiology consultant) did gentle brief laryngoscopy and intubated the patient with appropriate size cuffed endotracheal tube. All intubations were done by the same anesthesiologist. After confirming the proper placement of the endotracheal tube by auscultation and continuous square waveform capnography, the endotracheal tube was fixed. Observer 3(who was blinded) recorded the heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, at induction and at every minute thereafter till 10 min after intubation. Any adverse effects were observed and treated accordingly. Anaesthesia was maintained with 33% oxygen and nitrous oxide mixture(ratio 1:3)with inhalational agent titrated to MAC of 1.3 and intermittent boluses of vecuronium (0.02 mg /kg). Inhalational agent was cut off at the end of surgery and with the onset of spontaneous respiratory efforts, Nitrous oxide was cut-off and 100% oxygen was given and thenpatients were reversed with inj. neostigmine 50mcg/kg and inj. glycopyrrolate 10 mcg/kg slow IV. Patients were extubated after confirming adequate spontaneous respiratory efforts, and patient being awake, warm, obeying commands and maintaining saturation on room air.

Monitoring

The following cardiovascular parameters were recorded in all patients:

- Heart rate (HR) in beats per min (bpm).
- Systolic blood pressure (SBP) in mm Hg.
- Diastolic blood pressure (DBP) in mm Hg.
- Mean arterial pressure (MAP) in mm Hg.

The above cardiovascular parameters were noted as below

1) Basal before giving any study drugs and premedication.
2) One-minute interval for 10 min after laryngoscopy and intubation.

Results

Table 1: Demographic Features

	Group L(n=30)	Group N(n=30)	p value
Age (years)	Mean ± SD	Mean ± SD	0.423
	29.51+/- 5.53	30.18 +/-6.04	
Weight (kgs)	60.6±10.8	64.1±7.4	0.151
Sex-Male: Female	17:13	14:16	0.438

Table 2: Mean HR between the Groups

HR(bpm)	Group LN=30	Group NN=30	p value
	Mean±SD	Mean±SD	
Basal	81.2±10.1	82.5±12.1	0.2
After Intubation			
0 Minute	91.1±16.3	99.7±12.5	0.021*
1 Minute	92.1±14.9	97.4±14.0	0.033*
2 Minute	88.5±13.7	94.9±14.3	0.048*
3 Minute	85.1±14.2	94.6±14.7	0.012*
4 Minute	84.5±14.6	91.2±14.4	0.076
5 Minute	83.4±14.9	89.8±14.8	0.094
6 Minute	82.1±14.7	87.3±15.2	0.177
7 Minute	81.0±14.7	86.5±17.1	0.180
8 Minute	80.7±14.0	87.5±16.0	0.084
9 Minute	79.6±10.7	86.7±15.8	0.042*
10 Minute	79.0±9.4	85.8±13.5	0.025*

Note: *significant at 5% level of significance ($p<0.05$)

The basal mean heart rate between the two groups were comparable (p=0.2). There was statistically significant difference in the mean heart rate between the lignocaine and NTG groups for the first 3 minutes after intubation. However, clinically the difference was not significant (upto 8 bpm).

Table 3: Mean SBP between the Groups

SBP(mmHg)	Group L n-30	Group N n=30	p value
	Mean±SD	Mean±SD	
Basal	123.8±10.9	120.2±10.4	0.12
After Intubation			
0 Minute	130.7±25.0	120.4±17.8	0.046*
1 Minute	125.1±18.8	113.8±14.5	0.036*
2 Minute	121.1±18.5	112.6±18.0	0.042*
3 Minute	118.5±16.8	111.0±18.3	0.043*
4 Minute	116.0±16.3	110.5±16.5	0.190
5 Minute	118.3±16.4	109.0±13.7	0.019*
6 Minute	115.4±15.2	107.5±12.3	0.028*
7 Minute	113.6±15.3	107.4±11.4	0.075
8 Minute	114.4±17.9	107.4±12.6	0.083
9 Minute	114.0±14.3	107.0±13.2	0.090
10 Minute	113.6±14.4	105.2±11.9	0.042*

