

Efficacy Of Topical Lignocaine Spray (10%) Applied Before The Induction Of Anaesthesia In Attenuating The Pressor Response To Direct Laryngoscopy And Endotracheal Intubation In Controlled Hypertensive Patients

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Citation

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Abstract

We evaluated the efficacy of topical lignocaine spray (10%) applied prior to induction of anaesthesia in attenuating the pressor response to laryngoscopy and endotracheal intubation in 50 controlled hypertensive patients, undergoing different elective surgical procedures under general anaesthesia. Patients were allocated randomly to one of the 2 groups of 25 patients each. Group I received lignocaine 10 % oral spray 2 minutes prior to induction of anaesthesia for a total of 10 puffs (100 mg). Group II received normal saline spray 2 mts prior to induction of anaesthesia. Heart rate, systolic, diastolic and mean arterial pressure were measured. There was a statistically significant ($p < 0.05$) increase in heart rate, systolic, diastolic and mean arterial pressure in group II when compared to group I and also when compared to baseline values. It was concluded that topical lignocaine 10% when sprayed prior to induction of anaesthesia attenuated the pressor response to laryngoscopy and intubation, but did not abolish it completely.

INTRODUCTION

Laryngoscopy and endotracheal intubation provoke cardiovascular responses that include hypertension, tachycardia and dysrhythmias. These responses are serious enough in normotensive patients and are more so pronounced in hypertensive patients.^{1,2} A number of drugs including calcium channel blockers, beta adrenergic blockers, narcotics and vasodilators have been used with variable success.^{3,4,5}

Lignocaine has been used both topically and intravenously for the attenuation of the pressor response to laryngoscopy and intubation. The effect of topical lignocaine in attenuating the pressor response to laryngoscopy has been controversial. 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.^{6,7}

It has been found that topical lignocaine sprayed before induction of anaesthesia to be more effective than lignocaine sprayed after induction of anaesthesia in attenuating the pressor responses.⁸

With this background, the present study was conducted to ascertain the efficacy of topical lignocaine 10% applied prior to induction of anaesthesia for attenuation of pressor response to laryngoscopy and endotracheal intubation.

MATERIAL AND METHODS

Fifty controlled hypertensive ASA grade II patients of either sex aged between 35 and 65 years undergoing routine elective surgical procedures under general anaesthesia participated in this prospective randomized study. Ethical committee approval was received and informed consent was obtained from all patients. Patients with history of uncontrolled hypertension, significant hepatic or renal disease, patients with predicted difficult intubation, patients with history of hypersensitivity to amide group of local anaesthetics, patients with seizure disorder or increased airway resistance were excluded from the study.

All patients received 10 mgs of oral diazepam 2 hours before induction of anaesthesia. On arrival in the operating room an intravenous line was established. Routine monitoring including noninvasive blood pressure (NIBP) with Datex Cardiocap IITM, electrocardiogram (ECG), and oxygen

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saturation (SPO₂) was performed. Baseline values of heart rate, systolic blood pressure(SBP),diastolic blood pressure(DBP) and mean arterial pressure(MAP) were measured(in mmHg) before spraying lignocaine(10%).

Patients were randomly assigned one of 2 groups each comprising 25 patients. Group I received lignocaine 10% (LOX Topical spray, Neon Labs) spray 2 minutes before induction of anaesthesia. Group II received normal saline spray prior to induction of anaesthesia and served as control. Patients received oropharyngeal 10% lignocaine spray using a pump metered spray in the dose of 1.5mg/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 (for ten sprays in total).After a period of 2 minutes, patients were induced with sodium thiopentone 5 mg/kg followed by atracurium 0.6mg/kg to facilitate endotracheal intubation. Morphine 0.1mg/kg was given as analgesic. 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% N₂O in O₂ and 0.5-1% halothane. Heart rate, Systolic blood pressure, diastolic blood pressure and mean arterial blood pressure were noted at baseline, after induction, immediately after laryngoscopy and intubation, after 1mt,2mts,3mts and 5mts following laryngoscopy and intubation. During the study period no surgical stimulus was allowed. At the end of study, the results were subjected to statistical analysis using students paired and unpaired 't' test, more ever ANOVA was used for overall differences. 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) version 5.00, Chicago, USA for windows.

RESULTS

There was no statistically significant difference in the physical characteristics of the two groups (Table 1)

Figure 1

Table 1: Physical characteristics (mean+SD) of Group I and Group II

Physical characteristics	Group I (n=25)	Group II (n=25)	Statistical significance
Age (years)	47.20 ± 7.3	46.96 ± 8.2	P=0.997 (insignificant)
Sex (M/F)	10 M/15 F	12 M/13 F	X ² =0.962 P=0.811 (insignificant)

In group I, there was a significant rise in heart rate as compared to baseline values (p<0.05), which was maximal immediately after laryngoscopy and intubation (20.1%).After 5 minutes of intubation, heart rate returned to near normal values(p>0.05)(table 2). After intubation there was a statistically significant rise in systolic, diastolic and mean arterial blood pressure as compared to baseline values which was maximal immediately after laryngoscopy and intubation(p<0.05).

In group II,there was a statistically significant rise in heart rate as compared to baseline values which was maximal immediately after laryngoscopy and intubation(35.6%), and the rise in heart rate remained significant(p<0.05) throughout the study period.

