

Efficacy and Optimal Time of Oropharyngeal Topical 10% Lignocaine Spray Before Induction of Anaesthesia in Attenuating the Pressor Response to Direct Laryngoscopy and Endotracheal Intubation.

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Abstract: The study aimed to ascertain the efficacy of oropharyngeal topical 10% lignocaine spray applied prior to induction of anaesthesia and also to determine the optimal time for spraying to attenuate pressor response to laryngoscopy and endotracheal intubation. 80 patients of either sex, in the age group of 20-60 years (ASA I & II) undergoing different elective surgical procedures under general anaesthesia were taken up for the study. 10 puffs of Lignocaine 10% sprayed 1 min before induction in Group 1, 3 min before induction in Group 2, 5 min before induction in Group 3 and normal Saline in Group 4 as control. Heart rate (HR), systolic (SBP), diastolic (DBP) and mean arterial pressure (MAP) were measured at baseline, after intubation, after 1 min, 3 min and 5 min following intubation. There was statistically significant increase in HR, systolic, diastolic and MAP in control group when compared to baseline as well as to study groups. Following laryngoscopy and intubation attenuation of pressor response did not show a significant difference at 1 min, 3 min and 5 min of 10% lignocaine spray within study groups. Lignocaine 10% when sprayed to the oropharynx prior to induction of anaesthesia attenuated the pressor response to laryngoscopy and intubation irrespective of timing of the spray.

Keywords: Endotracheal Intubation, Lignocaine spray (10%), Laryngoscopy, Pressor response, Systolic Blood pressure (SBP), Diastolic blood pressure (DBP), Mean arterial pressure (MAP).

I. Introduction

In practice of anaesthesia, laryngoscopy and tracheal intubation forms the basis of controlling the patient's airway during general anaesthesia or for artificial ventilation. Laryngoscopy and endotracheal intubation has been a challenging procedure due to associated cardiovascular changes called *pressor response*. This response includes widespread release of norepinephrine from adrenergic nerve terminals and secretion of epinephrine from the adrenal medulla¹. In adults and adolescents, the more common response to this airway manipulation is hypertension and tachycardia, mediated by the cardio-accelerator nerves and sympathetic chain ganglia.

In addition to activation of the autonomic nervous system, laryngoscopy and endotracheal intubation result in stimulation of the central nervous system (CNS), as evidenced by increases in electroencephalographic activity, cerebral metabolic rate, and cerebral blood flow (CBF)²⁻⁵.

Pressor response has deleterious effects on patients with preexisting hypertension, ischaemic heart disease which may lead to tachycardia, arrhythmia, cerebrovascular accidents etc.

Several drugs like lignocaine⁶, clonidine⁷, fentanyl⁸ and betablockers⁹, has been tried to control the pressor response to laryngoscopy and intubation but it is not possible to completely abolish the response.

The present study was undertaken to study the efficacy of oropharyngeal topical 10% lignocaine spray in attenuating the pressor response as well as to find the optimal timing of application of lignocaine spray.

II. Materials And Methods:

We conducted a prospective, randomized, controlled study with 80 patients of ASA grade I & II, of either sex, aged between 20 and 60 years, undergoing routine elective surgical procedures under general anaesthesia. Study was conducted after getting institutional ethical committee approval and informed consent from all the patients. We excluded patients with uncontrolled hypertension, significant hepatic or renal disease, predicted difficult intubation, history of hypersensitivity to amide local anaesthetics, seizure disorder and patients taking any systemic medications. After pre-operative evaluation and necessary routine investigations, premedicated with tab Ranitidine 150mg, and tab Lorazepam 1mg 2 hour before induction. Glycopyrrolate 0.2mg was administered intramuscularly 30 min before induction. Routine monitoring was done using

noninvasive blood pressure (NIBP), electrocardiogram (ECG), pulse oximeter (SPO₂). Baseline values of heart rate(HR), systolic blood pressure(SBP), diastolic blood pressure(DBP) and mean arterial pressure(MAP) were measured before spraying with lignocaine(10%).

