EFFICACY OF LIGNOCAINE IN THREE DIFFERENT ROUTES IN ATTENUATION OF HAEMODYNAMIC RESPONSE ASSOCIATED WITH ENDOTRACHEAL INTUBATION : A COMPARATIVE STUDY

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Abstract

Background: Transient haemodynamic instability i.e tachycardia and hypertension is an inevitable outcome of laryngoscopy and endotracheal intubation which can have serious effects in patients with COPD, IHD or hypertension that can lead to ischaemia, infarction, arrhythmia or cardiac arrest in vulnerable patients. Administration of lignocaine is one of the techniques used to attenuate haemodynamic response associated with endotracheal intubation. The present study was designed to compare the changes in blood pressure and heart rate that occur before and after the endotracheal intubation in three different routes intravenous(i.v.) local spray over laryngeal inlet or nebulization.

Materials and methods : It was a randomized double blind clinical trial. It was conducted at the Department of Anaesthesiology, Chittagong Medical College Hospital, Chattogram from July 2005 to June 2007. A total of one hundred and fifty adult patients (ASA I, Mallampati grade 1 & 2) scheduled for elective surgery under general anaesthesia wereselected. Patients were randomly allocated in three groups A,B and C, by lottery .Group A received 2% lignocaine i.v., Group B received 10% lignocaine spray over the laryngeal inlet and vocal cords and group C patients were nebulized with 2% lignocaine solution.

Results: The rise in Systolic, Diastolic and mean arterial pressure with local spray was significantly lower than with intravenous and nebulized group and the rise in heart rate was significantly lower with local spray group than with nebulization group and intravenous group. Data were processed using software SPSS version 11.5. The test statistics used to analyse the data were Chi-squire(χ^2) test and Anova.

Conclusion: The findings of this study reflects the local spray of 10% lignocaine over laryngeal inlet prior to intubations attenuates the rise in blood pressure and heart rate more than intravenous administration of lignocaine or nebulization of lignocaine.

Key words
Lignocain; Attenuation of haemodynmic response; Endotracheal intubation.

Introduction

In general anaesthetic procedure laryngoscopy and endotracheal intubation is doneafter premedication with midazolum or injection pethidine and after induction with thiopentone sodium and a short acting muscle relaxant suxamethonium is given. Laryngoscope is placed over the right half of the tongue and then the tongue is then displaced upward forward and to the left not rotating the handle. The tip of the blade is placed at the valeculae then gentle lift will open the laryngeal inlet. then the endotracheal tube is inserted into the laryngeal inlet and trachea. The cardiovascular response to laryngoscopy and intubation is in the form of hypertension and tachycardia. Blood pressure is increased by 20%-40% of the initial mean arteria pressure and heart rate is increased by 20%. The changes are maximum at one minute after intubation and lasts for 5-10 mins[1]. Serious consequence of this haemodynamic response includes arrhythmia, myocardial infarction,stroke congestive heart failure and even cardiac arrest[1,2]. Marked transient rise inintracranial pressure and notable bronchial hyper- reactivity may result from inner airway irritation leading to bronchospasm. Induction of anaesthesia and intubation is often a period of haemodynamic instability In spite of preoperative BP control, many patients with hypertension shows exeserated hypertensive response to intubation upto 25%[3]. Sudden rise in blood pressure will raise

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the ICP in patient underwent operation for ICSOL or head injury. Raised ICP may aggravate is chemic injury to the brain. Moreover if a patient is suffering from heart disease like myocardial ischemia, rise in blood pressure may lead to myocardial infarction due to imbalance of oxygen demand and supply ratio[4].

One of the following techniques may be used before intubation to attenuate the hypertensive response[3].

i) Increasing the depth of anaesthesie by increasing the conc of volatile agents for 5-10 mins

ii) Administration of bolus of an opioid (e.g fentanyl 50-100 micro gm)

iii) Administration of lignocaine 1-2mg/kg i.v

iv) 4. B-Blocker (e.g) Propranolol .5-1 micro gm

v) Using topical airway anaesthesia.

