Section: Anesthesiology

Original Research Article

ATTENUATION OF HAEMODYNAMIC RESPONSES FOR LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION– A COMPARATIVE STUDY BETWEEN LIGNOCAINE ORAL VISCOUS 2% AND ORAL LIGNOCAINE SPRAY 10 % PRIOR TO GENERAL ANAESTHESIA

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ABSTRACT

Background: To compare efficacy between oral lignocaine viscous 2% and oral lignocaine spray 10% in attenuating the hemodynamic response for laryngoscopy & endotracheal intubation prior to general anaesthesia.

Materials and Methods: This study was carried out in 60 patients belonging to ASA I& II, aged between 20 to50 years undergoing elective surgeries. The study will be conducted for a period of 1year in Department of Anaesthesia in Kurnool Medical College, Kurnool. Patients who had hypersensitivity to study drug, patients with severe renal, hepatic, respiratory, cardiac disease, neurological, psychiatric disorders, Difficult Airway-Cormack & Lehane grade 3 and 4 were excluded from the study. Divided in to two groups. Group V receives 10ml of 2% or a lignocaine viscous gargle for 5 minutes prior to induction. Group S receives five puffs of oral lignocaine spray 10% prior to induction. The values for HR, SBP, DBP, and MAP, SPO2 were obtained baseline, after induction, immediately after intubation and 1,3,5 and10 minutes after intubation and compared among the groups.

Results: In the current study, after induction the mean HEART RATE starts increasing in group S and in group V mean heart rates tart decreasing after induction and slightly raised after immediately after intubation and gradually decreasing till 10 min after intubation. In group S immediately after intubation highest mean heart rate was observed, and start decreasing at 1 minute, 3 minute, 5minute and 10 minute after intubation but not reached baseline level, the mean heart rate between two groups from after induction to 10 minutes after intubation was significant. ($p<0.05$). In group S mean SBP start increasing immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation. The mean SBP between two groups from 1 minute after induction to 10 minutes after intubation was significant. ($p<0.05$). In group V mean DBP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean DBP between two groups from after induction to 10 minutes after intubation was significant. ($p<0.05$). The mean MAP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but reached baseline level at 3minutes.However, in group V mean MAP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean MAP between two groups from after induction to 10 minutes after intubation was significant. ($p<0.05$). After induction the mean SPO2 starts increasing in both groups and reached to 100% in both groups.

Conclusion: The study concluded that oral lignocaine viscous 2% was more effective in blunting the haemodynamic response to laryngoscopy and endotracheal intubation than lignocaine spray 10% No significant adverse events occurred during the study.
Keywords: Lignocaine, Hemodynamic parameters, MAP, Endotracheal intubation.

INTRODUCTION

A crucial part of general anaesthesia is endotracheal intubation. It assists in maintaining the patency of the upper airway, ensuring proper ventilation, lowering the risk of aspiration, and giving patients access to inhalational anaesthetics through breathing circuits.1The most important procedures during the induction of general anaesthesia are laryngoscopy and tracheal intubation, which activate somatic and visceralnociceptive afferent fibres that trigger reflex sympathoad renal responses and are associated with increased neuronal activity of cervical sympathetic efferent fibres.[2]

The percentage of difficult intubations ranges from1%to6% of all intubations, and the incidence of unsuccessful intubations ranges from 0.1% to 0.3% of all intubations, according to an incomplete statistical analysis.[3,4] The majority of the patients in the emergency room and intensive care unit are hospitalised for serious illnesses; as a result, medical personnel must quickly complete endotracheal intubation and, more importantly, provide airway management in circumstances where adequate emergency planning and facilities are not available.

Patients undergoing general anesthesia lose consciousness and the ability to control breathing and protect their airway. Tracheal intubation is considered a vital procedure that secures the airway and provides the possibility of continued oxygenation. Direct laryngoscopy and endotracheal intubation are mostly associated with hemodynamic changes caused by reflex sympathetic discharge, caused by epi pharyngeal and laryngopharyngeal stimulation.[6,7,8] Tachycardia, hypertension,[9] and other symptoms are caused by sympathoad renal activity. Arrhythmias,[10,11] whichare potentially dangerous. These changes are most pronounced at one minute after laryngoscopy and intubation and lasts for 5-10 minutes This increase of Blood pressure and heart rate are typically transient, variable, and unpredictable.[9]