Patients were randomly allotted into one of the four groups using lottery method; each group consisted of 20 patients. In group 1, patients were subjected to lignocaine 10% spray 1 min before induction, in group 2 lignocaine 10% spray was administered 3 min before induction, similarly in group 3 lignocaine 10% spray was administered at 5min before induction, patients in group 4 were administered normal saline spray who served as control. Lignocaine 10% oral spray was administered in the sitting position by asking the patient to fully open their mouth and using a disposable spray canula into the mouth without using laryngoscope, a total of 10 puffs (2 puffs to soft palate, 3 puffs to posterior oropharyngeal wall, 2 puffs to palatopharyngeal arch and 3 puffs to posterior 3rd of tongue, each puff delivering 10mg, resulting in a total dose of 100mg and not exceeding the toxic dose of 3-4mg/kg. All patients were induced with propofol 2mg/kg followed by rocuronium 0.9mg/kg to facilitate endotracheal intubation. Using Macintosh blade laryngoscopy and intubation was done by anesthesiologist having 5 years of experience. Patients with difficult intubation which require laryngoscopy more than 20 seconds and who require more than one attempt were excluded from the study. Fentanyl 2µg/kg was given as an analgesic. A second recording of HR, SBP, DBP and MAP were noted immediately after laryngoscopy and intubation, and then 1min, 2min, 3min and 5min intervals after laryngoscopy and intubation.

The results were subjected to statistical analysis using Student's unpaired 't' test and ANOVA was used for overall differences. Any p value less than 0.05 were taken as statistically significant. SPSS version 17 was used for analysis.

III. Results:

There was no statistically significant difference in the demographic characteristics among the 4 groups (Table 1)

There was significant rise of heart rate in the control group when compared to the baseline heart rate. There was also a statistically very highly significant ($p < 0.001$) difference compared to study groups 1, 2, 3. However, among the study groups, change in the heart rate was not statistically significant (Tables 2&3). SBP, DBP and MAP showed statistically highly significant change ($p < 0.001$) in control group (4) when compared to study groups and baseline. Moreover, there was statistically insignificant difference in SBP, DBP and MAP ($p = 0.321$, $p = 0.772$, $p = 0.520$) respectively between groups 1, 2, 3 (Tables 4-9).

IV. Discussion:

Laryngoscopy and intubation is associated with a sympathoadrenal response leading to increase in arterial blood pressure, heart rate and may also result in arrhythmias. This bears a particular significance in patients of cardiac condition like hypertension, patients with acute myocardial infarction¹⁻⁵.

The laryngoscopy and endotracheal intubation (ET) intubation is considered to be a stronger stimulus than a surgical incision. The amount of pressor response is related to the force applied to the base of tongue, the duration of laryngoscopy. There have been many different ways tried to decrease the pressor response ranging from non-pharmacological methods like using LMA¹⁰, various types of laryngoscope blades like McCoy¹¹ for doing laryngoscopy, to pharmacological methods like lignocaine, alpha-2 agonists, opioids, nitroglycerine, beta-blockers among others^{6-9,12}.

Lignocaine has been tried as intravenous administration prior to laryngoscopy, mouth gargles in various concentration, sprays. The mechanism of action of lignocaine in blunting pressor response differs according to the method of administration. Local administration like gargles and sprays may be effective due to its local anaesthetic property at the base of tongue and pharyngeal walls preventing the receptor stimulation. The intravenous administration mainly acts by decreasing the cerebral blood flow and depression of myocardial contractility¹³. Abou-Madi *etal*¹⁴ have discussed the possible mechanisms to account for these observations with IV lignocaine. These include a direct myocardial depressant effect, a peripheral vasodilation effect and finally an effect on synaptic transmission.

In our study we used oropharyngeal topical 10% lignocaine spray prior to induction to determine the efficacy in attenuating the pressor response and optimal timing of spraying. We used 10 puffs in total, each puff delivering 10mg, so total dose given was 100 mg. This dose is well below the toxic dose of lignocaine. We used glycopyrrolate¹⁵ 0.2 mg as a premedication which has been proved to be enhancing absorption, prolongs the analgesic action of lignocaine and has anti-sialagogue property. We found that the lignocaine spray was effective in attenuating pressor response (in terms of heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure) in all the three groups receiving the drug in comparison to the group 4 which received normal saline and to the baseline values.