Note: *significant at 5% level of significance ($p<0.05$)

The basal mean SBP between the two groups were comparable (p=0.12). Statistical evaluation between the groups showed the mean SBP was statistically significant (p=0.036) for initial 3mins after intubation and also at 5^{th} 6^{th}& 10^{th} minute. Even though statistically

significant, the change in the mean SBP between the two groups were not clinically

significant (maximum change was less than 10mm of Hg).
On evaluation of changes in the mean SBP within the groups, in lignocaine group, statistical evaluation showed change in the SBP(fall) from baseline was statistically significant from 6th to 10th minute (p=<0.05) after intubation.
In NTG group, statistical evaluation between the basal and after intubation showed fall in the SBP which was significant throughout from 1st to 10th minute(p=<0.05).

Table 4: Mean DBP between the Groups

DBP(mmHg)	Group Ln=30	Group Nn=30	p value
	Mean±SD	Mean±SD	
Basal	78.2±9.4	76.4±7.3	0.4
After Intubation			
0 Minute	83.7±19.4	74.5±12.5	0.03*
1 Minute	79.0±21.4	66.5±13.6	0.008*
2 Minute	73.1±14.9	69.8±13.5	0.362
3 Minute	74.4±15.1	67.7±14.4	0.077
4 Minute	72.2±14.3	64.9±10.8	0.027*
5 Minute	74.3±15.1	64.5±11.1	0.005*
6 Minute	74.0±14.1	65.1±11.4	0.008*
7 Minute	71.5±14.5	66.4±11.8	0.131
8 Minute	71.0±12.5	65.9±10.7	0.092
9 Minute	70.9±10.3	65.2±8.8	0.023*
10 Minute	70.0±10.6	65.7±11.9	0.135

Note: *significant at 5% level of significance (p<0.05)

The basal mean DBP between the two groups were comparable (p=0.4). Statistical evaluationbetween the groups showed that mean DBP 1st minute after intubation was statistically significant (p=0.008). Also, the difference was significant at 4,5,6 and 9 min after intubation. But the maximum difference was never beyond 10 mm of Hg which was clinically not significant (~10% difference only).
On comparison of DBP within the groups, in lignocaine group, statistical evaluation between the basal and after intubation showed an increase in the DBP for 1 minute after intubation which was neither statistically nor clinically significant and there was a fall in DBP at 4th,5th, 7th to 10th minute which was statistically significant(p=<0.05) but not clinically.
In NTG group, statistical evaluation between the basal and after intubation showed a fall in the DBP throughout but was statistically significant from 1st to 9th minute(p=<0.05). Even here, the fall was not clinically significant.

Table 5: Mean Map between the Groups

Map (mmHg)	Group Ln=30	Group Nn=30	p value
	Mean±SD	Mean±SD	
Basal	92.5±10.1	91.1±8.1	0.554
After Intubation			
0 Minute	98.9±22.2	90.1±14.7	0.072
1 Minute	92.4±21.5	83.7±13.3	0.061
2 Minute	87.5±15.6	83.6±13.6	0.298
3 Minute	88.5±15.6	83.1±14.4	0.157
4 Minute	86.5±14.7	80.1±11.3	0.056
5 Minute	89.4±15.1	79.8±10.4	0.005*
6 Minute	87.6±13.3	79.0±10.7	0.006*
7 Minute	85.8±12.8	80.0±11.3	0.067

8 Minute	84.1±11.6	80.1±10.5	0.157
9 Minute	84.4±11.2	79.2±9.1	0.052
10 Minute	83.2±11.1	79.2±11.7	0.166

Note: *significant at 5% level of significance (p<0.05).