Lignocaine is an aminoethyl amide. it is a prototype of amide group of local anaesthetics, introduced in 1948.Itis most widely used local anaesthetic. Lignocaine has been used both topically and intra venously for attenuation of pressor response during laryngoscopy and endotracheal intubation. Lignocaine blocks sodium channels in the myocardium, thus reducing the rate of rise of action potential and altering conduction velocity throughout the His-Purkinje system and atrium and ventricular musculature.[10]

Usually, it is administered via the intravenous route at 1.5mgkg−1 body weight for 3 min before intubation to suppress the hemodynamic response. However, such suppressionis not complete and a spike in SBP at 1-min and 3-minintervalspost-intubation has been reported.[11,12]

Need for the study

Laryngoscopy & endotracheal intubation are essential for anesthesiologist to maintain patent airway during general anesthesia & in intensive care unit for mechanical ventilation.

Laryngoscopy and endotracheal intubation induced presser responsesare associated with increase in blood pressure & heart rate due to increase in catecholamines release namely epinephrine and nor epinephrine. Increase Blood pressure & Heart rate are due to sympathoadrenal response which is short acting but they may have detrimental effect in high risk patients with cardio vascular disease. Therefore, it is important to find effective means of attenuating sympathetic response due to laryngoscopy & endotracheal intubation.

Hereby a study conducted to compare efficacy of oral lignocaine viscous 2% vs lignocaine spray 10% in order to attenuate haemodynamic responses to laryngoscopy & endotracheal intubation for patients posted electively for surgeries under general anaesthesia.

Aim of the study

To compare efficacy between oral lignocaine viscous 2% and oral lignocaine spray 10% in attenuating the hemodynamic response for laryngoscopy & endo tracheal intubation prior to general anaesthesia.

Objectives of the study

The following parameters will be compared using 10ml oral lignocaine viscous 2% & 5 puffs of oral lignocaine spray 10% before induction of Anaesthesia. Haemodynamic Changes-Heart Rate Blood pressure, oxygen saturation.

MATERIAL AND METHODS

Study design: The present study is cross–sectional study.

Sample size: This is a comparison study and the study will be conducted in 60 ASA grade I and II adult patients. Divided into two groups. Group V receives 10 ml of 2% or a llignocaine viscous gargle for 5minutes prior to induction. Group S receives five puffs of oral lignocaine spray prior to induction.

Study Area: Government general hospital, Kurnool.

Sampling Method: Simple Random sampling

Study Subjects: ASA I & II adult patients of either sex scheduled to undergo elective surgeries under general anaesthesia at Kurnool Medical College, Kurnool.

Ethical issues and ethical committee clearance: The study was taken up after the approval of the Ethical committee of the medical college. During the study, purpose of the study was explained to all study subjects in his/her own language and informed written consent was taken.

Study Period

The study will be conducted for a period of 1yearin department of Anaesthesia in Kurnool Medical College, Kurnool.

Inclusion Criteria

1. Belonging to ASA grade I and II.
2. Patients belonging to age 20 to 50 yrs
3. Patients giving informed written consent.
4. Patients scheduled to undergo elective surgeries under general anaesthesia

Exclusion Criteria

1. Patient refusal or procedure.
2. Patients belonging to ASA III &IV.
3. Active URTI and LRTI.
4. Patients with Asthma, Obstructive sleep apnea, Obesity,
5. Bleeding disorders,
6. Allergic to drugs.

Investigations required

Blood: Haemoglobin, Bleeding time, clotting time, Blood grouping and Typing. RBS, Urea, Creatinine. ECG and Chest X-ray.

Statistical analysis

Appropriate statistical analysis of data will be done using one of the following tests.

1. All the values will be analysed and expressed as Mean +/-SD
2. Student test and ANOVA test for parametric data.
3. Chi-square test for non-parametric data.
4. P<0.05 will be considered as statistically significant.