Materials and methods

A prospective randomized control trial was conducted in the Department of Anesthesiology, Chittagong Medical College Hospital, during the period July 2005 to June 2007. Obtaning permission from ethical committee of Chittagong Medical Medical College a total of 150 patients were enrolled in the study, who were from 18 to 55 years of age, Mallampati Class-I & Class- II. They were randomly allocated into three groups A, B and C by lottery in the pre anaesthetic checkup. Group-A received I.V. 2% lignocain, 1.5 mg/kg, Group-B received 10% lignocaine, 1.5mg/kg spray over laryngeal inlet and vocal cord and Group-C were nebulized with 2% lignocaine, 1.5mg/kg by the anaesthetists. In aresearch instrument, a case record form, data were collected by the investigator himself. Data were analyzed SPSS version 11.5. The test used to analyze the date were Chi square (χ^2) test and ANOVA. For all analytical tests the level of significance was set at .05 and $p< 0.05$ was considered significant. The summarized data were presented in the forms of table and charts.

Results

Table I : Monitoring of systolic BP throughout the observation period.

Systolic BP (mm Hg)	Group			p Value
	Group A (n=50) mean ± SD	Group B (n = 50) mean ± SD	Group C (n=50) mean ± SD	
At Baseline	111.6 ± 15.9	102.9 ± 18.7	109.3 ± 18.4	0.071
Before intubation	122.9 ± 16.4	117.0 ± 15.7	129.0 ± 24.9	0.008
Just after intubation	121.3 ± 27.7	122.2 ± 20.3	129.1 ± 31.2	0.281
1 minute after intubation	152.6 ± 22.4	138.1 ± 26.2	150.4 ± 32.1	0.010
2 minute after intubation	140.7 ± 21.9	129.4 ± 21.7	144.6 ± 26.6	0.004
3 minute after intubation	128.1 ± 17.6	121.7 ± 18.4	133.3 ± 26.6	0.027
4 minute after intubation	125.3 ± 20.5	116.4 ± 20.3	127.8 ± 25.5	0.029
5 minute after intubation	122.1 ± 25.5	113.7 ±18.2	127.6 ± 24.3	0.011
10 minute after intubation	122.8 ± 24.4	114.9 ± 19.7	119.7 ± 22.1	0.203
15 minute after intubation	120.1± 22.7	110.0 ± 21.01	118.9 ± 28.1	0.078

Date were analyzed using ANOVA statistics and are presented as mean ± SD .

The data show that systolic blood pressures of Group-A and Group-C at 1 and 2 minutes after intubation rose more above the base line compared to those of Group-B (p =0.010 and p = 0.004 respectively) while the Group-B maintained the blood pressure more nearer to base line throughout observation period (Table I).

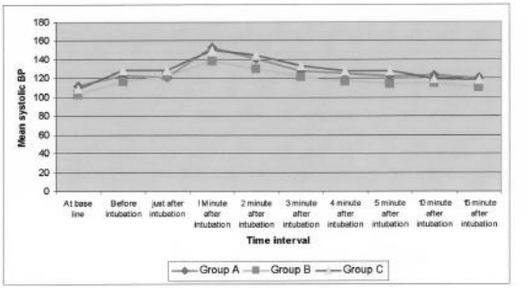

Fig 1 : Monitoring of systolic BP at different time interval.

Comparison of systolic BP at different time interval:

Fig. 2 compares to the changes in systolic BP among the groups. The mean systolic BPs of Group-A, Group-B and Group-C demonstrated a rise from their baseline status 111.6 ± 15.9, 102.9 ± 18.7 and 109.3 ± 18.4 mm Hg respectively to 152.6 ± 22.4, 138.1 + 26.2 and 150.4 ± 32.1 respectively at 1 minutes after intubation.

The curves then showed a declining trend throughout the observation period and came down to 120.1 ± 22.7, 120.1 ± 22.7 and 118.9 ± 28.1 mm Hg at 15 minutes after intubation.then showed a declining trend throughout the observation period and came down to 120.1 ± 22.7, 120.1 ± 22.7 and 118.9 ± 28.1 mm Hg at 15 minutes after intubation (Fig 1).

Table II : Monitoring of diastolic BP throughout the observation period.