We performed a single laryngoscopy prior to endotracheal intubation whereas laryngoscopy was performed twice by Mostafa *etal*¹⁶ because they administered the spray after induction, thus results are not

comparable. Hamill *et al*¹⁷ compared topical lignocaine with I.V. lignocaine. In the topical group, after induction of anesthesia they performed laryngoscopy to spray the oropharynx, two minutes later, they performed another laryngoscopy for endotracheal intubation. While in other group they performed only one laryngoscopy and hence noxious stimuli were given twice in the topical group. Mostafa¹⁶ and Hamill¹⁷ in their respective studies had concluded that topical lignocaine is ineffective in preventing the pressor response. Takita *et al*¹⁸ suggested that differing intervals between tracheal lignocaine and endotracheal intubation probably caused the inconsistent conclusion reported in other investigations and showed that endotracheal intubation performed two minutes after tracheal lignocaine attenuated the cardiovascular responses to endotracheal intubation. M. Jain *et al*¹⁹ performed a study on lignocaine 10 percent spray to study its efficacy in attenuating the pressor response and found it to be effective in attenuating and not abolishing the response, we had similar findings in lignocaine being able to attenuate the pressor response, but they didn't study the optimal timing of the spray and they didn't use glycopyrrolate as a premedication.

V. Conclusion

We conclude that spraying oropharynx with 10 puffs (100mg) of topical 10% lignocaine is effective in blunting the pressor response to laryngoscopy and intubation when it is applied before induction of general anaesthesia irrespective of timing of the spray.

Table 1: Demographic characteristics (mean ± SD) of four groups

Demographic characteristics	Group 1 (n=20)	Group 2 (n=20)	Group 3 (n=20)	Group 4 (n=20)	Statistical significance
Age	40.76± 5.8	41.10 ± 7.0	41.06 ± 7.2	40.82 ± 8.1	P = 0.986
Sex (Male/Female)	11/9	10/10	11/9	8/12	X ² = 0.973 P = 0.823

n = number of subjects

Table 2: Table showing Mean ± SD changes in Heart rate in groups 1, 2, 3 (Study) and group 4 (Control)

HEART RATE	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	86.5 ±12.1	87.0±11.3 ^{NS}
After L & I*	103.0±8.1	118.0±14.2 ^a
After 1 MIN	100.2±7.8	113.6±14.6 ^a
After 2 MIN	96.0±6.2	106.7±11.5 ^a
After 3 MIN	92.2±5.3	99.3±9.8 ^a
After 5 MIN	88.9±4.2	95.2±7.8 ^a

*Laryngoscopy and Intubation
^a = Highly significant (p<0.001)
^{NS} = Not significant (p>0.05)

Table 3 Table showing Mean ± SD changes in Heart rate in groups 1, 2, 3 (ANOVA)

HEART RATE	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value [#]
Baseline	86.8	85.9	88.0	0.839
After L & I*	103.1	103.2	102.8	0.989
After 1 MIN	100.4	100.2	100,0	0.989
After 2 MIN	96.1	95.4	96.4	0.867
After 3 MIN	92.0	91.3	93.3	0.411
After 5 MIN	88.8	88.1	89,9	0.338

*Laryngoscopy and Intubation
[#]P values >0.05 are insignificant

Table 4 Table showing Mean ± SD changes in Systolic blood pressure in groups 1, 2, 3 (Study) and group 4 (Control)

SYSTOLIC BLOOD PRESSURE	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	127.1 ±8.5	128.2±7.1 ^{NS}
After L & I*	141.6±7.0	161.2±5.7 ^a
After 1 MIN	138.6±6.6	155.4±5.6 ^a
After 2 MIN	135.8±6.0	149.6±5.6 ^a
After 3 MIN	132.9±5.6	144.9±5.1 ^a
After 5 MIN	131.8±5.7	140.8±6.4 ^a

* Laryngoscopy and Intubation

^a = Highly significant (p<0.001)

^{NS} = Not significant (p>0.05)

Table 5 Table showing Mean ± SD changes in Systolic blood pressure in groups 1, 2, 3 (ANOVA)

SYSTOLIC BLOOD PRESSURE	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value [#]
Baseline	125.8	126.9	128.7	0.578
After L & I*	140.2	141.7	143.0	<0.001
After 1 MIN	137.3	138.6	139.8	<0.001
After 2 MIN	134.6	135.8	136.8	<0.001
After 3 MIN	132.0	132.7	133.9	<0.001
After 5 MIN	130.6	131.2	133.4	<0.001