RESULTS

DEMOGRAPHICDATACOMPARISON

AGEDISTRIBUTION

Mean age of the patients in the groups was compared using independent sample 't' test. The average age in groupVwas37.33years and group S was 36.03. Therefore, we conclude that the difference between these groups was statistically insignificant (p value: 0.588). This is shown in Table no.1

VITALPARAMETERDATA

HEARTRATE

The table no.2 and graph no.4 show the trend of HEART RATE from baseline to over a period of 10 min after intubation. The basal (BL) mean heart rate of group S was 84.50±4.09 and group V was 84.17±7.64, and the difference in the mean heart rate at baseline among the groups was not statistically significant (p>0.05). After induction the mean heart rate starts increasing in group S and in group V mean heart rate start decreasing after induction and slightly raised after immediately after intubation and gradually decreasing till10min after intubation. In group S immediately after intubation highest mean heart rate was observed, and start decreasing at 1 minute, 3 minute, 5 minute and 10 minute after intubation but not reached baseline level, the mean heart rate between two groups from after induction to10 minutes after intubation was significant. (p<0.05). [Table 2]

SYSTOLICBLOODPRESSURE

The table no.3 shows the trend of SYSTOLIC BLOOD PRESSURE from baseline to over a period of 10 min after intubation.

At baseline, the mean systolic blood pressure (SBP) of group S was129.13±7.51, group V was 129.60±7.38 and the difference in mean SBP at baseline among the groups is not statistically significant (p>0.05). After induction the mean SBP starts decreasing in both groups. However, in group S mean SBP start increasing immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation. The mean SBP between two groups from 1 minute after induction to 10 minutes after intubation was significant. (p<0.05). [Table 3]

DIASTOLIC BLOOD PRESSURE

The table no.4 shows the trend of DIASTOLIC BLOOD PRESSURE from baseline to over a period of 10 min after intubation.

At baseline, the mean Diastolic blood pressure (DBP) of group S was 74.93±3.99, group V was 75.50±3.69, and the difference in mean DBP at baseline among the groups is not statistically significant (p>0.05). After induction the mean DBP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but never reached baseline level. However, in group V mean DBP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till10 min after intubation. The mean DBP between two groups from after induction to 10 minutes after intubation was significant. (p<0.05). [Table 4]

MEANARTERIALPRESSURE

The table no.5 shows the trend of MEAN ARTERIAL PRESSURE from baseline to over a period of 10 min after intubation.

At baseline, the average mean arterial pressures (MAP) in group S was 92.90±4.35, group S was 93.23±4.03 and the difference in mean MAP at baseline among the groups is not statistically significant (p>0.05). After induction the mean MAP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but reached baseline level at 3 minutes. However, in group V mean MAP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean MAP between two groups from after induction to 10 minutes after intubation was significant. (p<0.05). [Table 5]

MEAN SPO2

The table no. 6 shows the trend of MEAN SPO2 from baseline to over a period of 10 min after intubation. At baseline, the average MEAN SPO2 in group S was 97.70±0.75, group S was 97.73±0.83 and the difference in mean MAP at baseline among the groups is not statistically significant (p>0.05). After induction the mean SPO2 starts increasing in both groups and reached to 100% in both groups. [Table 6]

Table 1: Age Distribution of Patients Studied

Group	N	Mean	SD	P value
Oral lignocaine spray10%(S)	30	36.03	9.41	0.588
Lignocaine oral viscous 2%(V)	30	37.33	9.07	

Table 2: Comparison of Heart Rate (Bpm) In The Study Groups at Different Time Intervals

Time	Heart rate				
	Group	N	Mean	SD	P value
Base line(BI)	S	30	84.50	4.09	0.834
	V	30	84.17	7.64	
After induction (AI)	S	30	89.10	5.43	<0.001
	V	30	78.80	7.38	
Immediately after intubation (IAI)	S	30	105.73	7.14	<0.001
	V	30	83.50	7.97	
1min (T1)	S	30	102.76	7.24	<0.001
	V	30	80.67	6.32	
3min(T3)	S	30	96.47	7.26	<0.001
	V	30	77.30	5.57	
min (T5)	S	30	92.00	5.79	<0.001
	V	30	72.47	4.91	
10min(T10)	S	30	91.33	5.99	<0.001
	V	30	72.23	5.34	

Table 3: Comparison of SBP (Mm Hg) Among The Study Groups at different Time Intervals

Time	SBP				
	Group	N	Mean	SD	Pvalue
Baseline	S	30	129.13	7.51	0.232
	V	30	129.60	7.38	
After induction	S	30	124.80	9.49	0.064
	V	30	120.93	5.93	
Immediately after intubation	S	30	133.37	5.25	<0.001
	V	30	117.20	12.25	
1min	S	30	127.97	6.35	<0.001
	V	30	110.53	9.20	
3min	S	30	121.57	6.06	<0.001
	V	30	100.90	10.64	
5min	S	30	119.47	5.33	<0.001
	V	30	96.30	10.58	
10min	S	30	119.47	6.72	<0.001
	V	30	97.53	11.21	