Diastolic BP (mmHg)	Group A (n=50) mean ± SD	Group B (n = 50) mean ± SD	Group C (n=50) mean ± SD	pValue
At baseline	73.2 ± 11.4	68.2 ± 10.8	71.3 ± 10.0	0.067
Before intubation	78.4 ± 11.1	73.7 ± 10.9	79.8 ± 13.3	0.026
1 minute after intubation	93.8 ± 19.1	82.4 ± 13.9	92.3 ± 18.1	0.002
2 minute after intubation	83.6 ± 15.9	75.2 ± 11.9	85.2 ± 14.1	0.001
3 minute after intubation	80.0 ± 14.2	71.7 ± 12.5	80.8 ± 13.9	0.002
4 minute after intubation	75.6 ± 15.4	68.7 ± 15.2	77.7 ±13.7	0.007
5 minute after intubation	72.1 ± 16.9	66.6 ± 13.4	80.2 ± 21.6	0.001
10 minute after intubation	78.7 ± 16.4	71.4 ± 13.3	75.6 ± 15.1	0.054
15 minute after intubation	74.5 ± 12.8	70.2 ± 13.8	78.4 ± 16.3	0.019

Data were analyzed using ANOVA statistic and are presented as mean ± SD.

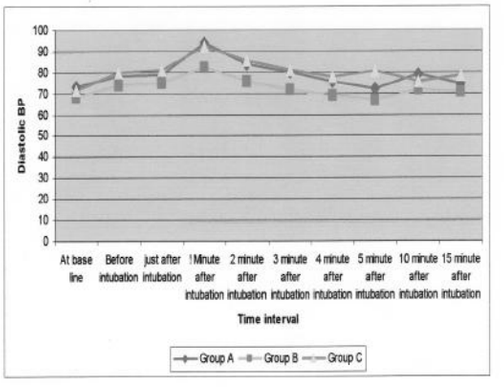

Fig 2 : Diastolic BP at different time intervals.

The diastolic BPs of Group-B were observed to be more close to base line records throughout the whole period of observation, where as those of Group-A and Group-C went beyond the base line at 1 minutes after intubation (p= 0.002) (Table II).

Table III : Monitoring of MAP throughout the observation period.

MAP (mm Hg)	Group A (n=50) mean ± SD	Group B (n = 50) mean ± SD	Group C (n=50) mean ± SD	p Value
At baseline	85.6 ± 12.0	80.3 ± 13.1	83.9 ± 11.5	0.085
Before intubation	93.3 ± 10.9	88.1 ± 11.4	96.3 ± 15.6	0.006
Just after intubation	93.1 ± 19.7	90.4 ± 14.0	96.9 ± 19.9	0.201
1 minute after intubation	113.4 ± 18.8	100.9 ± 17.2	111.7 ± 21.4	0.003
2 minute after intubation	102.6 ± 16.5	93.3 ± 14.0	105.4 ± 17.1	0.001
3 minute after intubation	96.1 ± 13.6	88.4 ± 13.3	98.3 ± 17.2	0.003
4 minute after intubation	92.1 ± 15.6	64.6 ± 15.9	94.4 ± 17.1	0.008
5 minute after intubation	88.8 ± 18.6	82.3 ± 14.0	96.0 ± 19.1	0.001
10 minute after intubation	93.4 ± 17.9	85.9 ± 14.0	90.6 ± 16.2	0.068
15 minute after intubation	89.7 ± 15.1	83.0 ± 14.9	91.9 ± 17.7	0.017

Date were analyzed using ANOVA statistics and are presented as mean ± SD.

The MAP of Group-A and Group-C were found to be significantly higher than those of Group-B at 1,2,3,4,5 and 15 minutes after intubation (p = 0.003, 0.001, p= 0.003,p= 0.008, p = 0.001 and p = 0.017 respectively) (Table III).

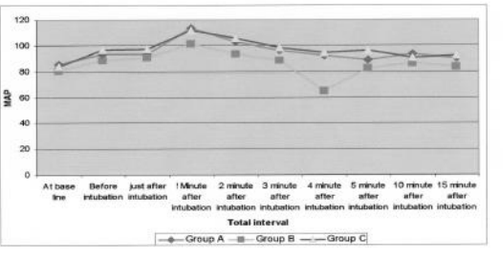

Fig 3 : Mean Arterial Pressure (MAP) at different time interval.