* Laryngoscopy and Intubation

[#] P values >0.05 are insignificant

Table 6 Table showing Mean ± SD changes in Diastolic Blood Pressure in groups 1, 2, 3 (Study) and group 4 (Control)

DIASTOLIC BLOOD PRESSURE	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	81.3±7.5	81.8±6.7 ^{NS}
After L & I*	93.4±7.4	105.3±6.1 ^a
After 1 MIN	90.8±7.0	100.6±4.7 ^a
After 2 MIN	87.6±6.9	96.3±4.9 ^a
After 3 MIN	85.3±6.5	91.9±5.0 ^a
After 5 MIN	82.9±6.4	87.8±4.9 ^a

* Laryngoscopy and Intubation

^a = Highly significant (p<0.001)

^{NS} = Not significant (p>0.05)

Table 7 Table showing Mean ± SD changes in Diastolic Blood Pressure in groups 1, 2, 3 (ANOVA)

DIASTOLIC BLOOD PRESSURE	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value [#]
Baseline	80.8	80.7	82.5	0.807
After L & I*	93.0	92.9	94.3	<0.001
After 1 MIN	90.4	90.5	91.5	<0.001
After 2 MIN	87.7	87.0	88.0	<0.001
After 3 MIN	85.4	84.7	85.8	<0.001
After 5 MIN	83.0	82.3	83.6	<0.001

*Laryngoscopy and Intubation

#P values >0.05 are insignificant

Table 8 Table showing Mean ± SD changes in Mean Arterial Pressure in groups 1, 2, 3 (Study) and group 4 (Control)

Mean arterial pressure	Groups 1,2,3 STUDY Mean ± S.D	Group 4 CONTROL Mean ± S.D
Baseline	97.6 ±6.8	97.2±5.6 ^{NS}
After L & I*	109.5±6.2	123.9±5.3 ^a
After 1 MIN	106.7±5.8	118.9±4.0 ^a
After 2 MIN	103.6±5.7	114.0±4.2 ^a
After 3 MIN	101.1±5.2	109.6±4.0 ^a
After 5 MIN	99.2±5.1	105.5±4.0 ^a

*Laryngoscopy and Intubation

^a = Highly significant (p<0.001)

^{NS} = Not significant (p>0.05)

Table 9 Table showing Mean ± SD changes in Mean Arterial Pressure in groups 1, 2, 3 (ANOVA)

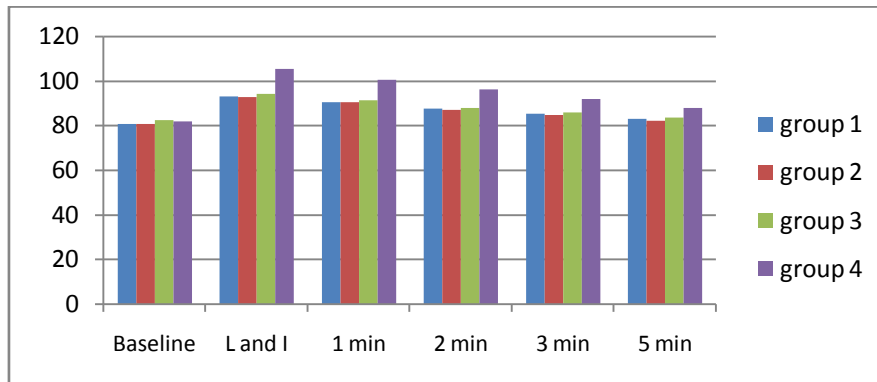
Mean arterial pressure	Group 1 Mean	Group 2 Mean	Group 3 Mean	P Value [#]
Baseline	95.8	96.1	97.9	0.496
After L & I*	108.7	109.2	110.6	0.553
After 1 MIN	106.1	106.5	107.6	0.641
After 2 MIN	103.3	103.3	104.3	0.780
After 3 MIN	100.9	100.7	101.8	0.713
After 5 MIN	98.9	98.6	100.2	0.501