Table 4: Comparison of DBP (Mmhg)Among the study Groups at Different Time Intervals

Table 4: Comparison of DBP (Mmhg)Among the study Groups at Different Time Intervals

Time	DBP				
	Group	N	Mean	SD	P value
Baseline	S	30	74.93	3.99	0.571
	V	30	75.50	3.69	
After induction	S	30	77.97	5.96	<0.001
	V	30	71.43	3.35	
Immediately after intubation	S	30	85.00	4.59	<0.001
	V	30	77.47	9.18	
1min	S	30	81.80	4.81	<0.001
	V	30	73.90	5.09	
3min	S	30	75.87	4.47	<0.001
	V	30	68.83	4.48	
5min	S	30	79.90	3.72	<0.001
	V	30	73.33	4.91	
10min	S	30	76.47	5.18	<0.001
	V	30	70.77	6.64	

Table 5: Comparison of MAP (MMHG) In the study Groups at Different Time Intervals

Time	MAP				
	Group	N	Mean	SD	P value

Baseline	S	30	92.90	4.35	0.759
	V	30	93.23	4.03	
After induction	S	30	93.67	6.18	0.001
	V	30	88.50	4.92	
Immediately after intubation	S	30	100.99	3.40	<0.001
	V	30	91.50	9.09	
1min	S	30	96.91	5.48	<0.001
	V	30	86.43	5.81	
3min	S	30	91.13	4.36	<0.001
	V	30	79.63	4.96	
5min	S	30	92.93	3.32	<0.001
	V	30	81.10	6.31	
10min	S	30	91.07	5.98	<0.001
	V	30	79.87	7.78	

Table 6: Comparison of Spo2 (%) in the Study Groups at Different Time Intervals

Time	SPO2				
	Group	N	Mean	SD	P value
Baseline	S	30	97.70	0.75	0.871
	V	30	97.73	0.83	
After induction	S	30	99.87	0.35	0.0.398
	V	30	99.93	0.25	
Immediately after intubation	S	30	100	0	-
	V	30	100	0	
1min	S	30	100	0	-
	V	30	100	0	
3min	S	30	100	0	-
	V	30	100	0	
5min	S	30	100	0	-
	V	30	100	0	
10min	S	30	100	0	-
	V	30	100	0	

DISCUSSION

AGE DISTRIBUTION

Mean age of the patients in the groups was compared using independent sample't' test. The average age in group V was 37.33 years and group S was 36.03. Therefore, we conclude that the difference between these groups was statistically insignificant (p value: 0.588).

ASA GRADE COMPARISON

The percentage of ASA grade 1 in group V was 56.7% and GROUP S was 53.3% which were comparable. The percentage of ASA grade 2 in group V was 43.3% and GROUP S was 46.7%. Chi-square test is used for comparison.

HEARTRATE

The trend of HEART RATE from baseline to over a period of 10 min after intubation. The basal (BL) mean heart rate of group S was 84.50±4.09 and group V was 84.17±7.64, and the difference in the mean heart rate at baseline among the groups was not statistically significant (p>0.05). After induction the mean heart rate starts increasing in group S and in group V mean heart rate start decreasing after induction and slightly raised after immediately after intubation and gradually decreasing till 10 min after intubation. In group S immediately after intubation highest mean heart rate was observed, and start decreasing at 1 minute, 3 minute, 5 minute and 10 minute after intubation but not reached baseline level, the mean heart rate between two groups from after induction to 10 minutes after intubation was significant. (p<0.05).

SYSTOLIC BLOOD PRESSURE:

At baseline, the mean systolic blood pressure (SBP) of group S was 129.13± 7.51, group V was 129.60±7.38 and the difference in mean SBP at baseline among the groups is not statistically significant (p>0.05). After induction the mean SBP starts decreasing in both groups. However, in group S mean SBP start increasing immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation. The mean SBP between two groups from 1 minute after induction to 10 minutes after intubation was significant. (p<0.05).

DIASTOLIC BLOOD PRESSURE

At baseline, the mean Diastolic blood pressure (DBP) of group S was 74.93±3.99, group V was 75.50±3.69, and the difference in mean DBP at baseline among the groups is not statistically significant (p>0.05). After induction the mean DBP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but never reached base line level. However, in group V mean DBP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean DBP between two groups from after induction to 10 minutes after intubation was significant. (p<0.05).