Figure 2

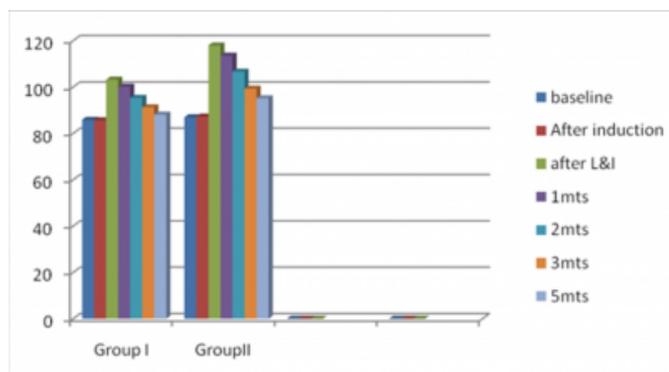
Table 2: Mean change in heart rate at various intervals between 2 groups

Groups	Baseline	Heart Rate (beats/mt) Mean ± SD					
		After induction	After Laryngoscopy and intubation	1mt after L&I	2mts after L&I	3mts after L&I	5mts after L&I
Group I	85.9±12.4	85.7±12.1	103.2±9.6	100.2±9.3	95.4±7.3	91.3±5.9	88.1±4.8
Group II	87.0±11.3	87.3±11.2	118.0±14.2	113.6±14.1	106.7±11.5	99.3±9.8	95.2±7.8

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Figure 3

Figure 1: Heart rate changes at various time intervals between 2 groups



After intubation there was a statistically significant rise in systolic, diastolic and mean arterial blood pressure as compared to baseline values, which was maximum after laryngoscopy and intubation ($p < 0.05$) (table-2). When group I was compared with group II, immediately after laryngoscopy and intubation there was a statistically significant ($p < 0.05$) rise in systolic, diastolic and mean arterial pressure in group I. Similarly there was statistically highly significant ($p < 0.001$) rise in systolic, diastolic and mean arterial pressure in group II when compared with group I and difference remained significant throughout the study period. (table-3)

Figure 4

Table 3: Mean change in systolic, diastolic and mean arterial pressure (mmHg) between the 2 groups.

Variable & Groups	Mean \pm SD						
	Baseline	After induction	After Laryngoscopy and intubation	1mt after L&I	2mts after L&I	3mts after L&I	5mts after L&I
Systolic Blood pressure(mmHg)							
Group I	126.9 \pm 9.5	126.7 \pm 9.4	141.7 \pm 6.9	138.6 \pm 6.7	135.8 \pm 6.3	132.7 \pm 6.0	131.2 \pm 6.3
Group II	128.2 \pm 7.1	128.3 \pm 6.8	161.2 \pm 5.7	155.4 \pm 5.6	149.6 \pm 5.6	144.9 \pm 5.1	140.8 \pm 6.4
Diastolic blood pressure(mmHg)							
Group I	80.7 \pm 8.0	80.8 \pm 7.8	92.7 \pm 7.9	90.5 \pm 7.6	87.0 \pm 7.7	84.7 \pm 7.3	82.3 \pm 7.1
Group II	81.8 \pm 6.7	81.8 \pm 6.7	105.3 \pm 6.1	100.6 \pm 4.7	96.3 \pm 4.9	91.9 \pm 5.0	87.8 \pm 4.9
Mean arterial pressure(mmHg)							
Group I	96.1 \pm 7.3	96.1 \pm 7.2	109.2 \pm 6.4*	106.5 \pm 6.3	103.3 \pm 6.3	100.7 \pm 5.9	98.6 \pm 5.7
Group II	97.2 \pm 5.6	97.3 \pm 5.5	123.9 \pm 5.3**	118.9 \pm 4.0	114.0 \pm 4.2	109.6 \pm 4.0	105.5 \pm 4.0

DISCUSSION

The precise mechanism which leads to hemodynamic

changes to laryngoscopy and intubation is not known but it probably involves intense sympathetic discharge caused by stimulation of epipharynx and laryngopharynx. This suggests that direct laryngoscopy is the major stimulus for pressor responses, with an additional stimulus caused by tracheal intubation. In addition the longer the duration of laryngoscopy, the greater is the pressor response.

Our results show that topical lignocaine significantly attenuates the rise in heart rate when compared with baseline as well as control group. Similarly systolic pressure, diastolic pressure and mean arterial pressure in group I (study group) when compared with baseline and control group showed a highly significant ($p < 0.001$) rise after direct laryngoscopy and intubation. Topical lignocaine did not completely abolish the rise in heart rate or systolic, diastolic and mean arterial blood pressure but only attenuated it when compared with the control or baseline levels. Several studies have examined the efficacy of topical lignocaine for attenuation of cardiovascular response to endotracheal intubation. Delinger et al showed that a single spray with lignocaine attenuated the hypertensive response to endotracheal intubation when compared to saline tracheal spray. Others showed that application of topical anesthesia to upper airway failed to prevent the pressor responses to endotracheal intubation. Takita et al 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. Hamill et al compared topical lignocaine with I.V. Lignocaine. In the topical group, after induction of anesthesia they performed laryngoscopy to spray the orolarynx. Two minutes later, they performed another laryngoscopy for endotracheal intubation while as in other group they performed only one laryngoscopy and hence noxious stimuli were given twice in topical group. Thus the results obtained were erroneous. Similarly laryngoscopy was performed twice by Mostafa et al in one of his study group and thus results could not be interpreted correctly.

In our study, we had applied lignocaine spray before the induction of anesthesia and we performed laryngoscopy only once in both the groups. Moreover, our procedure was simple to perform 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 draw back we encountered during our study 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 al¹⁵. Regarding dose of lignocaine spray, 2% to 10% sprays have been given. Keeping in view studies of Mostafa, Derbyshire, Sitzman and Scott et al 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. In our study none of the patients developed hypotension because of various drug combinations used.

We conclude that topical lignocaine (10%) is an effective method for attenuating but not abolishing the pressor response to laryngoscopy and intubation without producing an increased risk of hypotension.

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