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Attenuation of pressor response to laryngoscopy and intubation with topical lignocaine 10% spray in the controlled hypertensive patients

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Abstract
Laryngoscopy and tracheal intubation is invariably associated with a reflex sympathetic pressor response resulting in elevated heart rate and blood pressure. There are many studies conducted to attenuate this pressor response, the present study is undertaken to compare efficacy of 10% lignocaine spray with normal saline spray applied before induction in attenuating the pressor response to laryngoscopy and intubation in controlled hypertensive patients.
Methods: 60 patients of either sex aged between35-60 years, belonging to ASA I and II undergoing elective surgery requiring general anaesthesia with endotracheal intubation were included. Patients were randomly divided in two groups of 30 patients each. Group I (Study group) received topical lignocaine 10% spray 2 minutes before intravenous induction. Group II (Control group) received topical normal saline 2 minutes before intravenous induction.
Results: Showed that the topical lignocaine 10% spray significantly attenuates the rise in mean systolic blood pressure, diastolic blood pressure and heart rate when compared with the baseline as well as the control group. There was a statistically significant ($p<0.05$) increase in heart rate, systolic and diastolic blood pressure in group II when compared to group I and baseline values**.**
Conclusion: Study concluded that topical lignocaine 10% spray did not completely abolish the rise in heart rate, SBP and DBP but only attenuate it when compared with the baseline or control group.

Keywords: pressor response, laryngoscopy and intubation and 10% lignocaine spray

Introduction
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 medulla [1]. Pressor response has deleterious effects on patients with pre-existing hypertension, ischemic heart disease which may lead to tachycardia, arrhythmia, cerebrovascular accidents etc [2]. These responses are serious enough in normotensive patients and are more so pronounced in hypertensive patients [3]. The pressor response to laryngoscopy is due to sympathetic reflex proved by mechanical stimulation of epipharynx and laryngopharynx [4, 5]. Laryngoscopy alone generate the same pressor and sympathoadrenal response as compared to laryngoscopy followed by intubation. The major cause of sympathoadrenal response to tracheal intubation arises from stimulation of supraglottic region by tissue tension induced by laryngoscopy. Placing endotracheal tube (ETT) through the cords and inflating cuff in the infraglottic region contributes very little additional stimulation.
Rise in blood pressure and heart rate with laryngoscopy is usually transitory variable and unpredictable. The hypertensives are prone to have significant increase in blood pressure [6, 7]. The pressor response increases progressively with the maximum rise at 45 seconds and further 15 seconds of laryngoscopy causes additional stimulation. This suggests that the magnitude of haemodynamic response depends upon the duration and the intensity of the stimulus exerted during laryngoscopy and related to extended period of airway intervention [8]. Hypertension usually lasts less than 10 minutes [7]. The ECG changes observed following endotracheal intubation usually last from 15 seconds to 10 minutes [9]. Many different ways have been tried to decrease the pressor response ranging from non-pharmacological methods like using LMA [10], various types of laryngoscope blades like McCoy [11] for doing

laryngoscopy, to pharmacological methods like lignocaine, alpha-2 agonists, opioids, nitroglycerine, beta-blockers among others [8].

Lignocaine has been used both topically and intravenously for the attenuation of the pressor response to laryngoscopy and intubation. Lignocaine is absorbed following topical administration and its rate and extent of absorption being dependent upon concentration of total dose administered, the specific site of action and duration of exposure [12].

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 contractility [13, 14].

With this background, the present study was undertaken to compare the efficacy of topical lignocaine 10% spray with the normal saline spray applied before induction in attenuating the pressure response to laryngoscopy and intubation in controlled hypertensive patients.

Methodology

After obtaining approval from hospital ethics committee, the study was conducted in the Postgraduate Department of Anaesthesiology and Intensive Care Government Medical College Jammu. Patients between 35 to 60 years of either sex scheduled for routine elective surgical procedure were included. Patients with uncontrolled hypertension, hepatic and renal disease, difficult intubation, history of drug allergy to amide group, seizure disorders, pregnant and lactating women were excluded from the study.

An informed written consent was taken from patients and Pre-anaesthetic check-up was done a day before surgery including a detailed history, a thorough physical and systemic examination. Routine investigations were carried out. Patients were kept 8 hours overnight fasting before surgery and were premedicated with tablet alprazolam 0.5 mg at bed time. Intradermal test for lignocaine sensitivity was done in all patients. Intravenous canulla was placed and I.V. RL started. Injection tramadol 1 mg/kg i/v+ injection midazolam 0.03 mg/kg i/v given 10 minutes before anaesthesia. The patients were divided randomly into two groups of 30 patients each. Group I (study group) received topical lignocaine 10% spray 2 minutes before intravenous induction. Group II (control group) received topical normal saline 2 minutes before intravenous induction.