*Laryngoscopy and Intubation

#P values >0.05 are insignificant

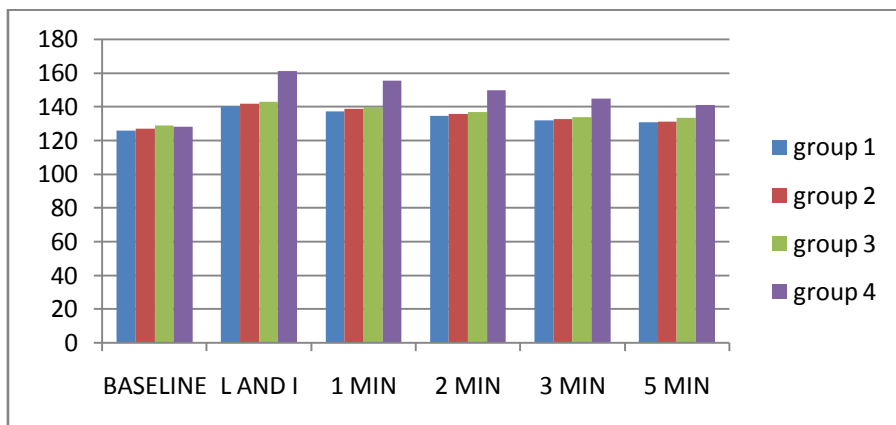
Mean ± SD changes in Heart rate in groups 1, 2, 3 (Study) and group 4 (Control)

Heart rate



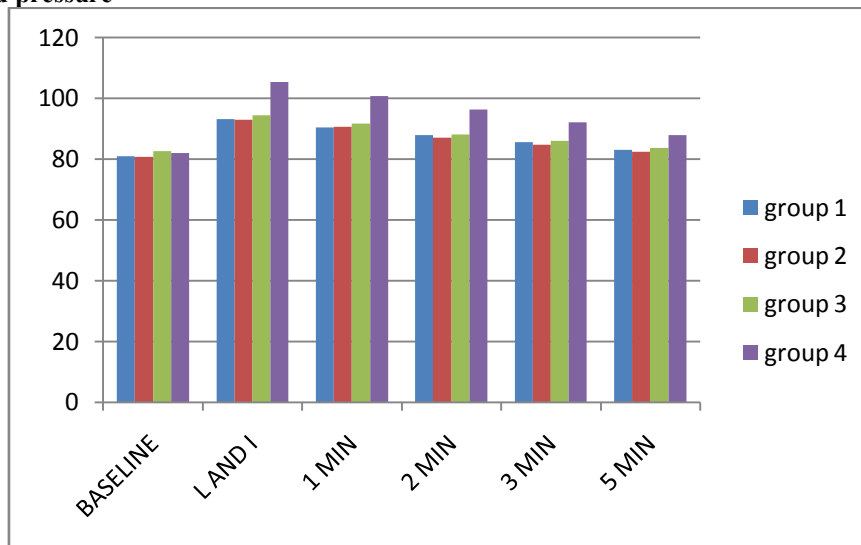
Mean ± SD changes in Systolic blood pressure in groups 1, 2, 3 (Study) and group 4 (Control)

Systolic BP

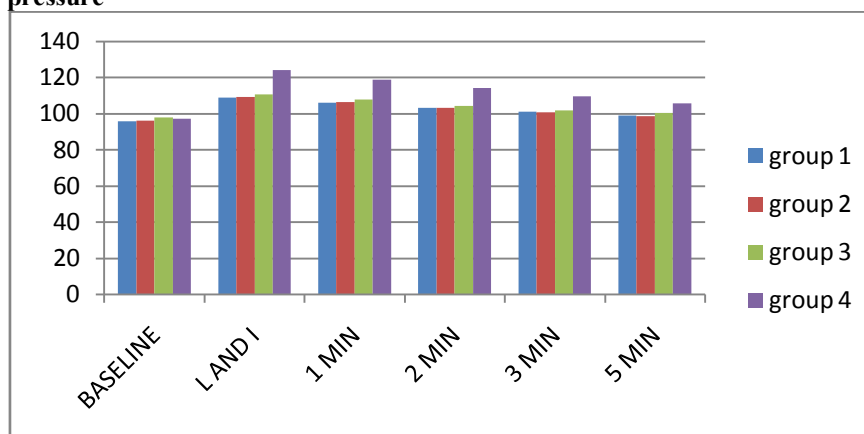


Mean ± SD changes in Diastolic Blood Pressure in groups 1, 2, 3 (Study) and group 4 (Control)

Diastolic blood pressure



Mean \pm SD changes in Mean Arterial Pressure in groups 1, 2, 3 (Study) and group 4 (Control)
 Mean arterial pressure



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