The basal mean MAP between the two groups were comparable (p=0.554).Statistical evaluation between the groups did not show any statistical difference in the MAP except at 5th& 6th minute after intubation. Even at those times, the difference was not clinically significant (8-10 minutes only).

On statistical evaluation of change in mean MAP within the groups, we observed that mean MAP increased till 1 minute after intubation (statistically not significant) and then fell after that and the fallwas statistically significant from 5th to 10th minute only(p=<0.05).

In NTG group, statistical evaluationbetween the basal and after intubation showed fall in the MAPwhich was statistically significant from 1st to 10th minute(p=<0.05).

Discussion

Lignocaine has been used by the following routes to blunt the haemodynamic responses to intubation: gargle, aerosol, intravenous, topical spray.

Lignocaine has been successfully used to blunt the haemodynamic responses to intubation with added benefits throughits various mechanisms like suppression of airway reflexes elicited by irritation of epipharyngeal and laryngopharyngeal mucosa,effective prevention and treatment of laryngospasm,excellent cough suppression, myocardial depression, peripheral vasodilation,Antiarrhythmic properties & analgesic properties when given intravenously.

Bromage *et al*.[8]used 4% lignocaineto provide topical tracheal and bronchial analgesia, prior to intubation under thiopentone anesthesia. No toxic signs were seen in any of the patients studied.

In our study, we observed that there was statistically significant difference in the mean heart rate between the lignocaine and NTG groups for the first 3 minutes after intubation. However, clinically, the difference was not significant (upto 8 bpm). Similar to our study, Madhuri gopal[5]noticed that there was no significant difference in the mean heart rate between the two groups after intubation, clinically or statistically, at any point of time.

On comparison of mean heart rate within the groups, within the lignocaine group, though we observed a statistically significant rise in the HR from baseline till 3 minutes post intubation(p=<0.05), it was not clinically significant(only 10 bpm rise). Similarly Bahaman venus[9] and Qureshi *et al*.[10], in their studies, also observed there was only a modest increase in HR by upto 8bpm and 13.7bpm respectively. However, Madhuri Gopal[5] observed heart rate increased by 24bpm from pre-induction levels, did not come to the pre-induction levels even by tenth minute. This is because of the difference in the timing of administration oflignocaine spray, which was administered 3 mins before induction in her study while we administered the lignocaine spray after induction.

In the NTG group, statistical evaluationbetween the basal and after intubation we found a risein the HR which was statistically significant throughout from 1st to 10th minute(p=<0.05). Similarly, Madhuri Gopal[5] noticed an increase in the heart rate by 21bpm from pre-induction levels which did not come to the pre-induction levels even by 10th minute. Also, Pareek *et al*.[11], Kailash Chandra *et al*.[12] and Tagalpallewar AA *et al*.[10]observed the significant increase in the heart rate in NTG group.

This common finding in NTG group in all the studies can be attributed to the reflex tachycardia caused by NTG.

In our study, Statistical evaluation between the groups showed mean SBP which was statistically significant (p=0.036) for initial 3 min after intubation and also at 5th, 6th& 10th minute. In concurrence with our study, Madhuri Gopal[5]also noticed statistically significant

difference in the mean SBP for initial 3 minutes after intubation, though, in both the studies,

the difference has not been clinically significant.

On evaluation of change in SBP within the groups, in lignocaine group, Statistical evaluation between the basal and after intubation showed initial raise in the SBPupto 1 min after intubation followed by a fall which was statistically significant from 6^{th} to 10^{th} minute only($p=<0.05$).

Madhuri Gopal[5] observed increase in SBP by 17.1mmHg in lignocaine group at 1^{st} minute. The increase in mean SBP was observed till 5 min after intubation which was statistically significant when compared to basal SBP. Beyond 5 mins, there was fall in SBP, but it was not statistically significant. This difference in finding is because of the difference in timing of administration of the lignocaine spray, which was 3 mins before induction while in our study it was administered after induction.