Table IV : Monitoring of heart rate throughout the observation period.

Heart rate (/min)	Group A (n=50) mean ± SD	Group B (n = 50) mean ± SD	Group C (n=50) mean ± SD	p Value
At baseline	81 ± 11	86 ± 14	82 ± 11	0.054
Before intubation	91 ± 20	91 ± 18	91 ± 18	0.935
Just after intubation	99 ± 19	93 ± 18	91 ± 18	0.065
1 minute after intubation	110 ± 18	99 ± 19	99 ± 22	0.007
2 minute after intubation	105 ± 21	95 ± 17	98 ± 24	0.058
3 minute after intubation	99 ± 18	90 ± 18	94 ± 23	0.075
4 minute after intubation	96 ± 19	86 ± 15	89 ± 23	0.032
5 minute after intubation	91 ± 18	85 ± 21	86 ± 19	0.218
10 minute after intubation	87 ± 22	79 ± 14	81 ± 19	0.065
15 minute after intubation	82 ± 17	80 ± 15	79 ± 22	0.560

Date were analyzed using ANOVA statistics and are presented as mean ± SD.

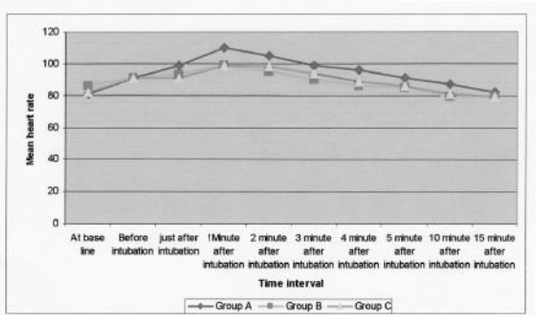

Fig 4 : Monitoring of heart rate at different time interval.

Although heart rates of the three groups were within normal range, the Group A demonstrated a significantly higher rate at 1 and 4 minutes after intubation than Group-B and Group-C did (p =0.007 and p = 0.032 respectively) (Table IV).

Discussion

Laryngoscopy and endotracheal intubation can cause striking change in haemodynamics and intracranial pressure as a result of intense sympathetic nervous system stimulation. There is an increase of 40 - 50% in the mean arterial blood pressure and 20% increase in heart rate the changes are maximum at one minute after intubation and lasts for 5 - 10 minutes[1,5]. Serious consequences of the haemodynamic responses include arrhythmias, myocardial ischaemia, infraction, stroke, congestive heart failure and even cardiac arrest[1,2]. The present study was intended to compare the efficacy of lignocaine in three different routes - (a) intravenous route (b) local spray and (c) nebulisation showed that baseline characteristics of the three groups were almost identical.

The heart rate of intravenous group just after intubation, rose sharply to nearly 100/minute and at 1 minute after intubation to 110/min. However, the heart rate of local spray and nebulization group did not exhibit any demonstrable change just after intubation and rose to 107/ min. at 1 minute after intubation. Thereafter the heart rate of all the three groups began to fall steadily and dropped to their baseline level at 15 minutes after intubation. Intravenous administration of lignocaine demonstrates a significantly higher rate at 1 and 4 minutes after intubation than local spray and nebulisation did. This is consistent with the findings of the Stoelting's study. In Stoelting's study the patients were divided into three treatment groups, with 12 patients in each category. Control patients received only laryngotracheal administration of lignocaine (2mg/kg) just before placement of the tracheal tube. In the second group, viscous lignocaine (25 ml, 2%) was utilized as mouthwash and gurgle 10 minutes before anaesthetic induction. Lignocaine 1.5 mg/kg was administered i.v. to the remaining 12 patients 90 seconds before the start of laryngoscopy and intubation. The heart rate after one minute of intubation increased during laryngoscopy and intubation, but the magnitude was significantly lower in the viscous lignocaine group than the control and intravenous group. Topical oropharyngeal anaesthesia produced by local anaesthetics seems more specific as this should anaesthetize that area in contact with laryngoscope blade or endotracheal tube. Stimulation of these areas would seem the most likely explanation for rise in blood pressure and heart rate during laryngoscopy and intubation.