In the operating room routine monitoring including non-invasive blood pressure (NIBP), electrocardiogram (ECG) and peripheral oxygen saturation (SPO_2) was performed. Baseline values of heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured before spraying lignocaine 10%. Patients received oropharyngeal 10% lignocaine spray using a pump metered spray in the dose of 1.5 mg/kg up to a maximum of 100 mgs in sitting position during inspiration. Lignocaine 10% oral spray was applied as single spray bilaterally to the soft palate, posterior oropharyngeal wall, palatopharyngeal arch and base of tongue, as well as 2 sprays to the vallecular region using a disposable spray cannula. (10 spray in total)

Induction: After a period of 2 minutes, patients were induced with sodium thiopentone 5 mg/kg followed by atracurium 0.6 mg/kg to facilitate endotracheal intubation. Two minutes after induction, direct laryngoscopy with a standard Macintosh laryngoscope blade was performed which was followed by endotracheal intubation. Anaesthesia was maintained with 65% N_2O and O_2 (35%) plus 0.5 - 1% halothane. During the study period no surgical stimulus was allowed. Later on, after completion of surgery patients were reversed with injection neostigmine 0.05 mg/kg and glycopyrolate 0.01 mg/kg. Heart rate, systolic blood pressure and diastolic blood pressure were noted at baseline and after 1 minute, 2, 3 and 5 minutes following laryngoscopy and intubation.

At the end of study, the results were subjected to statistical analysis using Student's 't' test. Any p-value less than 0.05 were taken as statistically significant. The analysis of data was performed on statistical package for social sciences (SPSS) for Windows.

Observation and Results

There was no statistically significant difference as per demographic profile of patients (age, sex and weight), both groups were comparable. In group I there was increase in Heart rate, SBP and DBP upto 2 minutes and then declined to almost baseline by 5 minutes. Whereas, in group II Heart rate, SBP and DBP kept on increasing up to 3 minutes and did not reach baseline values even at 5 minutes.

Table 1: Age distribution of the patients

Age group (in years)	Group I (Study group) No. (%)	Group II (Control group) No. (%)
35-44	19 (63.33)	23 (76.67)
45-54	10 (33.33)	7 (23.33)
55-60	1(3.34)	0
Total	30 (100.00)	30 (100.00)

Majority of the patients were in the age group of 35-44 years in both the groups, followed by 45-54 years. There was only one patient in the age group of 55-60 years in Group I.

Table 2: Sex distribution of the patients

Sex	Group I (Study group) No. (%)	Group II (Control group) No. (%)
Males	17 (56.67)	16 (53.33)
Females	13 (43.33)	14 (46.67)
Total	30 (100.00)	30 (100.00)

All the patients of both the sexes were equally distributed in both the groups.

Table 3: Comparison of mean weight of patients in two groups

Variable	Group I (Study group) Mean ± Standard deviation	Group (Control group) Mean ± Standard deviation	Statistical inference
Mean Weight (in Kg)	57.75 ± 5.7	57.20 ± 5.9	$p>0.05$ (insignificant)

The mean weight in both the groups was comparable in Group I 57.75 ± 5.7 Kg and Group II 57.20 ± 5.9 Kg, the difference between the two groups was statistically insignificant ($p>0.05$).

Table 4: Comparison of mean systolic blood pressure (SBP) of patients in two groups

SBP at	Group I (Study group) Mean (mmHg)	Group II (Control group) Mean (mmHg)	Statistical inference
Baseline	130.66	132.43	P=0.47 (Insignificant)
1 minute	142.06	140.43	P=0.56 (Insignificant)
2 minutes	146.53	145.33	P=0.69 (insignificant)
3 minutes	134.83	148.83	P=0 (significant)
5 minutes	120.83	138.53	P=0.000 (Significant)

In Group I, mean SBP increased during the procedure up to 2 minutes and then started declining to almost baseline. In Group II, means SBP increased up to 3 minutes and then started declining but did not reach baseline even at 5 minutes. The differences in mean SBP between the two groups at 3 and 5 minutes were statistically highly significant.