In NTG group, Statistical evaluationbetween the basal and after intubation showed fall in the SBP which was statistically significant from 1^{st} to 10^{th} minute ($p=<0.05$).

Similarly Madhuri Gopal[5], Vijayalakshmi B Channaiah *et al*.[13]and Indira kumara *et al*.[14]also observed decrease in mean SBP from the basal in NTG group.

Thus, in our study, on comparison, we observed both NTG and lignocaine sprays blunted SBP response to intubation, NTG produced more exaggerated fall in SBP within the group.

In our study, there was significant difference in DBP between Lignocaine and NTG groups in response to laryngoscopy and intubation at 0,1,4,5,6 & 9 mins following intubation. But the maximum difference was never beyond 10 mm of Hg which was clinically not significant (~10% difference only).

Madhuri Gopal[5], in her study, found statistically significant difference in, mean DBP between the two groups throughout for 10 mins after intubation and the difference was also clinically significant (>20%).

Contrary to our study Madhuri Gopal[5],observed an increase in mean DBP at 1,3,5 min after intubation which was statistically significant. At 10th minute, there was fall in DBP, but it was not statistically significant. This difference in finding is because of the difference in timing of administration of the lignocaine spray, which was 3 mins before induction while in our study it was administered 3minutes before intubation.

In NTG group, Statistical evaluationbetween the basal and after intubation showed a fall in the DBP which was statistically significant throughout, from intubation upto 10 min($p=<0.05$).

Similar to our study, Madhuri Gopal [5]observed that there was fall in mean DBP from intubation throughout upto 10 mins after intubation when compared to basal DBP.

Even in a study by Indira Kumari *et al*.[14]it was observed that there was a statistically significant decrease in DBP compared to baseline in N1 group (400mcg NTG) and in N2 group (800mcg NTG) at intubation & 1 min after.

Thus, we observed that both NTG and lignocaine sprays blunted DBP response to intubation and that NTG produced more exaggerated fall in DBP within the group.

On intergroup comparison, Madhuri gopal[5]observed that mean MAP was statistically significant between the groups from 1^{st} to 10^{th} minute after intubation. Whereas in our study, Statistical evaluation between the groups showed statistical difference in the MAP only at 5^{th}& 6^{th} minute after intubation, which we think is largely due to the different timing of administration oflignocaine spray.

In lignocaine group, Statistical evaluation between the basal and after intubation showed initial rise in MAP for a minute(not significant) followed by statistically significant fall in the MAP from 5^{th} to 10^{th} minute after intubation ($p=<0.05$). Like SBP & DBP, even the fall in MAP was statistically significant from 5 mins after intubation with the maximum fall from baseline being upto around 9.3mm of Hg. Thus, we found even MAP response also was not clinically important.

Whereas Madhuri Gopal[5] observed an increase in MAP from baseline, maximum of upto

16.47mmHg(>20%), in lignocaine group and returned to near baseline values by 10 mins after intubation. Qureshi *et al*.[10]also observed increase in MAP from baseline till 5 mins after intubation. Again, this difference in finding is because of the difference in timing of administration of the lignocaine spray, which was 3 mins before induction in these studies while in our study it was administered at induction.

In NTG group, Statistical evaluation between the basal and after intubation showed fall in the MAP was statistically significant from 1st to 10th minute(p=<0.05).

Similar to our study Madhuri Gopal[5] and Indira Kumari *et al*.[51]also observed a decrease in MAP till upto 10 mins after intubation in NTG group.

Thus, we found bothNTG and lignocaine to blunt the MAP response to intubation but the suppression was more exaggerated by NTG.

Conclusion

From our study it can be concluded that:

1. Both NTG and Lignocaine sprays successfully blunted the intubation response.
2. Sublingual NTG spray was better in suppressing the BP response to laryngoscopy and intubation than oropharyngeal lignocaine spray but lignocaine controlled the HR response better than NTG.

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