MEANARTERIALPRESSURE

At baseline, the average mean arterial pressures (MAP) in group S was 92.90±4.35, group S was 93.23±4.03 and the difference in mean MAP at

baseline among the groups is not statistically significant (p>0.05). After induction the mean MAP starts increasing in group S till immediately after intubation and gradually start decreasing from 1 minute after intubation to 10 minutes after intubation but reached baseline level at 3 minutes. However, in group V mean MAP start decreasing immediately after intubation and slightly increased immediately after intubation and gradually decreasing till 10 min after intubation. The mean MAP between two groups from after induction to 10 minutes after intubation was significant. (p<0.05).

MEAN SPO2

At base line, the average MEAN SPO2 in group S was 97.70±0.75, group S was 97.73±0.83 and the difference in mean MAP at baseline among the groups is not statistically significant (p>0.05). After induction the mean SPO2 starts increasing in both groups and reached to 100% in both groups.

They showed that the inter group comparison resulted in a statistically significant reduction in HR by dexmedetomidine than normal saline. These findings correlated with findings in our study in that it reduced HR significantly with dexmedetomedine 0.5mcg/kg. The 10 previously treated patients were given permission to inhale 6–8 ml of a solution comprising a third of 2% viscous lignocaine and a second-third of 4% aqueous lignocaine (nebulizing lignocaine), while the other 10 patients received normal saline as a comparison. Systolic blood pressure rose by an average of 10.3% in pre-treated patients, while pulse rates rose by 16.8%. In contrast, blood pressure rose by 56% in the control group, while pulse rates rose by 38.8%. In addition, when compared to Vishalakshi Patil et al., (2012),[13] study, group III (4% lignocaine nebulization) experienced an average rise in HR of 8.5%, group II (2% lignocaine nebulisation) of 10.7%, and control experienced a jump of 23.75%.SBP increased on average by4% in group III, 13.3% in group II, and 16.1% in the control group. In contrast to the aforementioned study, we found that the average increase in HR and SBP in the group receiving 4% lignocaine nebulization was 5% and 2%, respectively, in our investigation.

Covino BG concluded that intravenously administered lignocaine is less effective in decreasing the pressor response so a better alternative need to be used following further comparative studies.[14]

In a prospective, randomised, double-blinded, and placebo-controlled study, Vishalakshi Patil et al. (2012) compared the effects of 2% and 4% lignocaine nebulization on the pressor response to laryngoscopy and intubation. They concluded that these procedures significantly raised blood pressure and heart rate. In order to lessen the pressor reaction to laryngoscopy and intubation, they found that 4% lignocaine nebulization was more successful than 2% lignocaine nebulization.[13]

HEART RATE CHANGES

The least increase in heart rate are in nebulised group when compared to intravenous group as he had used higher dose of drug, which is seen in other studies where the nebulised group received higher dose of drug. No episodes of bradycardia were there in any of the groups of our study with clinically significant.[15]

BLOOD PRESSURE CHANGES

Considering that they utilised 1.5 mg/kg as opposed to 2 mg/kg, maximum rise in mean arterial pressure of 21.2 mm Hg noted with intravenous group and minimum with nebulized lignocaine of 120 mg of10.1mmHg which did not concur with our study as the pressor response was much better statistically significant in group I when compared with group N. Because nebulization was administered using a simple face mask, the drug concentration was lower, and there may have been drug loss during exhalation, there were not many significant changes in blood pressure in the control and nebulized groups in the current study.

There was a significant rise in pulse rate during laryngoscopy and endotracheal intubation in both groups, heartrate rises with NTGand lignocaine but more rise with NTG was statistically significant. The mean heart rate did not come to the pre induction levels even by 10th minute both group. We observed that NTG and lignocaine spray does not attenuate the rise in HR

CONCLUSION

Oral lignocaine viscous 2% and lignocaine spray 10% contributein blunting haemodynamicresponse to laryngoscopy and intubation in patients undergoing surgical procedures under general anaesthesia. Oral lignocaine viscous 2% was more effective in blunting the haemodynamic response to laryngoscopy and endotracheal intubation than lignocaine spray 10% No significant adverse events occurred during the study.

Limitations

As the sample size is ver yless and it single center study the generalize ability of the results is doubtful.

Recommendations

Further research should be done in different centers to consolidate the results of the study.

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