Table 5: Comparison of mean diastolic blood pressure (DBP) of patients in two groups

DBP at	Group I (Study group) Mean (mmHg)	Group II (Control group) Mean (mmHg)	Statistical inference
Baseline	82.16	78.33	P=0.09(Insignificant)
1 minute	94.33	84.63	P=0.00 (Significant)
2 minutes	97.63	89.64	P=0.000 (Significant)
3 minutes	89.93	92.63	P=0 (Significant)
5 minutes	82.53	81.46	P=0.037(Insignificant)

In Group I, mean DBP increased during the procedure up to 2 minutes and then started declining to almost baseline. However, in Group II mean DBP increased up to 3 minutes and then started declining but did not reach baseline even at 5 minutes. The differences in mean DBP between the two groups at 1, 2 and 3 minutes were statistically highly significant.

Table 6: Comparison of mean heart rate (HR) of patients in two groups

HR at	Group I (Study group) Mean (beats per minute)	Group II (Control group) Mean (beats per minute)	Statistical inference
Baseline	85.22	79.84	P=0.19(Insignificant)
1 minute	97.83	84.23	P=0.002 (Significant)
2 minutes	103.56	88.93	P=0.000 (Significant)
3 minutes	96.74	92.36	P=0.17 (Insignificant)
5 minutes	87.43	83.18	P=0.01(Significant)

In Group I, mean HR increased during first 2 minutes of intubation and then started declining to almost baseline. Whereas, in Group II mean HR increased up to 3 minutes and then started declining but did not reach baseline even at 5 minutes. The differences in mean HR between the two groups at 1, 2 and 5 minutes were statistically significant.

Discussion

In practice of anaesthesia, laryngoscopy and tracheal intubation forms the basis of controlling the patient's airway during general anaesthesia or for artificial ventilation. H G Manjunath and Ravi L [15]. Direct laryngoscopy and intubation leads to reflex release of catecholamines resulting in hemodynamic response meaning transient increase in heart rate, systolic blood pressure, diastolic blood pressure and occasionally cardiac arrhythmias. Russell *et al.* [16] and Derbyshire [17].

King *et al.* [6] reported that reflex response to laryngoscopy and intubation in the form of increase in heart rate, increase systolic and diastolic blood pressure in 1951.Shribman A J *et al.* [5] found that blood pressure increase was due to laryngoscopy even without intubation but tachycardia was massively due to intubation while comparing the haemodynamic response to laryngoscopy with or without intubation.

Various studies have examined the efficacy of topical lignocaine for attenuation of cardiovascular response to endotracheal intubation. Delinger *et al.* [18] showed that a single spray with lignocaine attenuated the hypertensive response to endotracheal intubation when compared to saline tracheal. Others showed that application of topical anesthesia to upper airway failed to prevent the pressor responses to endotracheal intubation [19]. In our study, we applied lignocaine 10% spray before the induction and performed laryngoscopy once in both the groups. Our procedure was simple because we sprayed lignocaine before the induction of anesthesia and hence only one laryngoscopy was needed compared to other studies which were more laborious because of necessity of two laryngoscopies. The drawback we encountered was that topical lignocaine was bitter in taste and provoked cough reflex in few patients. This can be decreased by swish and gargle with viscous lignocaine prior to spray as performed by Sitzman *et al.* [20] Keeping in view studies of Derbyshire *et al.* [16], Sitzman *et al.* [8] and the safe plasma lignocaine levels, our method of using 10 puffs of 10% lignocaine (100 mgs) was effective in attenuating the pressor response to laryngoscopy and intubation.

Hypertensive and tachycardia responses to laryngoscopy and tracheal intubation have been attributed to increased sympathetic activity caused by stimulation of the upper respiratory tract [4, 6], a concept supported by the observation that increases in arterial pressure during tracheal intubation are associated with increases in plasma norepinephrine concentration [16].

If the trachea is intubated immediately after spraying of the larynx and trachea with lignocaine, cardiovascular responses are not attenuated Hamill *et al.*, [19] Stoelting [7]. This is expected because laryngoscopy itself, without intubation of the trachea, is associated with similar cardiovascular

responses.

However, as in accordance to our study when the trachea is intubated 5 minutes after laryngotracheal spray of lignocaine, hypertensive responses to laryngoscopy and tracheal intubation are significantly decreased [18], perhaps because of absorption of lignocaine into the systemic circulation [19].

Conclusion

The topical lignocaine 10% is an effective method for attenuating the pressor response but not abolishing it completely to laryngoscopy and intubation without causing an increased risk of hypotension in controlled hypertensive patients.

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