The mean systolic BPs in intravenous injection, local spray and in nebulisation demonstrated a rise from their baseline status at 1 minute after intubation followed by a declining trend throughout the observation period and brought around 120 mmHg at 15 minutes after intubationshowed that systolic blood pressure of intravenous group and nebulisation group at 1 and 2 minutes after intubation rose far above base line to those of local spray group. The local spray group maintained the blood pressure more nearer to baseline level throughout the observation period. Which is consistent with the study of Takita etal[6]. In the study by Takita etal seventy five patients were studied in three groups[6]. In group A (n= 25) endotracheal intubation was performed without lignocaine. In group B (n=25) endotracheal intubation was done just after tracheal lignocaine spray (4% Lignocaine, 4ml). In group C (n=25) endotracheal intubation was performed 2 minutes after tracheal lignocaine spray. The changes in SBP caused by endotracheal intubation in 2 minutes after tracheal lignocaine in group C is significantly less than those caused by endotracheal intubation in Group-A and Group-B. Denlinger et al showed that a simple tracheal spray with lignocaine attenuated the hypertensive responses to endotracheal intubation when compared with saline tracheal spray[7]. Others showed that the application of topical anesthesia to upper airway and trachea failed to prevent the pressor responses to endotracheal intubation[8-10]. In the study of Denlinger et al. (1974) endotracheal intubation was performed more than 2

minutes after a tracheal spray with local anesthetics, while in others, endotracheal intubation was performed less than 1 minute after topical anesthesia that indicated the in effectiveness of tracheal lignocaine[9]. The present study suggests that the differing intervals between tracheal lignocaine and endotracheal intubation probably caused the inconsistent conclusions reported in other studies.

The diastolic blood pressures of intravenous group, local spray group and nebulization group first increased insidiously from baseline to 94 mm Hg, 83 mm Hg and 92 mm Hg respectively at 1 minute after intubation. The intravenous group and local spray group then experienced a steady decrease up to 5 minutes after intubation when they fell to 72 and 66 mmHg respectively and then again rose to 74 and 72 mmHg respectively at 15 minutes after intubation. Although the diastolic blood pressure of intravenous and local spray group experienced frequent changes, they did not fall below the lower limit of normal physiological range which is consistent with the findings of Takita et al[6].

The mean pressure of intravenous and nebulization groups increased sharply from about 93 and 97 mmHg just after intubation to about 113 and 112 mmHg at 1 minute after intubation and then decreased insidiously to 90 and 92 mmHg respectively at 15 minutes after intubation. However, the MAP in cases of local spray of lignocaine exhibited significant changes. Throughout the observation period with 80 mmHg at baseline, 90 mmHg just after intubation, about 101 mmHg 1 minute, 93 mmHg at 2 minutes, nearly 65 mmHg at 4 minutes, returning to about 86 at 10 minutes and dropped again to 83 mmHg at 15 minutes after intubation. This study is consistent with the study of stoelting et el[11]. His study shows MAP increases were significantly greater ($p<0.05$) at all times in the patients of control group compared with the viscous lignocaine treated groups.

Limitations

- The study should be conducted on large sample with size
- Lack of direct arterial and venous pressure monitoring system in the operation theatre.

Conclusion

The finding of this study reflected that local spray of 10% lignocaine over laryngeal inlet prior to intubation attenuates the blood pressure and heart rate more than intravenous administration or nebulization of lignocaine.

Recommandation

- Orotracheal spray of 10% lignocaine before intubation is recommended for endotracheal intubations
- Furter study with a large sample size is needed.
- Multicetred study needs to be conducted to generalize the findings.

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Contribution of authors

AANC-Conception, design, acquisition data, drafting & final approval.
AKMSA-Design, analysis of data, critical revision & final approval.
GAC-Data analysis, interpretation of data & final approval.

Disclosure

All the authors declared no competing interest.

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